AORTIC REGURGITATION

Valvular disease (primary AI)
- Bicuspid valve
- Endocarditis
- RAA (with or not combined AS)
- Radiation
- Subaortic stenosis (rare – jet lesions cause destruction of the valve)
- Anorexic drugs (fenfluramine, phentermine, ergots)
- Congenital (unicuspid and quadricuspid valve)
- Balloon valvulotomy
- Trauma
- LED, PAR, ankylosing spondylitis, Whipple, Crohn disease, Takayasu

Aortic root disease (secondary AI)
- Dilatation
  - Age (degenerative)
  - Hypertension
  - Bicuspid valve and Marfan (cystic medial necrosis)
  - Aortitis: syphilis, collagen-vascular disease (Takayasu, ankylosing spondylitis, giant cell arteritis. Behçet), arthritis with ulcerative colitis and psoriasis, reactive arthritis, giant cell arteritis, osteogensis imperfecta
  - Supracristal VSD
- Dissection
- Trauma

Acute: trauma, endocarditis (perforation of leaflet or non coaptation of the cusps), and dissection

PATHOPHYSIOLOGY

- Severity related to **regurgitated volume** (severe has >50% RV)
- **Volume overload state**
  - Initial EF increase – chronic pressure overload
  - LV dilates, increase SV but EF constant + eccentric hypertrophy (LVEDP remains normal)
  - LV filling pressure constant with dilated LV
  - Chronic AR – increase LV compliance
  - LV decompensate – eccentric LVH, chamber enlarges but proportionally smaller increase in wall thickness, LVEDP increase as for afterload = ↓ EF
    - Fibrosis, decline of compliance LV, LV end-diastolic pressure and volume ↑
    - Coronary perfusion pressure ↓ = reduction of coronary reserve
- Largest end-diastolic volumes = ***cor bovinum***

◆ ACUTE AR

- Severe acute volume overload
- Elevated LVEDP, CO falls
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* End-systolic volume is a strong predictor of adverse clinical outcomes

◆ CHRONIC AR

Symptoms: exertional dyspnea, orthopnea, DPN, angina, heart pounding, fatigue

BP
- ***SBP increases, DBP falls = ↑ pulse pressure***

- HD significant AR, DBP<70, SBP >140
- SBP not more than 160 unless systemic HTN
- Severe AI, DBP 40-50, approaching LVEDP
- Use Korotkoff muffling; DBP may persist to 0 (although DBP doesn't fall below 30 mmHg)

Carotid

- ***High amplitude*** (large SV and enhanced rate of ejection) – brisk systolic upstroke with diastolic collapse
  - ***Exaggerated if arm elevated simultaneously with palpation of radial artery***
  - Rapid falloff in late systole/collapsing – early diastolic reflux into LV = low diastolic pressure
- Bounding pulses throughout body
- *Bisferiens* – Hallmark of AI – double systolic impulse
  - Best felt with light finger pressure over carotids
  - May feel systolic thrill or bruit
  - May have shudder with or without AS
  - Bisferiens present in mod/severe AR or AR with mild AS
- *Corrigan's sign* – visible pulsations of supraclavicular and carotids with high amplitude and rapid collapse
- *De Musset's* sign – visible oscillation or head bobbing with each heart beat

Peripheral signs

- d/t forceful ejection of increased SV into a dilated arterial bed; never in mild AR
- Attenuated with low CO or onset of heart failure
- *Quincke's* – capillary pulsation with light pressure at the distal bed
- *Water-hammer pulse (Corrigan)* – high amplitude, abruptly collapsing pulse
- *Palmar click* – palpable, abrupt palm flushing in systole
- *Muller's sign* – visible systolic pulsation of uvula
- *Pistol shot or Traube* – loud systolic and diastolic sound, over femoral artery, stethoscope lightly placed
- *Duroziez's* – to-and-fro bruit over femoral artery when light pressure to artery (systolic if pressure proximal of the femoral artery and diastolic if compressed distally)
- *Hill's* sign – SBP popliteal > 20 mmHg SBP brachial *** **NO longer considered a sign of severe AR (artifact of TA measurements)**

Precordial

- *Mild/moderate*
  - Apex not displaced
  - LV impulse N size, maybe hyperdynamic
- *Severe AR*
  - Systolic thrill at the base
  - Apex hyperdynamic, diffuse
  - Apex displaced **inferolaterally**
  - Palpable S4 (presystolic distention) in left decubitus
  - Visible and palpable rapid LV filing wave (S3)

S1

- Mild/mod – normal
- Severe – **soft**
- Aortic ejection click can be present
  - More common if mild AR d/t good LV fct
  - valve (BAV) or root (sudden systolic expansion of AscAo in early systole) origin

S2

- **Severe - soft or absent A2** if disease caused by the valve itself
- **Normal or loud A2** if AR caused by disease of aortic root

