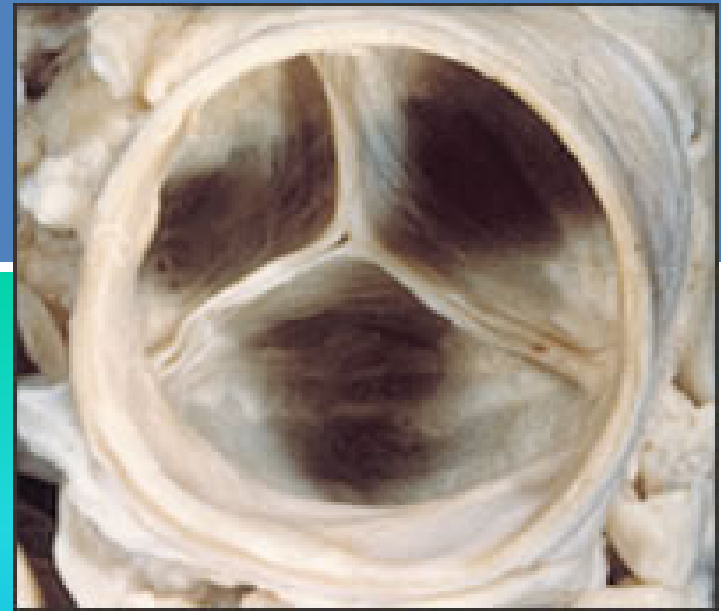


# Aortic valve disease



- Aortic stenosis .
- Aortic regurgitation .

DR YAHYA KIWAN

Consultant cardiologist  
& interventionist (DH )

# Aortic stenosis

- The normal aortic area is 3-4 cm<sup>2</sup>, in severe valvular AS (< 1cm<sup>2</sup>)
- Normally, little or no pressure difference across the aortic valve
- In significant aortic stenosis LV pressure may exceed that of the aorta by >50 mmHg .
- AS causes progressive obstruction of LV out flow tract resulting in pressure hypertrophy of LV .
- Untreated, AS is assoc with significant morbidity & mortality .

# Types Aortic stenosis

valvular

Subvalvular

Supravalvular

**Congenital condition  
fibromuscular membrane  
in the LVOT below AV**

**(uncommon)**

# Etiology of Valvular AS

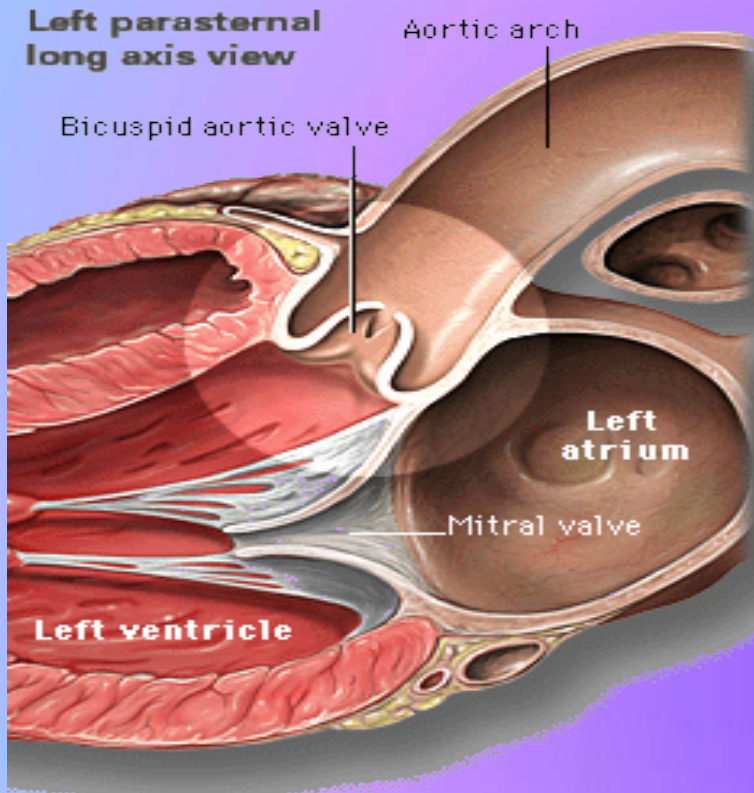
## ❖ Congenital

❖ Rheumatic (often coexist with AR & MVD)

