

# Cardio-Vascular Disease

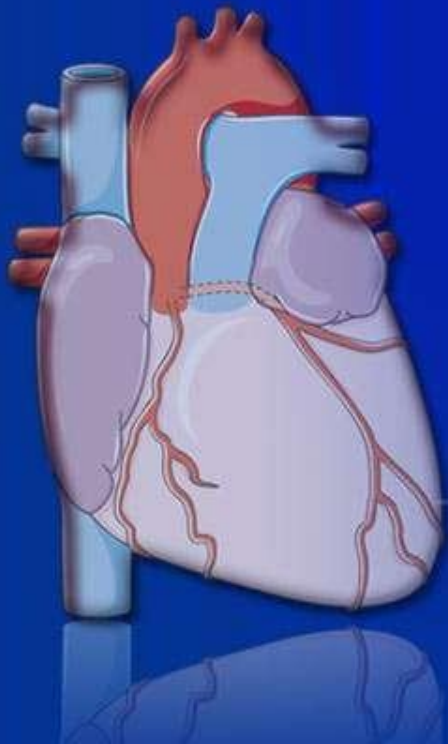
## **APRTIC REGURGITATION**

**Dr Sandeep Rai**



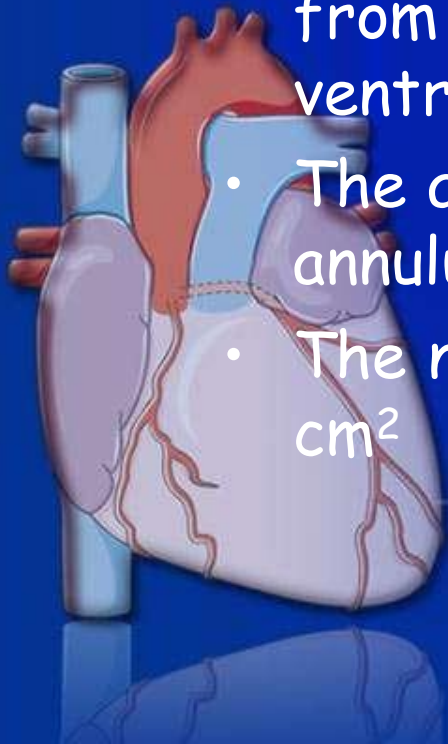
# Aortic Regurgitation

- Valve Anatomy
- Etiology
- Pathophysiology
- Epidemiology
- History & Physical Examination
- Natural History
- Differential diagnosis
- Investigations
- Assessing severity
- Medical Management
- Surgical Management
- Prognosis

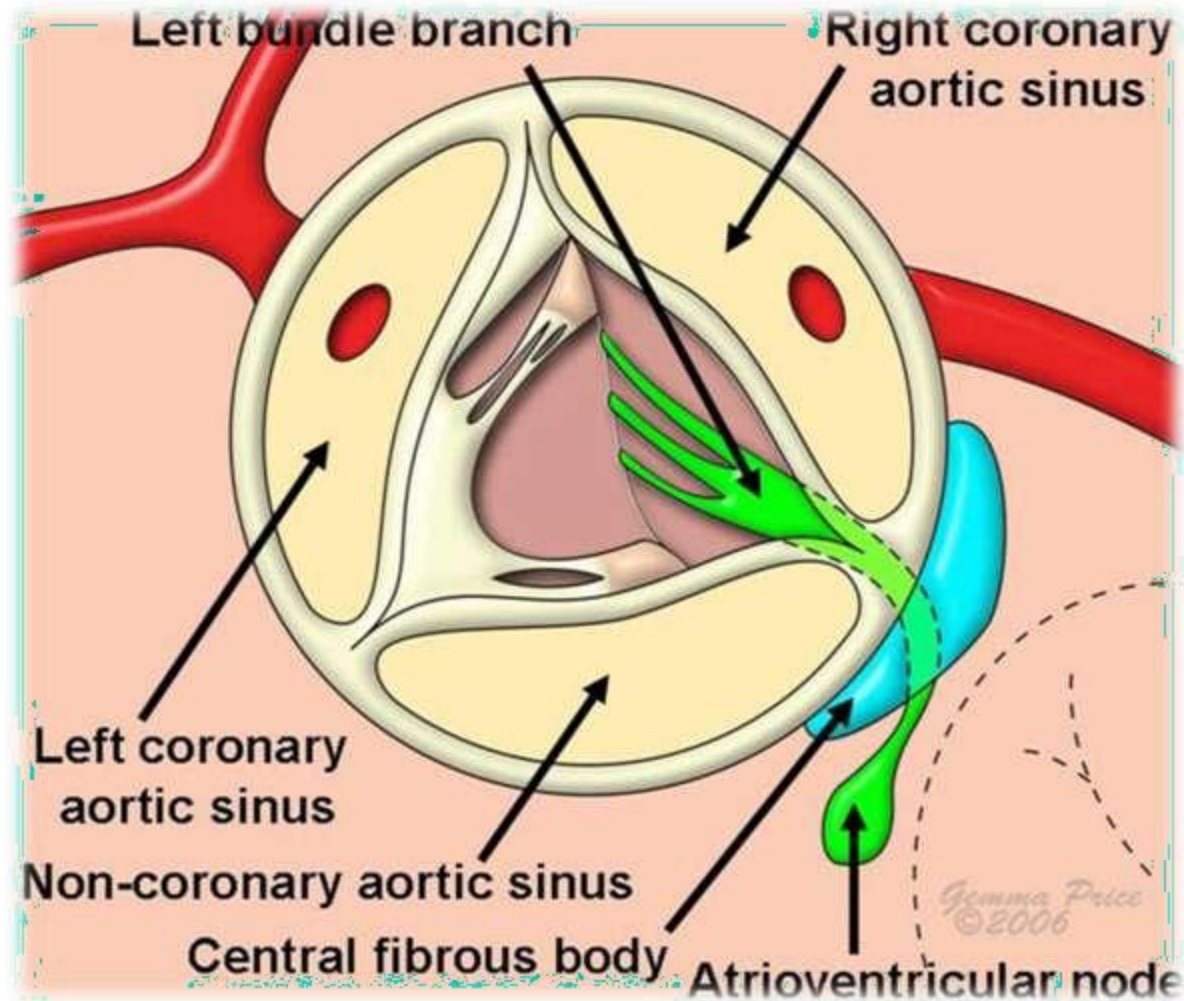


# Anatomy

- Located between the left ventricular outflow tract and the ascending aorta
- The aortic valve functions to prevent the regurgitation of blood from the aorta into the left ventricle during ventricular diastole and to allow the appropriate flow of blood—the cardiac output — from the left ventricle into the aorta during ventricular systole.
- The aortic valve has 3 principle components: the annulus, cusps, and commissures.
- The normal aortic valve area in adults is 3.0 to 4.0 cm<sup>2</sup>