S3

- not in mild/mod
- Severe – **S3 common**
- Before Austin flint

S4

- rare in mild

- seen in mod; common in severe
- if long PR – S4 louder

DIASTOLIC MURMUR

- Decrescendo
- **Early after A2** (vs pulmonary regurgitation)
- Volume and velocity tapers off in mid-late diastole
- Length depend on severity except severe acute AR
- **Listen in held expiration, leaning forward**
- May not hear in trace/mild – too high freq/pitch
    - mild AR – not audible in late systole
- **Low amplitude, high frequency, really have to focus to hear**
    - If DBP<60, pandiastolic murmur anticipated
        - **More severe may get shorter that mild, if LV decompensation (rapid equilibration of Ao and LV pressure)**
        - Free AR – quieter
- Site
    - Parasternal LICS 3-4 = valvular disease
    - May be best heard in apex esp. elder or if grade 2 or more
    - If RICS = Aortic root disease (aneurysm, sinus of valsalva aneurysm, dissection)
- Aids to auscult
    - Optimal – lean forward, end expiration, firm pressure with diaphragm
    - Sustained handgrip or squat can bring out (increased SVR/afterload)
    - Younger pt – heard better supine
    - Elder – apex

SYSTOLIC MURMUR – ejection murmur

- mod-severe – common
- large SV ejected with rapid force = abrupt distension of Ao
- Higher pitch and often systolic thrill
- short and peaks before second half of systole
- lengthens in proportion to increase LV ejection time

AUSTIN FLINT

- low pitched rumbling apical diastolic murmur sounds exactly like MS - Ao flow on anterior mitral leaflet
- Mid-diastolic and late diastolic apical rumble
- best heard with bell, light pressure, pt left decubitus
- Indicates large leak – RF>50%

DIFFERENTIAL DIAGNOSIS

	Austin flint	MS
Rhythm	NSR	Afib
LV heave	Common	Absent
RV lift	Absent	Present
S1	**N to decreased**	**Loud**
OS	Absent	**Present (closer to S2 than S3)**
S3	**Present (+/- palp)**	Absent

- MS can be med to high frequency and may radiate to LLSB

Pulmonary regurgitation : severe PHTN, high-pitched blowing murmur (**graham steel**)
- PR associated with other signs of PHTN (RV lift, increased P2)

NATURAL HISTORY OF CHRONIC AR

- Quantitative measures of AR severity, LV size and EF predicts clinical outcomes
- In asymptomatic patients with normal EF, 10 year survival superior mild AR vs severe AR
- Death at 4 years after angina and 2 years after heart failure
- Gradual LV deterioration before onset of symptoms
    - More likely to be reversible if detected early (less dilated, EF less depressed and less symptoms)
    - **TIMING of surgery before irreversible changes is mandatory**

ACUTE AR

? Why signs are absent?

- Hypotension, tachycardia, heart failure, pulmonary congestion – ↓ CO
- LV failure = systemic vasoconstriction – **peripheral signs absent**
- Sinus tachycardia + premature closure of mitral valve shortens diastole
- **Difficult to appreciate diastolic murmur because LVEDP and Ao diastolic pressure equilibrates rapidly**
- Premature closure of mitral valve in early diastole by LVEDP >>> LAEDP
- No LV dilatation and increase SV – **no wide pulse pressure**
- High LVEDP → **premature closure MV**
    - S1 soft, late diastolic mitral sound can be heard (valve partly closed), apex normal
    - LVEF acute drops
    - S3 and S4
    - **Less gradient – early diastolic murmur is lower pitched and shorter**
    - **Signs of pulmonary HTN (P2 accentuated)**

	Chronic	Acute
Resting HR	Normal	Tachycardia; can confuse systole for diastole
BP	SBP>140 DBP<70 Increased PP	SBP N or low DBP may not be low
Peripheral pulses	**Bisferiens** Increased amp/volume Peripheral signs	Contour unremark Little/no peripheral signs
JVP	N	Mean elevated V wave if functional TR
Precordium	ICS 5/6 to AAL Hyperdynamic or heaving +/- palpable S3/S4	LV N or slight enlarged Palpable S3 RV impulse if severe PHTN
S1	N or low	Low or absent (**premature MV closure**)
S2	Unremarkable A2 increase/decrease	Single (soft/absent A2) Increase P2
S3	Very common	Always
S4	Uncommon	Common
Ejection click	Possible	Ejection sound common Middiastolic MV closure sound
AR murmur	Med freq Usually holodiastolic Maybe short with rapid decrescendo Gr 3 unless CHF	Med freq, often harsh Musical if ruptured cusp Not holodiastolic, **maybe short and rapid decrease**
AF murmur	Diastolic +/- presys murmur	Always middias, no presystolic
Sys murmur	Usually present Can simulate AS or MR	Usually present MR common

Bounding pulses: Differential diagnosis

Sympathetic hyperactivity, anemia, fever, pregnancy, thyrotoxicosis, large AV fistula, PDA, and severe bradycardia

ELECTROCARDIOGRAM

LVH, LV strain

CHEST X-RAY

Cardiomegaly, ascendant Ao aneurysm, calcifications of aortic valve if AS associated

ECHOCARDIOGRAM

Most sensitive and accurate noninvasive technique for AR evaluation

LV size, EF, aortic pathology, valvular morphology, assessment of AR severity

- Chronic AR: **LV dilatation**
- Acute AR: eccentric jet towards anterior mitral leaflet = premature closure of mitral valve with ↑PTDVG
  - **M-mode**: fluttering of mitral valve with acute and chronic AR, with early closure (C')

- TTE is indicated in patients with symptoms or sign of AR for accurate diagnosis of the cause of regurgitation, severity and LV size and systolic function, and for determining clinical outcome and timing of valve intervention.