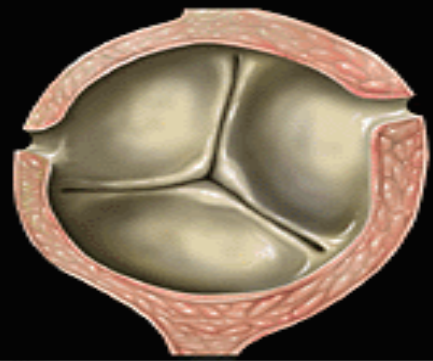
❖ Bicuspid (1-2%, predominate in males, in 9% of 1st degree, may be stenotic or regurgitant)

❖ Age-related calcific degeneration

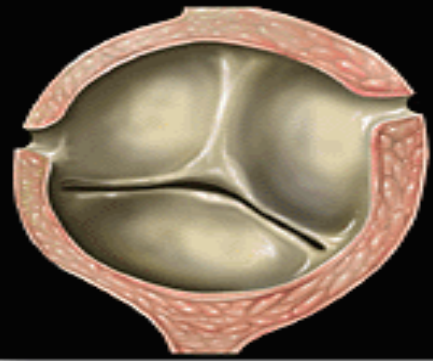
**Short axis views from above aortic valves**



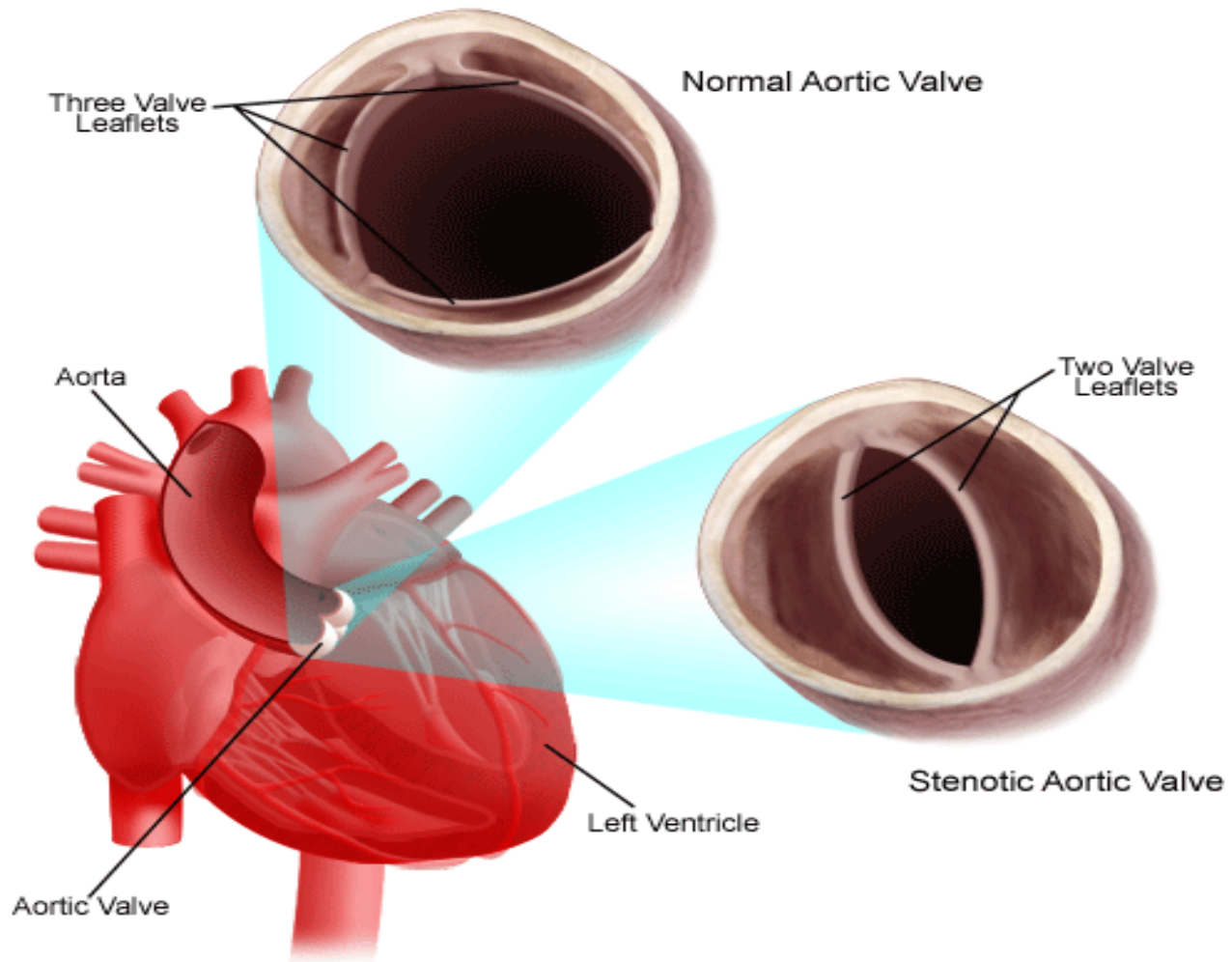
**Normal aortic valve**



**Bicuspid aortic valve**



## An Example of Aortic Stenosis



# Symptom of AS

- **Angina** (↓ myocardial perfusion, associated CAD (occurs in 40-80% of pts with angina & 25% in pts with out angina))
- **Syncope**
- **Heart failure**
- **Sudden death** (hypotension, arrhythmia due to ischaemia, LVH or impaired LVF )