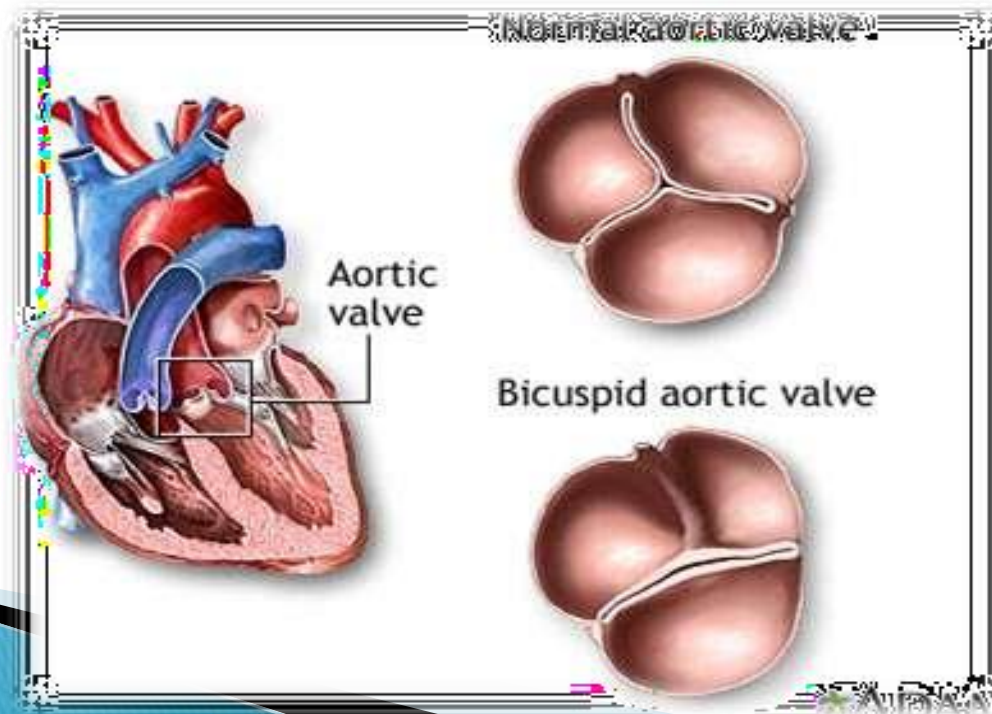
# Picture : Aortic Valve.



# Pathophysiologic Variants

## Bicuspid aortic valve

- Bicuspid aortic valves are the **most common cardiac valvular anomaly**, occurring in 1- 2% of the general population.
- It is twice as common in males as in females.



# MAIN

## Causes of Aortic Regurgitation

| Leaflet abnormalities | Aortic root or ascending aorta |
|-----------------------|--------------------------------|
| Rheumatic fever       | Systemic hypertension          |
| Endocarditis          | Aortitis (eg, syphilis)        |
| Trauma                |                                |
| Bicuspid aortic valve | Ankylosing spondylitis         |
|                       | Trauma/ Dissecting aneurysm    |
|                       |                                |
|                       | Marfan syndrome/ EDS           |
|                       | Inflammatory bowel disease     |

AR is seen more commonly in **men than in women**.