- TTE is also indicated with dilated aortic sinuses or ascending aorta or with a bicuspid aortic valve to evaluate the presence and severity of AR.

★ **Suggested reference:** Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. J Am Coll Cardiol 2014;63:e57-185 – **table 11**

Aortic dissection: TTE – 60% sensitivity and 80% specificity; TEE 98% sensitivity and 95% specificity

	Mild	**Moderate**	**Severe**
Signs of AR severity	Central jet width < 25% LVOT Vena contracta < 0.3 cm^2 No or brief early diastolic flow reversal in descending aorta	Signs of AR > mild but no criteria for severe AR	Central jet, width **≥ 65%** of LVOT Vena contracta **> 0.6 cm^2**
Supportive signs	PHT > 500 Normal LV size	Intermediate values	PHT **< 200** Holodiastolic aortic flow reversal in descending aorta Moderate to greater LV enlargement
Quantitative parameters R Vol (ml/beat) RF (%) EROA (cm^2)	 < 30 < 30 < 0.10	 30-44 30-39 0.10-0.19	 ≥ 60 ≥ 50 ≥ 0.30

★ **Suggested reference:** Zoghbi W. et al. Recommendations for the evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. 2003 J Am Society Echocardiography;16:777-802.

MRI

Most accurate noninvasive technique for assessing **end-systolic LV volume, diastolic volume and mass**

* Accurately quantifies severity of AR with antegrade and retrograde flow volumes in ascendant Ao and recommended when TTE is suboptimal

CATHETERIZATION

Not routinely recommended, except:
- Equivocal results with TTE
- C-I to MRI (device)
- Hemodynamic assessment of AR
- Coronary angiography

EXERCISE TESTING

Use to assess symptomatic patients and functional capacity in patients with AR.

MANAGEMENT

Medical treatment
Patients considered inoperable, treat aggressively heart failure with ACEI, digoxin, diuretics, BB (may be beneficial)
Treat systemic diastolic hypertension – ACEI and nefidipine are preferred, BB agents used with great caution (bradycardia poorly tolerated).
No indications of long-term treatment with ACEI of CCB in chronic AR patients (lack of evidence).
In acute AR = **vasodilators (nitroprusside)** + **inotropes (dobutamine), BB and IABP C-I**

Surgical treatment
★ **Suggested reference:** Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. J Am Coll Cardiol 2014;63:e57-185 – **Figure 2, table 12**

- NYHA 3-4 and LV dysfunction with EF less than 50% are **independent risk factors for poor postoperative survival**
  - Patients should be operated in NYHA 2 before severe LV dysfunction has developed.
  - EF < 25% = high risk (even if successful surgery)
- Severe AR with **normal LV function** – less 6%/year requires operation because of development of symptoms or LV dysfunction (higher if > 60 years) = differ surgery
- **LV end-systolic dimensions** predicts outcome in asymptomatic patients
  - < 40 mm = stable
  - > 50 mm = 19%/year developing symptoms and LV dysfunction
  - > 55 mm = increase risk of irreversible LV dysfunction
- Indexed end-systolic dimension more robust indicator for timing of surgical intervention = ≥ 45 cc/m^2
- **Chronic AR**
  - Differ surgery in asymptomatic patients with normal LV function
  - Recommended for symptomatic patients with mild or moderate LV dysfunction
  - Surgery is advised in asymptomatic patients with progressive LV dysfunction and dilatation

Procedures
- AVR: valve replacement in majority
  - Surgical repair (aortic cusp resuspension, resection or pericardial patch if perforated)
- Concurrent aortic replacement when Ao dilatation
  - Aneurysm: excision of ascending Ao, replacement with a graft with AVR, reimplantation of coronary arteries (+/- valve sparing when aortic root is replaced or repaired)
  - Mortality rate 3-8%

Content of this summary from these references:

- Otto C & Bonow R. Valvular Heart Disease. (2012) In Bonow R. *et al*. Braunwald's Heart Disease, 9th edition, pp. 1468-1539. Philadelphia, PA: Elsevier.
- Nishimura R. A. et al. 2014 ACC/AHA guidelines for the management of patients with valvular heart disease. J Am Coll Cardiol 2014;63:e57-185.
- Zoghbi W. et al. Recommendations for the evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography. 2003 J Am Society Echocardiography;16:777-802.