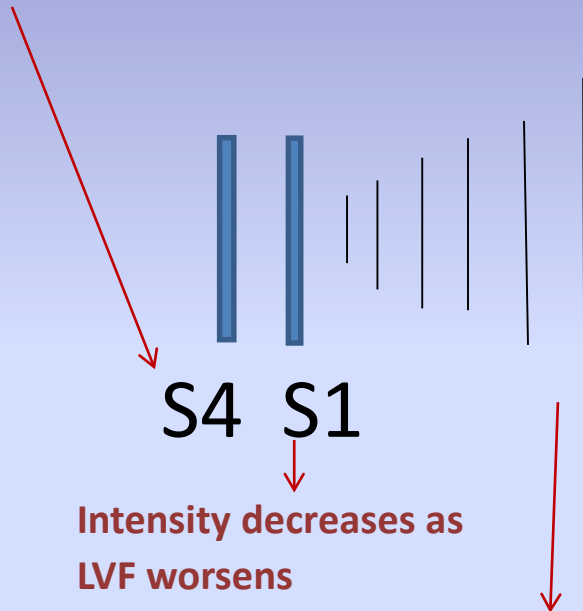
# Physical finding in AS

- Arterial examination:-  
Diminished & delayed carotid upstroke  
(pulsus parvus et tardus )
- Palpation :-  
None displaced ,diffuse, sustained apical impulse , double apical impulse (palpable a wave or S4 caused by noncompliant LV .  
systolic thrill in the 2<sup>ND</sup> Rt ICS .



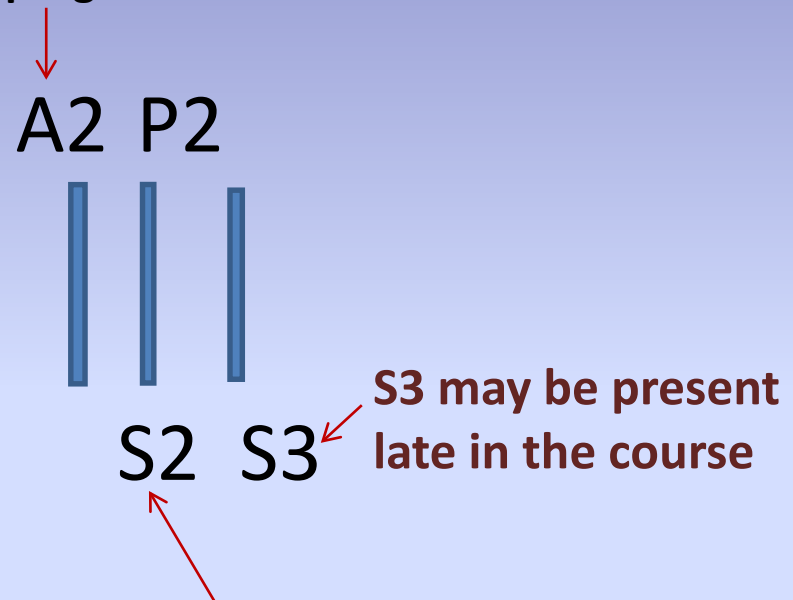
# Auscultatory finding in AS

Often present due to LVH & poor LV compliance



Murmur is systolic ,harsh best at Rt upper sternal border radiates to carotids peaks in early to mid systole until late in the course when it peaks later & is more intense