# Valvular Abnormalities

Rheumatic Disease



Aortic Root Dilation



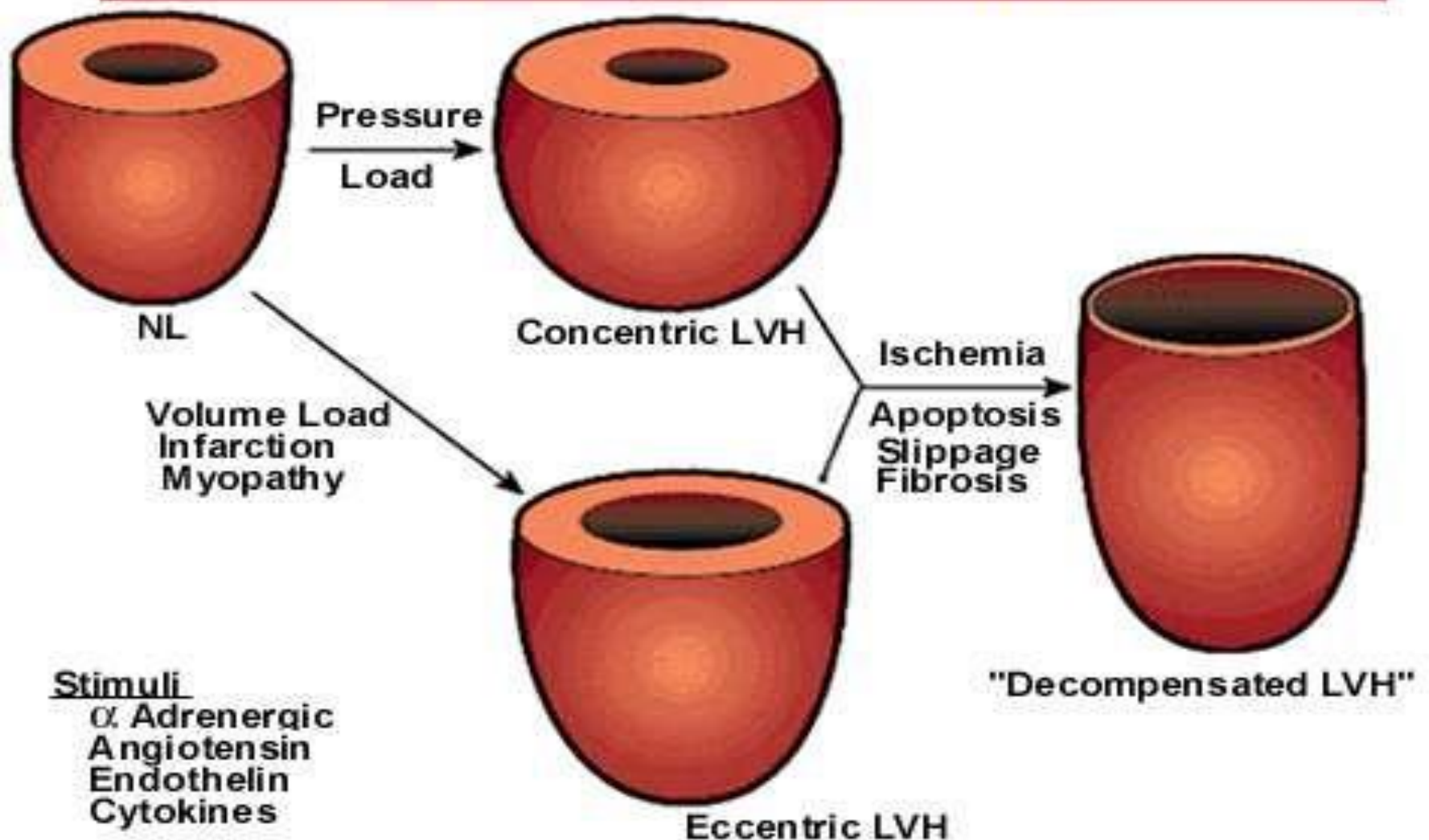
Endocarditis

# Pathophysiology of chronic AR

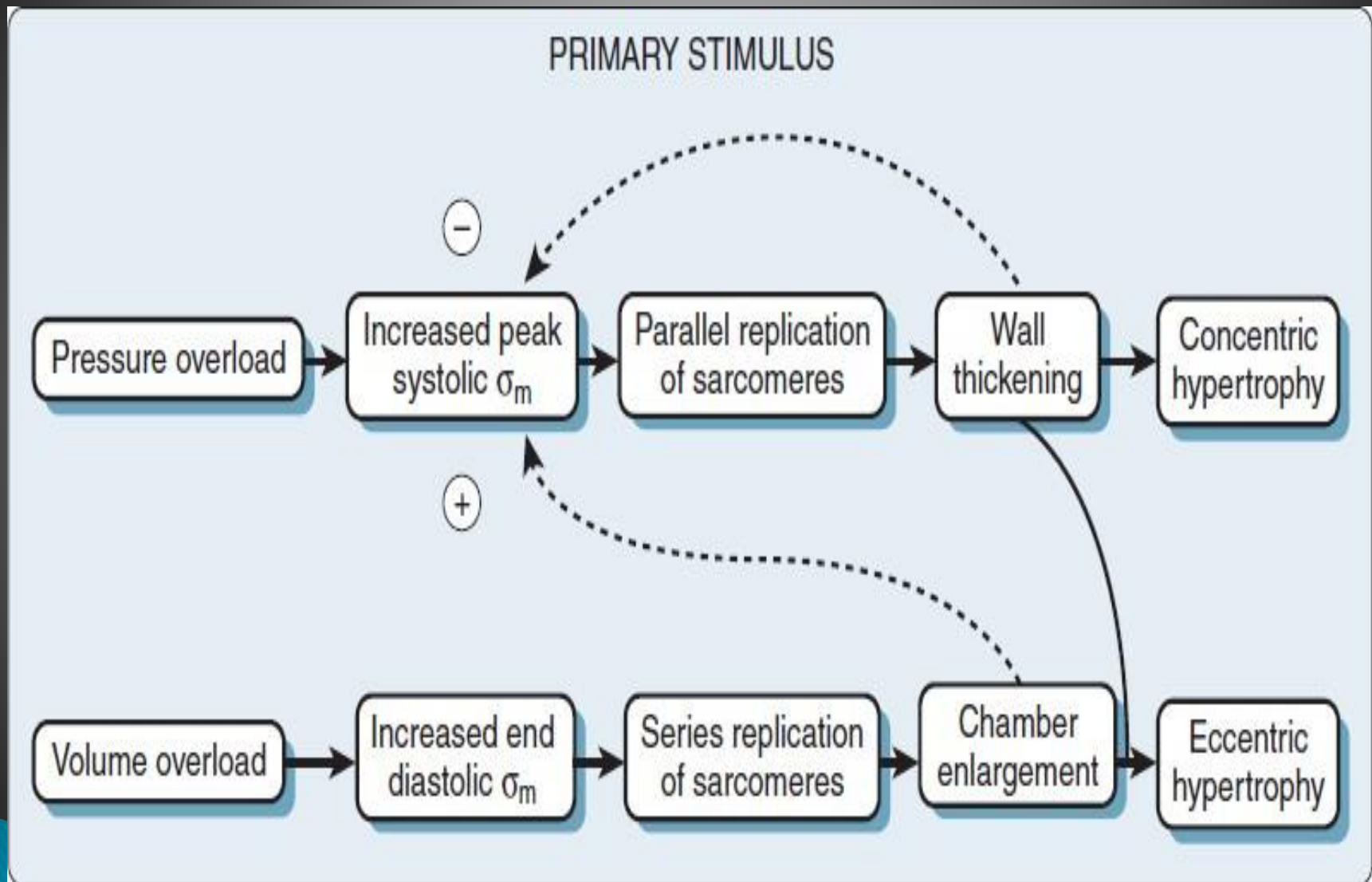
- AR leads to increased end diastolic volume of LV
- To accommodate this regurgitant volume LV compliance increases so there is augmented LVEDV.
- There is increase in total stroke volume resulting in forward stroke volume within normal range (starling law)
- In addition to increase in compliance, ventricle produces new sarcomers in sereies leading to **eccentric hypertrophy**. In AS **concentric LVH** occurs as the sarcomeres replicate in parallel

# Aortic Regurgitation Pathophysiology

## Schematic Representation of the Various Forms of Left Ventricular Hypertrophy



# Aortic Regurgitation Pathophysiology



# Pathophysiology of chronic AR

- As a result of maintenance of normal LV systolic function and normal forward stroke volume despite the increase in LV diastolic volume, most patients with chronic AR remain **asymptomatic for decades**
  - As AR progresses, LV enlargement surpasses preload reserve on the Frank-Starling curve, with the EF falling to normal and then subnormal levels.
  - The LV end-systolic volume rises and is a sensitive indicator of progressive myocardial dysfunction.
- 

# Pathophysiology of chronic AR

- This after load mismatch results in reduction in systolic ejection performance (reversible phase)
- Impaired myocardial contractility also contribute to LV systolic dysfunction (irreversible phase)
- At this point in natural history patient goes in decompensated phase and becomes symptomatic

Aortic regurgitation

Diastolic regurgitation

↑LV volume

↑Stroke volume

↓ Ao diastolic pressure

↓ Effective stroke volume (fatigue)

↑LV mass

↑ Systolic pressure

↑ LVET

↓ Diastolic time

LV dysfunction

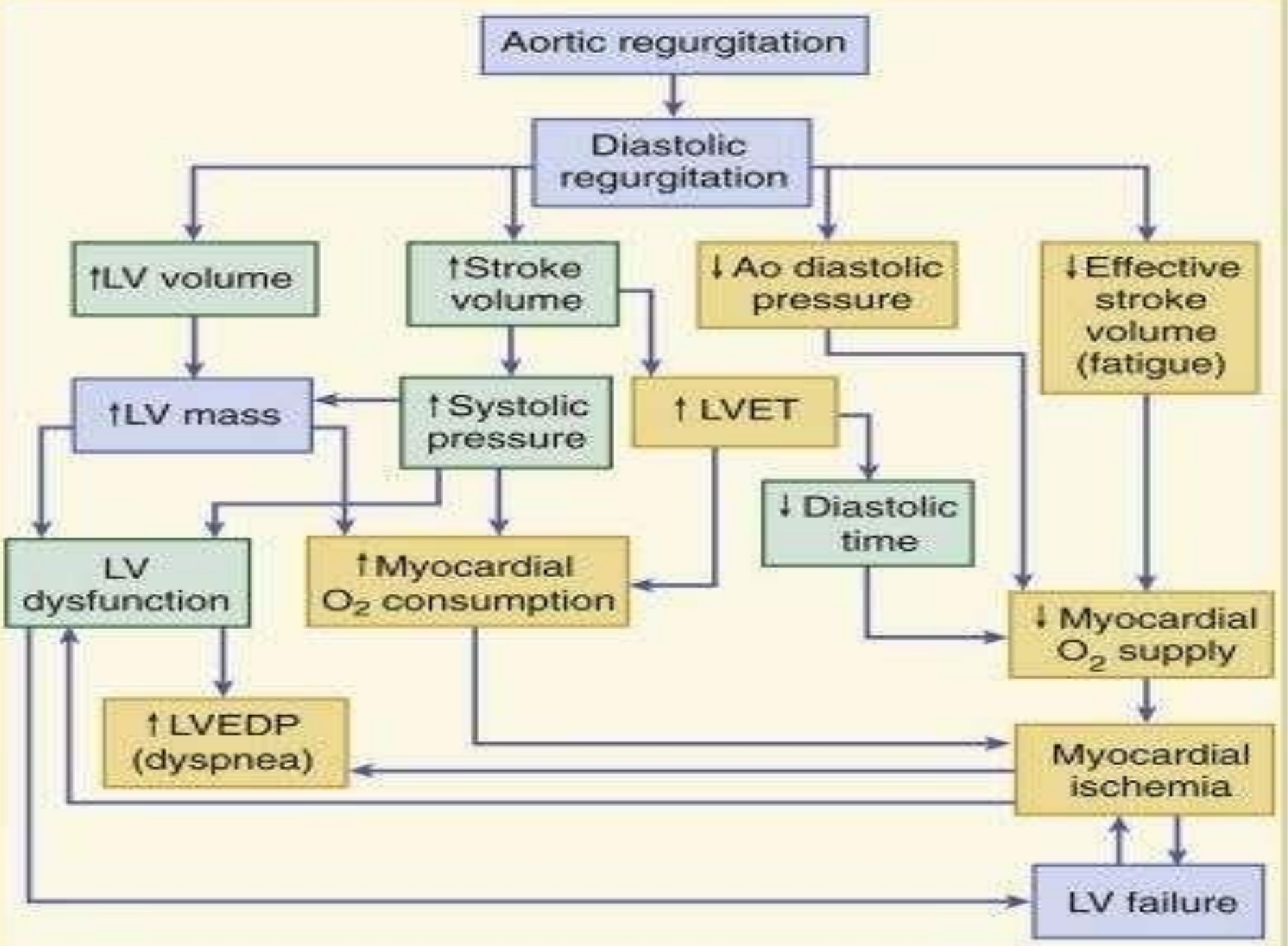
↑ Myocardial O<sub>2</sub> consumption

↓ Myocardial O<sub>2</sub> supply

↑ LVEDP (dyspnea)

Myocardial ischemia

LV failure



# Pathophysiology – Acute AR

- Acute AR of significant severity leads to increased blood volume in the LV during diastole. The LV does not have sufficient time to dilate in response to the sudden increase in volume. As a result, LV end-diastolic pressure increases rapidly, causing an increase in pulmonary venous pressure and altering coronary flow dynamics. As pressure increases throughout the pulmonary circuit, the patient develops dyspnea and pulmonary edema.
- In severe cases, heart failure may develop and potentially deteriorate to cardiogenic shock. Decreased myocardial perfusion may lead to myocardial ischemia.

# Physical Examination – Acute AR

- Peripheral vasoconstriction
  - Cyanosis
  - Pulmonary edema
  - Arterial pulsus alternans; normal LV impulse
  - Early mitral valve closure
  - Early diastolic murmur (lower pitched and shorter than in chronic AR) may be present.
  - A murmur at the right sternal border is associated more often with dissection than it is with any other cause of aortic regurgitation.
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# Epidemiology

- Approximately three-fourths of patients with pure or predominant valvular AR are men.
- Chronic AR often begins in patients when they are in their late 50s. In general, the prevalence and severity of AR increase with age.
- However, there are many exceptions to this observation. Patients with bicuspid aortic valve and, especially, those with Marfan syndrome tend to present much earlier

# Clinical presentation of AR

- **SYMPTOMS OF CHF (MOST COMMON)**
  - Exertional dyspnoea
  - Orthopnea
  - PND
- **Late--Right heart failure** (pedal oedema, ascitis, hepatomegaly)

# Clinical history

- **Palpitation**
  - Initial symptom due to vigorous contraction of volume overload LV
- **Angina**
  - Less common
- **Dyspnoea**
  - Initially on exertion
  - PND

# Clinical presentation of AR

**patients are asymptomatic for decades**

- **Palpitations** (it is awareness of forceful left ventricular contraction)
- **Non-specific chest pain**
- **Angina (less common)**
  - May be related to CAD
  - Or it may occur with normal coronaries (low diastolic pressure leads to decreased myocardial perfusion in the face of increased myocardial oxygen demand secondary to LVH)

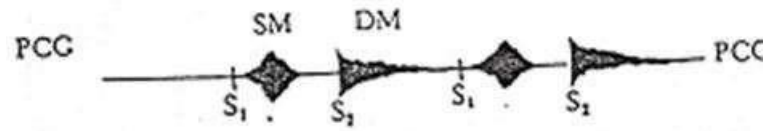
# Myocardial ischemia

- Decrease in diastolic pressure - coronary perfusion pressure is reduced.
- So increased O<sub>2</sub> demand & decreased O<sub>2</sub> supply sets myocardial ischemia

# Physical Examination – Chronic AR

- Manifestations of severe chronic AR are often the result of **widened pulse pressure** because (1) elevated stroke volume exists during systole and (2) the incompetent aortic valve allows the diastolic pressure within the aorta to fall significantly.
- On palpation, the point of maximal impulse may be diffuse or **hyperdynamic** but is often displaced inferiorly and toward the axilla. Peripheral pulses are prominent or bounding. Auscultation may reveal an  $S_3$  gallop if LV dysfunction is present.
- A **diastolic thrill** may be palpable along the left sternal border in thin-chested individuals, and a prominent systolic thrill may be palpable in the suprasternal notch and transmitted upwards along the carotids .

# Auscultation



- **Early-diastolic murmur**, immediately after A2, usually as a high-pitched blowing sound that is loudest at the left sternal border, decrescendo, best heard in at end-expiration & in sitting & leaning forward position. The duration of the murmur correlates better with the severity of AR than does the loudness of the murmur.
- When the murmur is musical (**cooing dove murmur**), it usually signifies eversion or perforation of an aortic cusp.
- **Harvey sign** - When regurgitation is caused by primary valvular disease, the diastolic murmur is heard best along the left sternal border in the 3rd and 4th ICS.
- When it is caused mainly by dilation of the ascending aorta, the murmur is often more readily audible along the right sternal border

# Auscultation

- An **Austin-Flint murmur** may be present at the cardiac apex in severe AR; it is a low-pitched, mid-diastolic rumbling murmur due to blood jets from the AR striking the anterior leaflet of the mitral valve, which results in premature closure of the mitral leaflets.
- $A_2$  may be normal or accentuated when AR is caused by disease of the aortic root but is soft or absent when the valve is causing AR.  $P_2$  may be obscured by the early diastolic murmur.

# Auscultation

**third heart sound ( $S_3$ )** correlates with an increased LV end-diastolic volume. Its development may be a sign of impaired LV function, which is useful in identifying patients with **severe AR** who are candidates for surgical treatment.

- The auscultatory features of AR are intensified by strenuous and sustained handgrip, which augments systemic vascular resistance.

# Peripheral Signs

**Becker sign** - Visible systolic pulsations of the retinal arterioles

**Corrigan pulse ("water-hammer" pulse)** - Abrupt distention and quick collapse on palpation of the peripheral arterial pulse

**de Musset sign** - Bobbing motion of the patient's head with each heartbeat

**Hill sign** - (Popliteal cuff systolic blood pressure 20 mm Hg higher than brachial cuff systolic blood pressure) is an **artifact** of sphygmomanometric measurements and is no longer considered a sign of severe AR.

**Duroziez sign** - Systolic murmur over the femoral artery with proximal compression of the artery, and diastolic murmur (Duroziez murmur) over the femoral artery with distal compression of the artery