Intensity decreases as AS progresses



S2 becomes soft as A2 decreases in intensity. may paradoxically split as severity increases may become soft & single late in the course.

# Diagnostic testing of AS

- ECG ( LA abnormality ,LVH)
- CXR (boot shape,cardiomegally,calcification)
- Echocardiography (T TE,TEE)
- Cardiac catheterization.

# Therapy of AS

## ✦ Medical

- **Antibiotic prophylaxis**

- **Asymptomatic :- (1ry prevention of CAD  
maintenance of SR , blood pressure control,**

- **symptomatic :-treatment of heart failure**

## ✦ Percutaneous aortic balloon valvuloplasty (PABV)paediatric congintal,noncalcific,Rh AS)

## ✦ Surgical AVR (main stay in severe AS )

## Indication of PABV in adults with AS

- # Bridge to surgical aortic valve replacement in haemodynamically unstable patient .
- # Palliative therapy for nonoperative candidate
- # (high surgical risk due to multiple comorbidities or pt refuses surgery)
- # Pts with critical AS who require urgent noncardiac surgery

# Recommendation of AVR

- **Symptomatic Pts with severe aortic AS**
- **Pts with severe AS undergoing CABG .**
- **Pts with severe AS undergoing aortic or other valve surgery .**

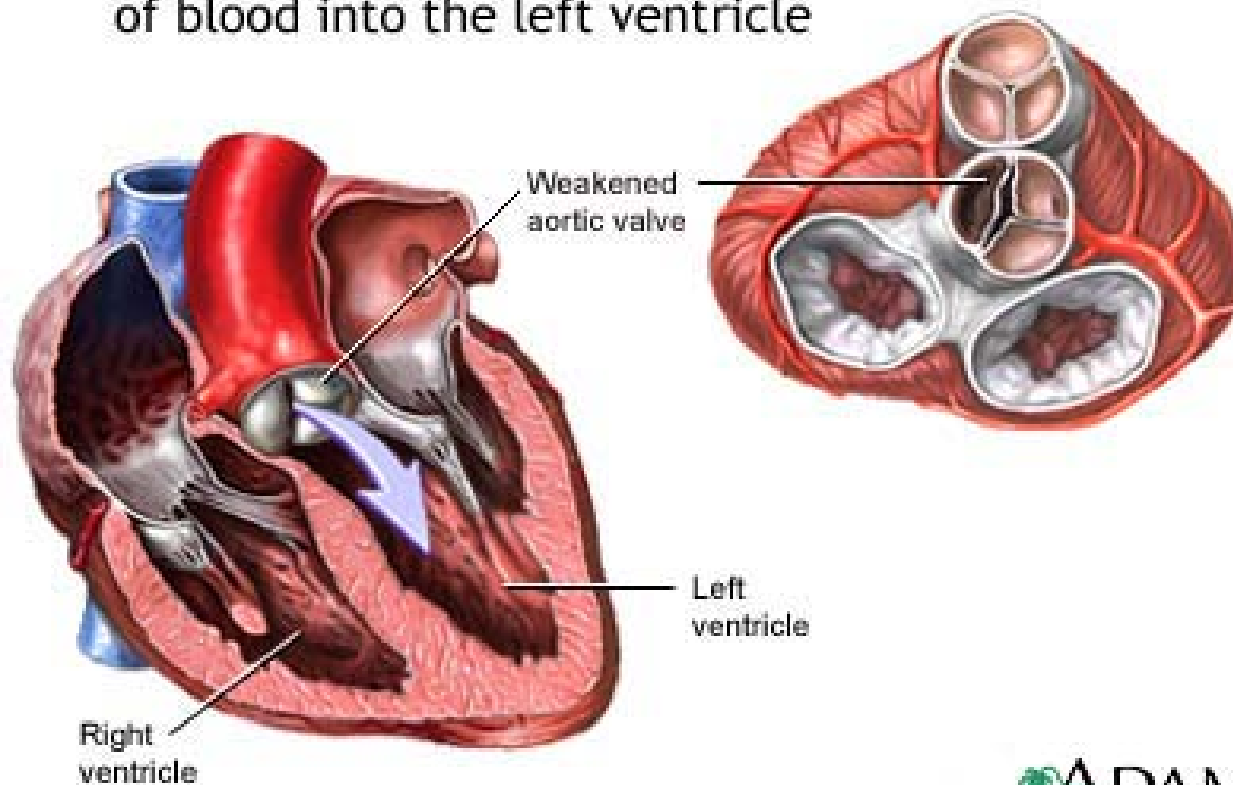
## **Possibly indicated :-**

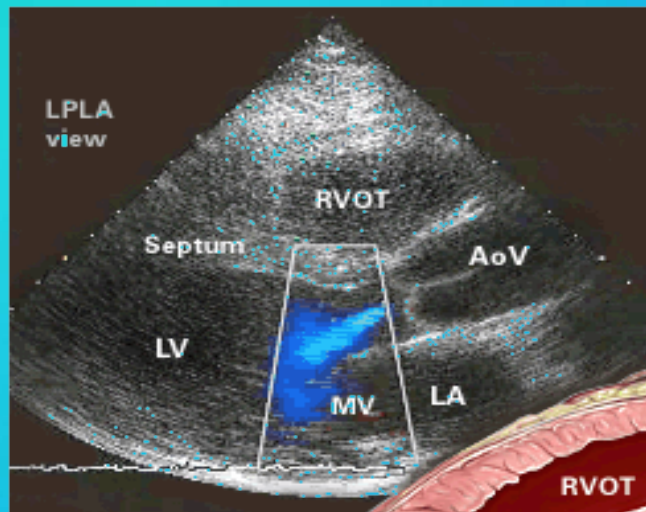
- Pts with moderate AS who require CABG or aortic or other valve surgery .
- A symptomatic Pts with severe AS & :-
  - impaired LV F (EF < 50%)
  - symptoms, hypotension or ventricular arrhythmias **during exercise .**

# Aortic regurgitation

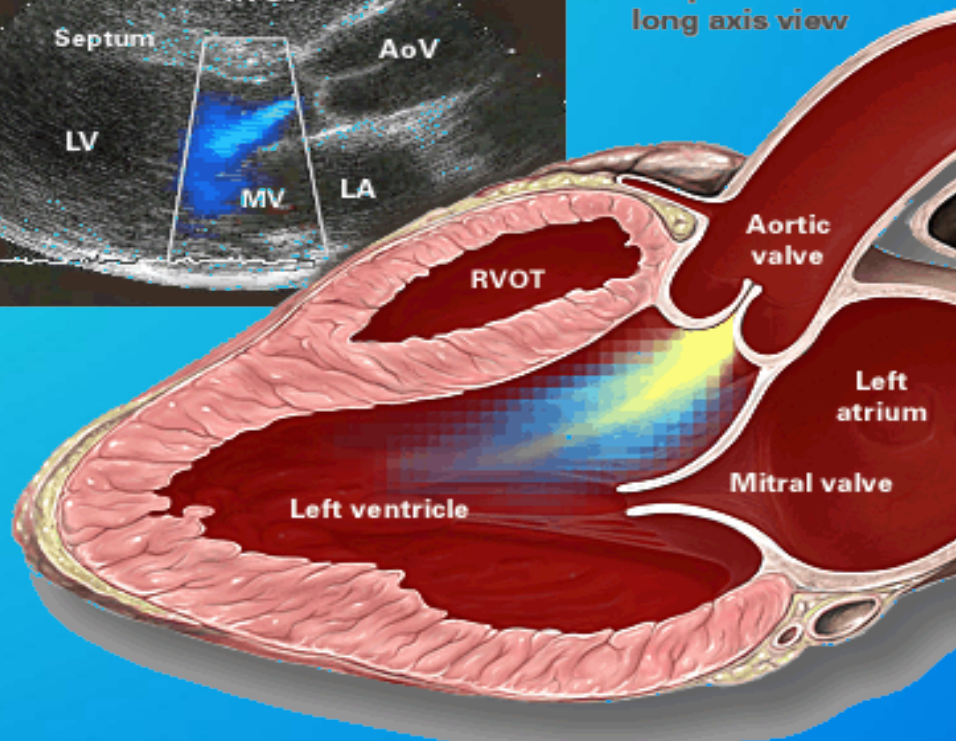
- Can develop from
  - ✖ 1ry disease of the valve leaflets
  - ✖ Abnormality of aortic root or ascending aorta.
- Can be acute or chronic