**Müller sign** - Visible systolic pulsations of the uvula

**Light-house sign** - Alternate flushing & blanching of forehead

**Landolfi's sign** - Change in pupil size with each systole

**Gerhardt's sign** - Visible systolic pulsations of spleen

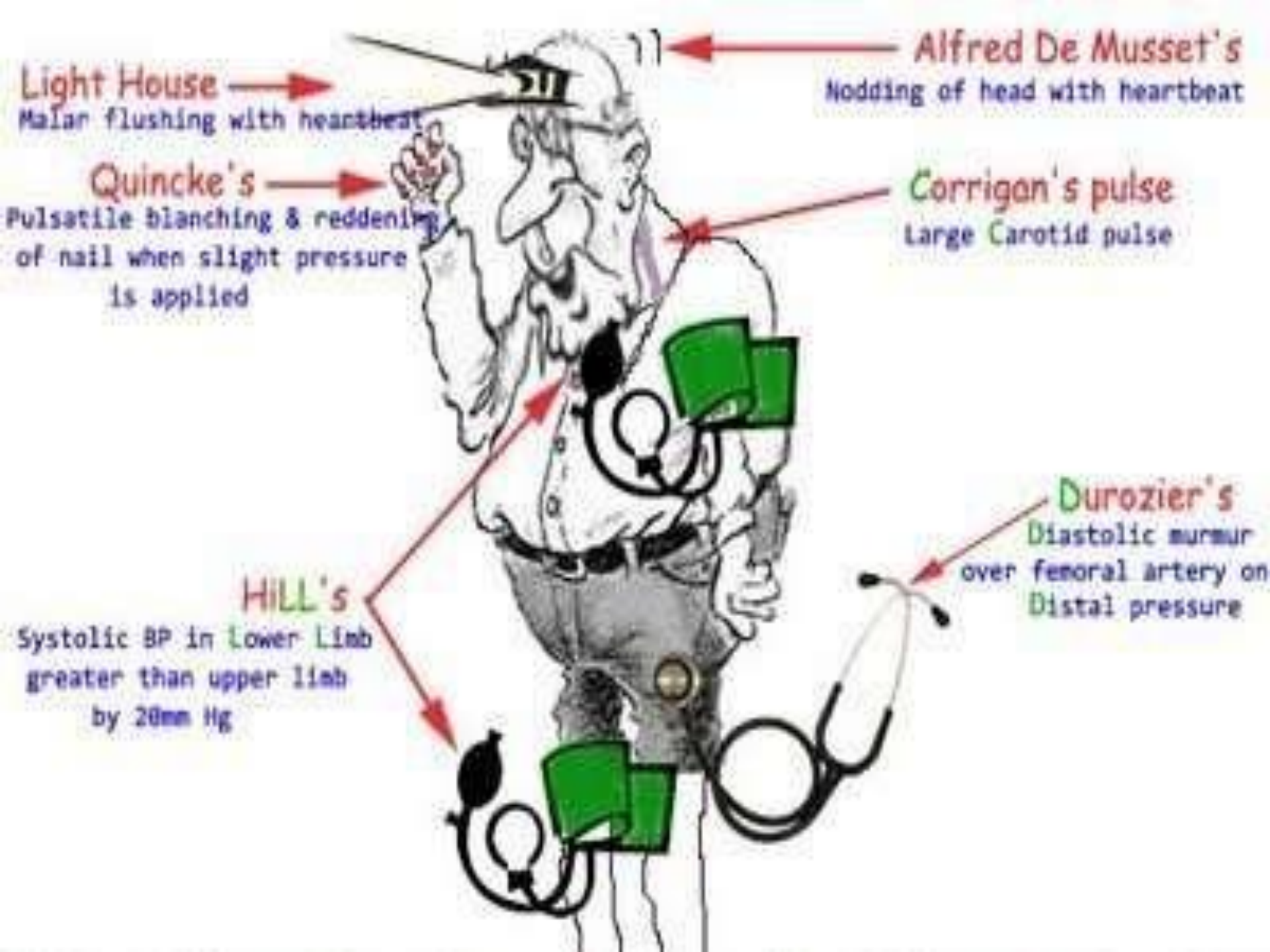
**Rosenbach's sign** - Visible systolic pulsations of liver

**Corrigan's sign** - Dancing carotid in neck

**Quincke sign** - Visible pulsations of the fingernail bed with light compression of the fingernail

**Traube sign ("pistol-shot" pulse)** - Booming systolic and diastolic sounds auscultated over the femoral artery

- **A bisferiens pulse** in AS + AR - readily recognized in the brachial and femoral arteries than in the carotid arteries. (Two systolic waves separated by a trough)



**Light House** →  
Malar flushing with heartbeat

**Quincke's** →  
Pulsatile blanching & reddening  
of nail when slight pressure  
is applied

→ **Alfred De Musset's**  
Nodding of head with heartbeat

→ **Corrigan's pulse**  
Large Carotid pulse

**HILL's** →  
Systolic BP in lower limb  
greater than upper limb  
by 20mm Hg

→ **Durozier's**  
Diastolic murmur  
over femoral artery on  
Distal pressure

# Assessing Severity of AR

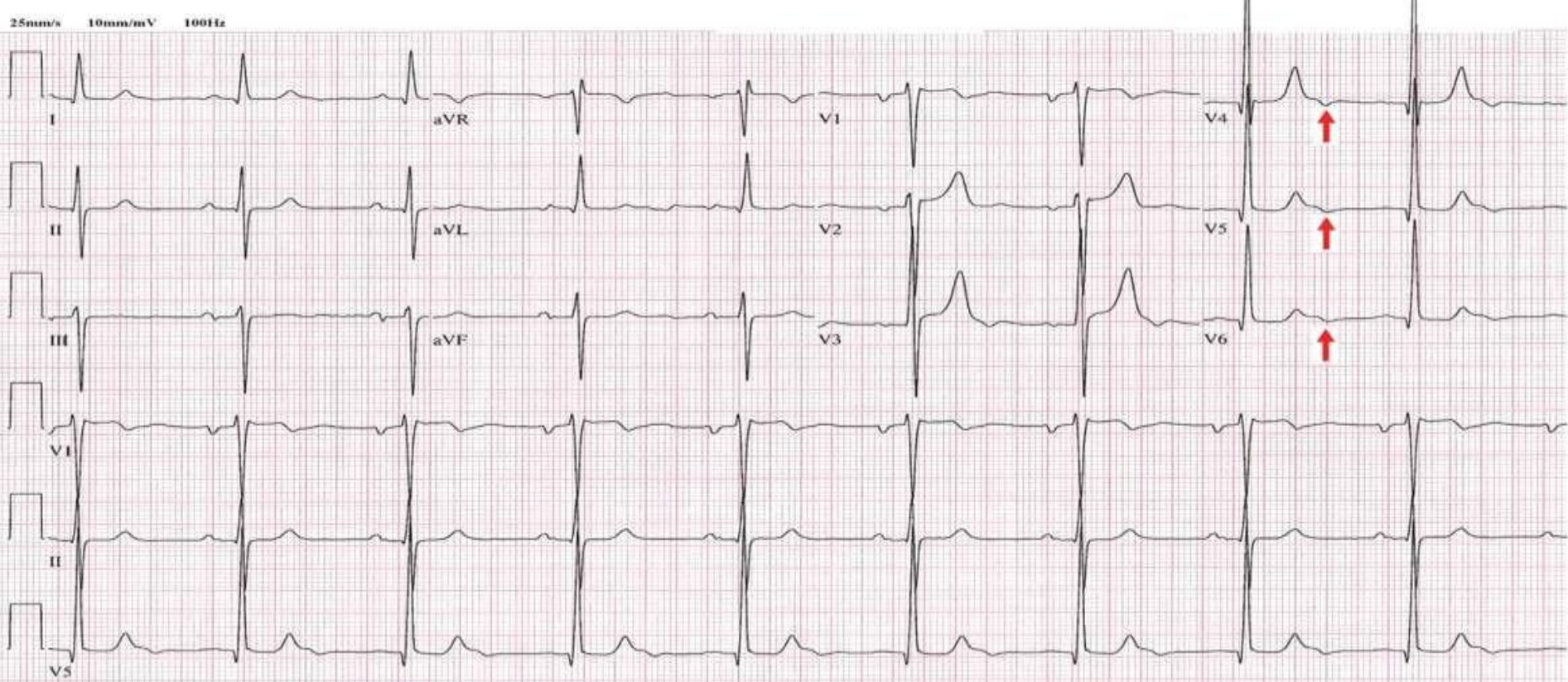
- Assess severity by impact on peripheral signs and LV
    - peripheral signs positive = severity
    - LV Dysfunction S3 present= severity
    - 
    - Long duration of diastolic murmur= severity
    - Austin -Flint murmur= severity
    - LVH
    - radiological cardiomegaly
- 

# Differential Diagnosis

- Hyperdynamic circulation (wide pulse pressure), including the following:
    - Thyrotoxicosis
    - Severe anemia
    - Pregnancy
    - Thiamine deficiency (wet beriberi)
    - Arteriovenous fistula - Such as patent ductus arteriosus or peripheral arteriovenous malformations
    - Volume depletion
    - Sympathetic overdrive
- 