Failure of the aortic valve to close tightly causes back flow of blood into the left ventricle





Left parasternal long axis view





# Major causes of chronic AR

<b>Leaflet abnormalities</b>	<b>Aortic root or Ascending Aorta Abnormalities</b>
<b>Rheumatic fever</b>	<b>Age related aortic dilatation</b>
<b>Infective endocarditis</b>	<b>Annuloaortic ectasia</b>
<b>Trauma</b>	<b>Cystic medial necrosis of aorta (isolated or marfan syndrome)</b>
<b>Bicuspid aortic valve</b>	<b>Systemic hypertension</b>
<b>Myxomatous degeneration</b>	<b>Aortitis (syphilis ,giant cell arteritis )</b>
<b>Congenital aortic regurgitation</b>	<b>Reiter's syndrome</b>
<b>SLE</b>	<b>Ankylosing spondylitis</b>
<b>Rheumatoid arthritis</b>	<b>Behçet syndrome</b>
<b>Ankylosing spondylitis</b>	<b>Psoriatic arthritis</b>
<b>Takayasu's arteritis</b>	<b>Osteogenesis imperfecta</b>
<b>Crohn's disease</b>	<b>Relapsing polychondritis</b>
<b>Drug induce valvulopathy</b>	<b>Ehlers –Danlos syndrome</b>

# Major cause of acute AR

<b>Leaflet abnormalities</b>	<b>Aortic rot or ascending aorta abnormalities</b>
<b>Traumatic rupture</b>	<b>Acute aortic dissection</b>
<b>Acute infective endocarditis</b>	<b>Perivalvular leak or dehiscence of prosthetic valves</b>
<b>Acute prosthetic valve dysfunction</b>	
<b>Poor aortic balloon valvuloplasty</b>	

# Symptoms of AR

## Chronic AR

Usually asymptomatic for along time .

After LV dysfunction – symptoms of pulmonary congestion - dyspnea ,PND

Chest discomfort (due to LV enlargement exaggerated after pvc's & in supine position

Angina (uncommon, due to latent CAD, reduced diastolic coronary perfusion, nocturnal bradycardia , LVH, subendocard ischaemia

## Acute AR

Sudden haemodynamic deterioration hypotension, tachycardia, pallor, cyanosis, Diaphoresis, cool extremities, pulmonary congestion

Weakness, altered mental status , SOB syncope .

If severe chest pain is present aortic dissection should be suspected .

# Physical findings in chronic AR

- ✦ characteristic peripheral pulses, cardiac auscultatory findings.
- ✦ Pts with chronic AR should be examined for:-
  - Peripheral manifestation of infective endocarditis
  - Signs of Marfan syndrome .
  - Evidence of chronic aortic dissection
  - Signs for collagen vascular disorder .

# Physical signs associated with hyperdynamic pulse in chronic AR

Physical sign	Description
Water hammer or Corrigan's pulse	Rapid upstroke followed by quick collapse
De Musset's sign	Head bob with each heart beat
Traube's sign	Pistol shot sounds heard over the femoral arteries in both systole & diastole
Müller's sign	Systolic pulsation of the uvula
Duroziez's sign	Systolic murmur over the femoral artery when compressed proximally & diastolic murmur when compressed distally or systolic-diastolic murmur with increasing compression over femoral artery
Quincke's sign	Capillary pulsations visible in the lunula of the nail bed
Hill's sign	Popliteal cuff systolic pressure > brachial cuff systolic pressure by >60mmHg
Becker's sign	Arterial pulsations visible in the retinal arteries & pupils