# ECG

- LV hypertrophy
  - Left axis deviation
  - Left atrial enlargement
  - LV volume overload pattern - Prominent Q waves in leads I, aVL, and V<sub>3</sub> to V<sub>6</sub> and relatively small r waves in V<sub>1</sub>
  - LV conduction defects - Typically late in the disease process
  - poor prognosis.
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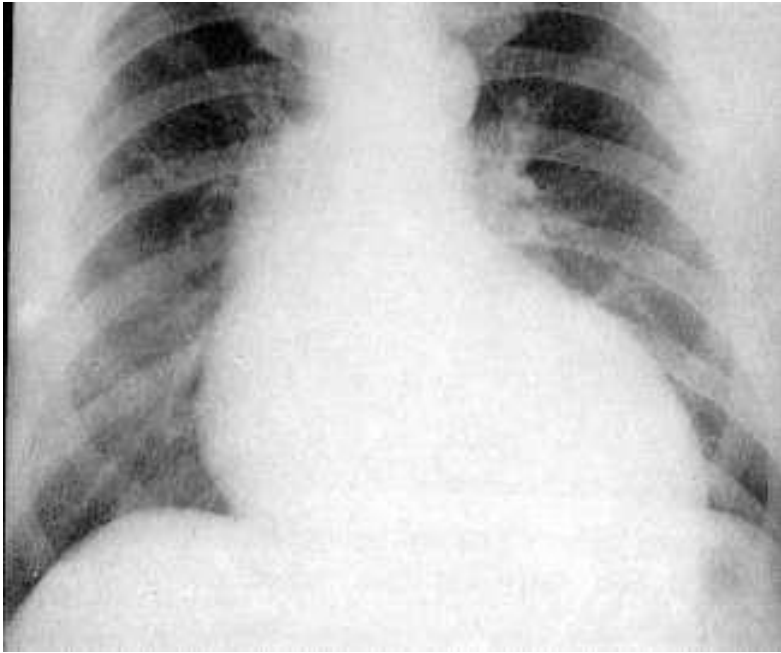


The patient's 12-lead electrocardiogram shows normal sinus rhythm and a rate of 55 beats per minute. The frontal-plane QRS complex vector is deviated leftward. Left atrial abnormality is present, given the terminally negative P wave in lead V<sub>1</sub> and the bifid P wave in lead II. Left ventricular volume overload is supported by the following findings: increased QRS complex voltage, best seen in the precordial (chest) leads, indicative of increased left ventricular mass;

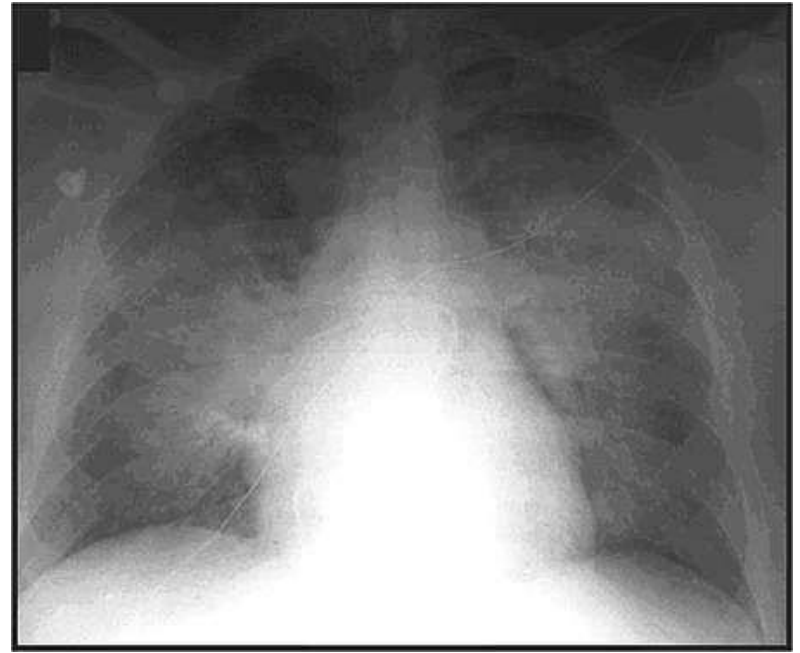
# Chest X-Ray

- In acute AR, there may be minimal cardiac enlargement, but marked enlargement is a common finding in chronic AR.
- The apex is displaced downward and to the left in the frontal projection. In the left anterior oblique and lateral projections, the LV is displaced posteriorly and encroaches on the spine.
- Distinct left atrial enlargement in the absence of heart failure suggests associated mitral valve disease.
- When AR is caused by primary disease of the aortic root, aneurysmal dilation of the aorta may be noted, and the aorta may fill the retrosternal space in the lateral view.

# Aortic Regurgitation CxR



LVE with normal  
pulmonary vasculature



Normal size LV with pulmonary  
vascular congestion

# Echocardiography

- Aortic valve structure and morphology - Bileaflet versus trileaflet, flail, thickening

Presence of vegetations or nodules - May require transesophageal echocardiography in selected cases

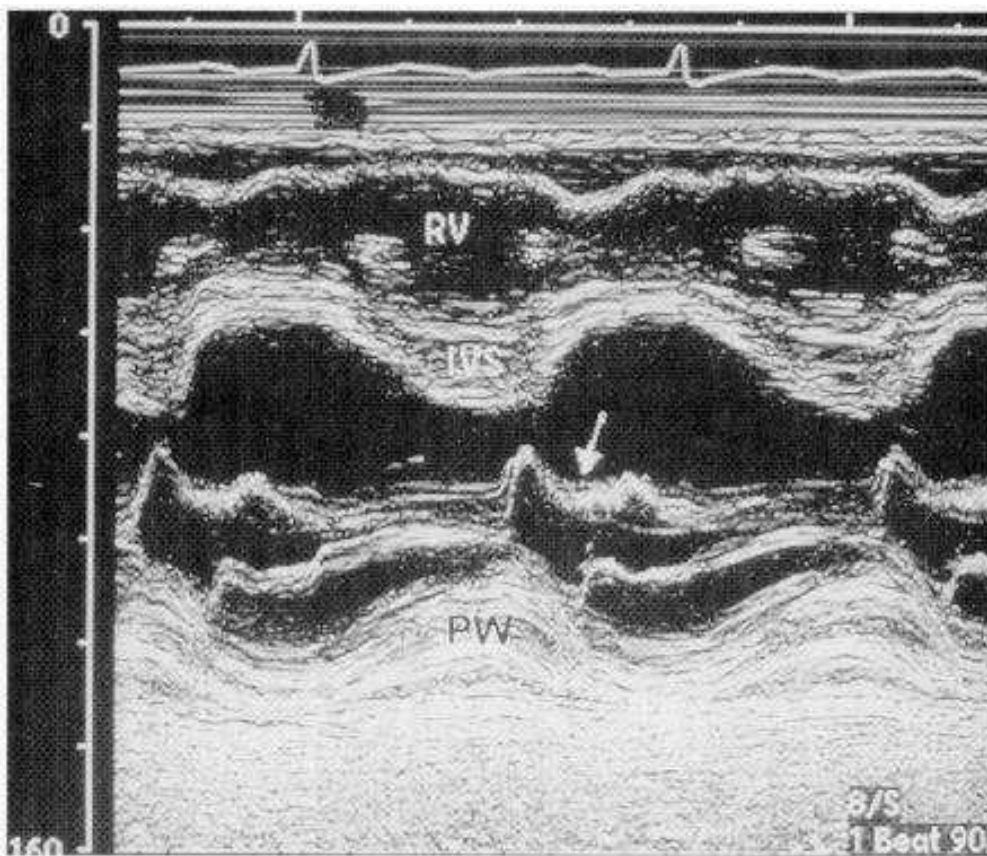
- Severity of AR
  - Color Doppler central jet width & Vena contracta (narrowest portion of the jet located at or just distal to its orifice) width -
  - In severe AR, the vena contracta width is usually more than 65% of the width of the LV outflow tract
  - Regurgitant volume, fraction, and orifice area is calculated
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## Echocardiographic stages of chronic aortic regurgitation

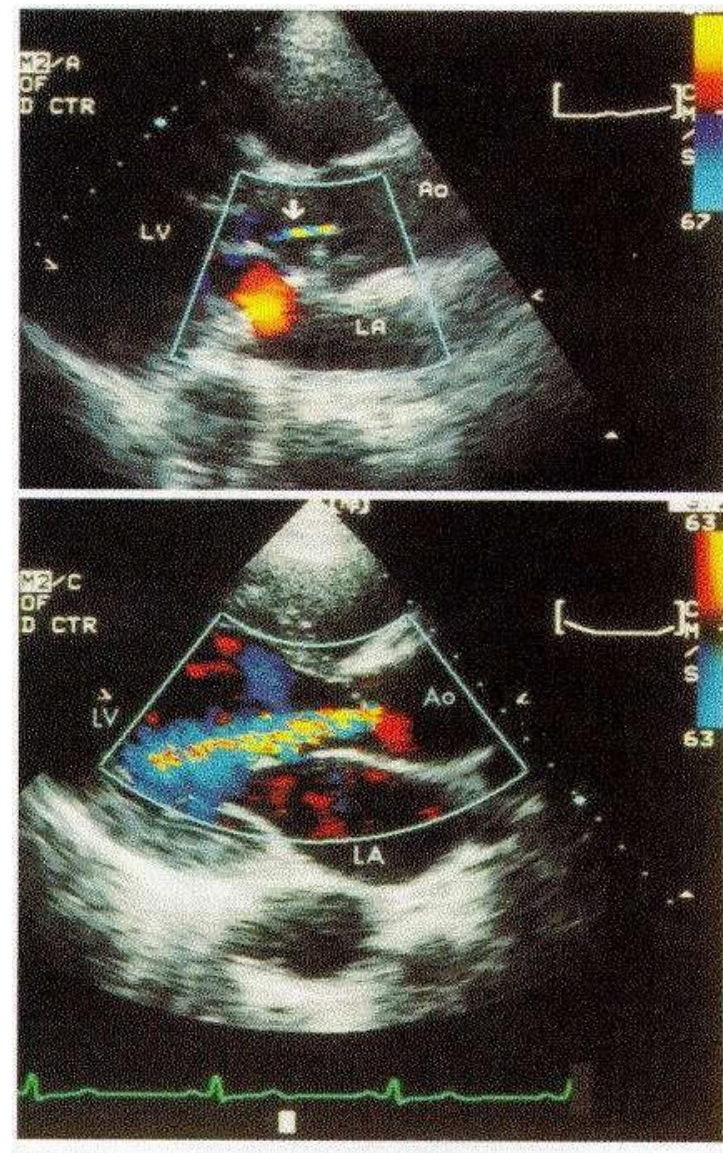
|                                           | Compensated | Transitional | Decompensated* |
|-------------------------------------------|-------------|--------------|----------------|
| <b>Dimensions</b>                         |             |              |                |
| End-diastolic dimension (mm)              | <60         | 60 to 70     | >75            |
| End-systolic dimension (mm)               | <45         | 45 to 50     | >55            |
| <b>Volumes</b>                            |             |              |                |
| End-diastolic volume (mL/m <sup>2</sup> ) | <120        | 130 to 160   | >170           |
| End-systolic volume (mL/m <sup>2</sup> )  | <50         | 50 to 60     | >60            |
| <b>Left ventricular function</b>          |             |              |                |
| Ejection fraction (percent)               | >55         | 51 to 55     | ≤50            |
| Fractional shortening (percent)           | >32         | 30 to 31     | <29            |

Patients with intermediate values between transitional and decompensated disease (end-diastolic dimension 70 to 75 mm and end-systolic dimension 50 to 55 mm) can be referred for exercise testing. Those with an abnormal response are referred for valve replacement, while those with a normal response to exercise are closely monitored.

\* Patients with decompensated aortic regurgitation are referred for aortic valve replacement. Lower threshold values can be considered in patients of small stature.



AMVL fluttering

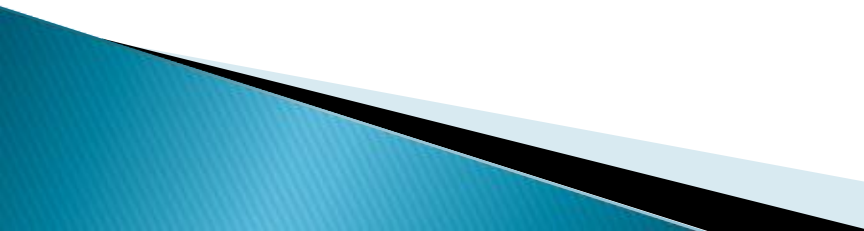


Flow – top mild, bottom moderate

# Cardiac MRI

- CMR provides accurate measurements of regurgitant volumes and the regurgitant orifice in AR. It is the most accurate noninvasive technique for assessing LV end-systolic volume, diastolic volume, and mass

# Management – Acute AR

- **Prompt surgical intervention** is indicated
  - Administer a **positive inotrope** (eg, dopamine, dobutamine) and a vasodilator (eg, nitroprusside).
  - Administration of **vasodilators** may be appropriate to improve systolic function and to decrease afterload.
  - The administration of cardiac glycosides (eg, digoxin) for rate control may in rare cases be necessary.
  - **Beta-blocking agents and intra-aortic balloon counterpulsation are contraindicated**, because either lowering the heart rate or augmenting peripheral resistance during diastole can lead to rapid hemodynamic decompensation.
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# Management – Chronic MR

- The current ACC/AHA guidelines say the following about **vasodilator therapy (Nifedipine, ACE Inhibitors)**:

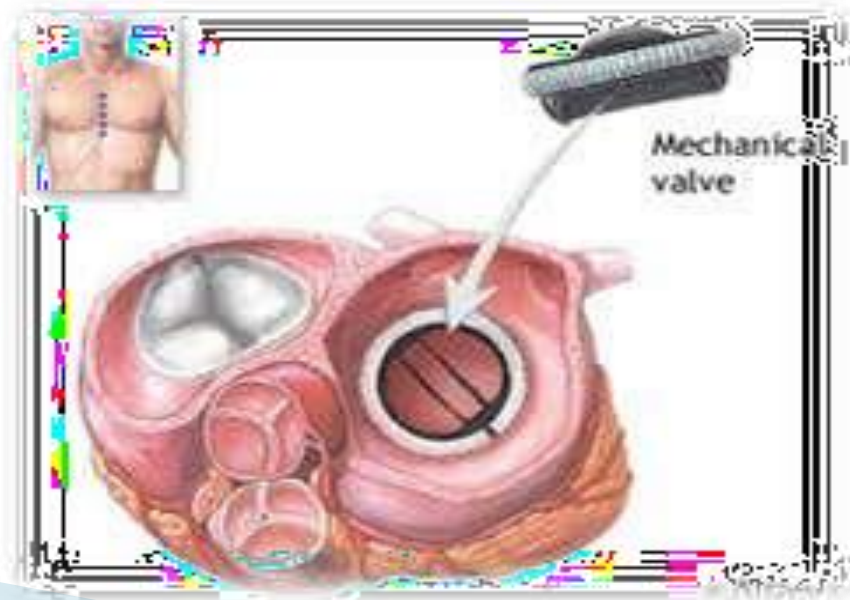
Vasodilator therapy is indicated for **long-term** treatment in patients who have **severe chronic AR and symptoms of LV dysfunction** but who are not candidates for surgery.