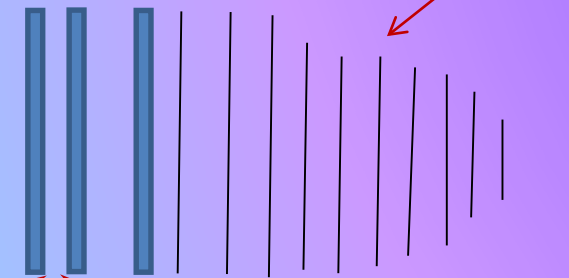
# Auscultatory finding in AR

A2 may be soft due to poor coaptation

P2 may be obscured by the murmur

Murmur is diastolic, blowing, Decrescendo best heard at the Lt upper SB severity correlates more with duration than intensity

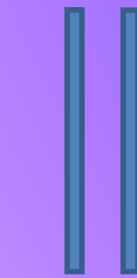
A2 P2



S2 S3

S3 may be present late in the course

May be narrowly split or paradoxically split due to prolonged systolic ejection time



S4 S1

Intensity decreases as LVF worsens

Often present due to LVH & poor LV compliance

Note :- Austin flint murmur mid-late diastolic rumble (vibration of AML) may be audible

# Physical examination maneuver in AR

Increase murmur	Decrease murmur
Isometric exercise (e.g., hand grip )	Standing from squatting position
squatting	Strain of Valsalva maneuver
Inotrope infusion	Inhalation of amyl nitrite

# Physical findings in acute AR

- Signs of hyperdynamic circulation characterize chronic AR often absent .
- Pulse pressure normal or slightly wide
- Heart size normal (undisplaced apex)
- S1 may be diminished due to preclosure of MV P2 may be loud, S3 (if cardiac decompensation)
- Murmur is shorter & lower in pitch , if severe may not be audible
- systolic flow murmur may be audible .
- Austin flint, if present , is shorter



# Diagnostic testing of AR

- **ECG** :- LVH,LAD,Lt atrial abnormality ,in acute AR non specific ST-T changes.
- **CXR** :- cardiomegaly (acute AR may be normal) pulmonary congestion .
- **Echo**:- TTE(2D M mode,Doppler color flow),TEE}.
- **Cardiac catheterization** :- coronary angiography to exclude CAD.

# Medical Therapy of AR

## Chronic AR :-

- ❖ Antibiotic prophylaxis .
- ❖ Drugs: (vasodilator (hydralazine ,ACEI,CCB) to slow progression of LV dysfunction &LV dilatation,B blocker in in aortic root dilatation

## Acute AR :-

- IV vasodilators & inotropic agents in pt with cardiogenic shock .
- Antibiotic if assoc with infective endocarditis
- management of acute AR Usually surgical according to the cause .

# Surgical therapy of AR

## Indications of aortic valve replacement in AR

### Class 1

NYHA class 1 or 1V or CHA class 1 to 1V symptoms (with or without CAD) with normal LVF (EF  $\geq$  50%)

NYHA class 1 & preserved LV systolic function (EF  $\geq$  50%) with progressive LV dilatation or declining EF at rest or declining exercise tolerance.

A symptomatic or asymptomatic pts with mild to moderate LV dysfunction at rest (EF 25 - 49%)

Pts undergoing CABG or surgery in the aorta or other heart valves.

### Class 1a

NYHA class 1 & preserved LV SF with stable LV SF, size & exercise tolerance

A symptomatic pts with normal LV F but with severe LV dilatation  
EDD  $>$  75 or ESD  $>$  55

# Surgical therapy of AR

## Indications of aortic valve replacement in AR( continued)

### **Class 11b**

**Pts with severe LV dysfunction (EF < 25%)**

**Asymptomatic pts with normal LVSF at rest & progressive LV dilatation which is moderately severe (EDD70-75,ESD 50-55% mm)**

**Asymptomatic pts with normal LVSF at rest but with decline in EF during(stress echo or exercise radionuclide angiography )**

**Asymptomatic pts with normal LVSF at rest &LV dilatation which is not severe (EDD <70 mm,DSD < 50 mm)**

Pt with chronic AR should be observed closely for the development of LV systolic dysfunction.

Follow up evaluation typically is conducted with serial echocardiography .

If signs o of LV systolic dysfunction develop surgical therapy should be considered .



THANKS