- Vasodilator therapy is reasonable for **short-term** therapy in patients with **severe LV dysfunction and heart failure symptoms**, in order to improve their hemodynamic profile before surgery
  - Vasodilator therapy is acceptable for **long-term** therapy in **asymptomatic patients with severe AR and LV dilation with normal EF**
- (To control diastolic BP)

- Chronic medical therapy for some patients who refuse surgery or are considered to be inoperable because of comorbid conditions - These patients should receive an aggressive heart failure regimen with **ACE inhibitors (and perhaps other vasodilators), digoxin, diuretics, and salt restriction**; beta blockers may also be beneficial
- Although nitroglycerin and long-acting nitrates are not as helpful in relieving anginal pain as they are in patients with ischemic heart disease, they are worth a trial.
- **Beta blockers (contraindicated in Valvular AR)** and the angiotensin receptor blocker, may be useful to retard the rate of aortic root enlargement in young patients with Marfan's syndrome and aortic root dilation.
- Avoid isometric exercises
- Avoidance of atrial fibrillation and bradycardia are important as these are poorly tolerated

# Current ACC/AHA guideline for AV Sx

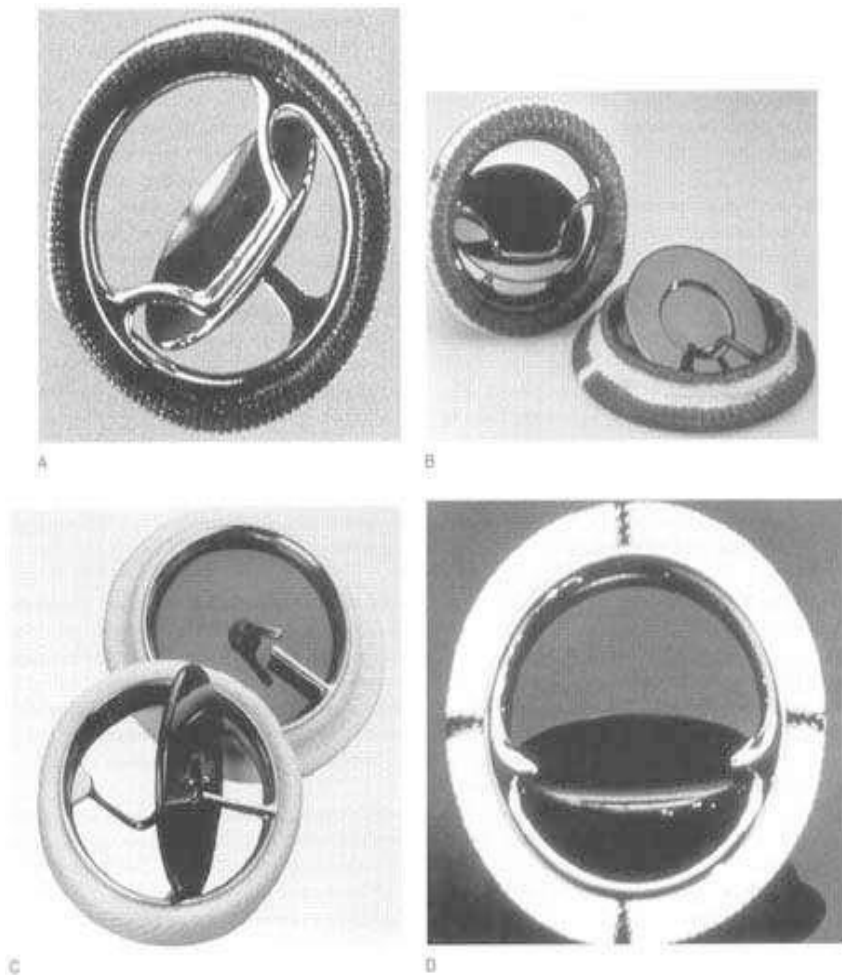
- Patient is symptomatic.
- Patient is asymptomatic, with a resting EF of  $\leq 55\%$ .
- Patient is asymptomatic, with LV dilation (LV end-systolic dimension  $>55$  mm).



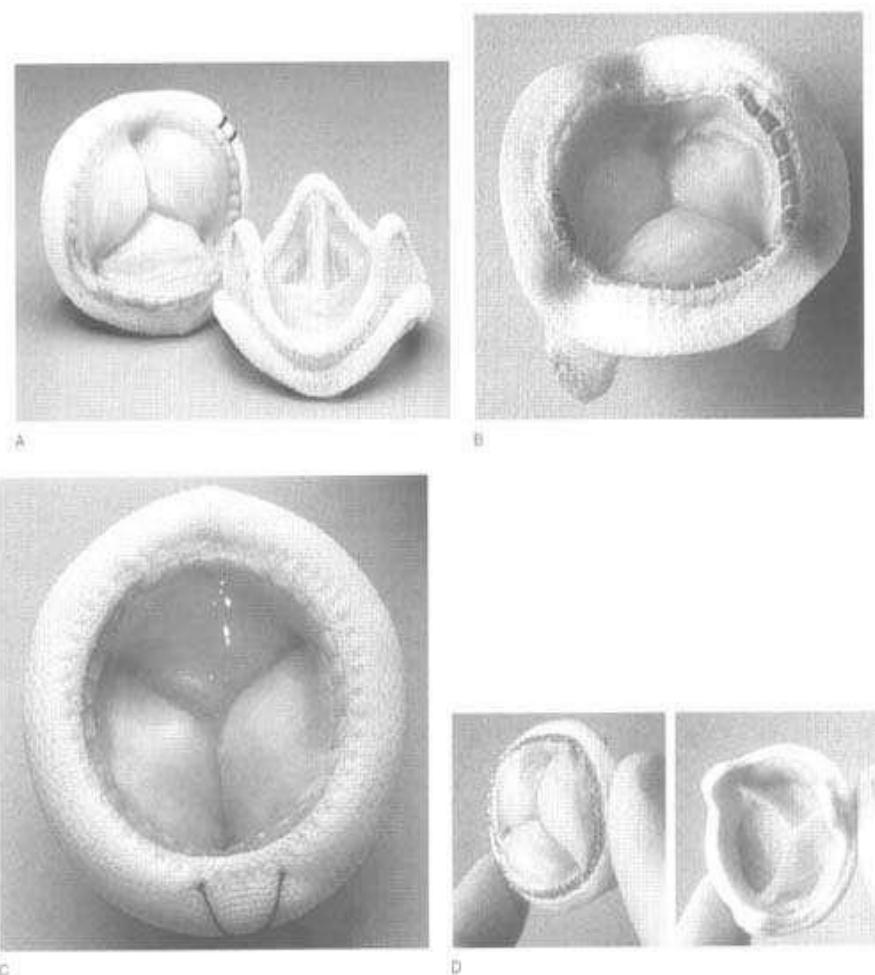
# Surgical treatment – ACC/AHA guideline summary

**Class I - There is evidence and/or general agreement that aortic valve replacement or repair (AVR) is indicated in patients with chronic AR in the following settings**

- Symptomatic patients with severe chronic AR, irrespective of left ventricular ejection fraction (LVEF).
- If the presence of symptoms in patients with severe chronic AR is equivocal, the development of symptoms during an exercise test.
- Asymptomatic patients with severe chronic AR and an LVEF  $\leq 50$  percent at rest.



A. Björk-Shiley Monostrut mechanical prosthesis. B. Sorin Allcarbon monoleaflet mechanical prosthesis. C. Medtronic-Hall mechanical prosthesis. D. Omnicarbon mechanical prosthetic porcine bioprosthesis.



**Figure 29-16** A. Carpentier-Edwards Supra-annular porcine bioprosthesis. B. Hancock II porcine bioprosthesis. C. Hancock modified orifice porcine bioprosthesis. D. St. Jude

# Transcatheter aortic valve replacement

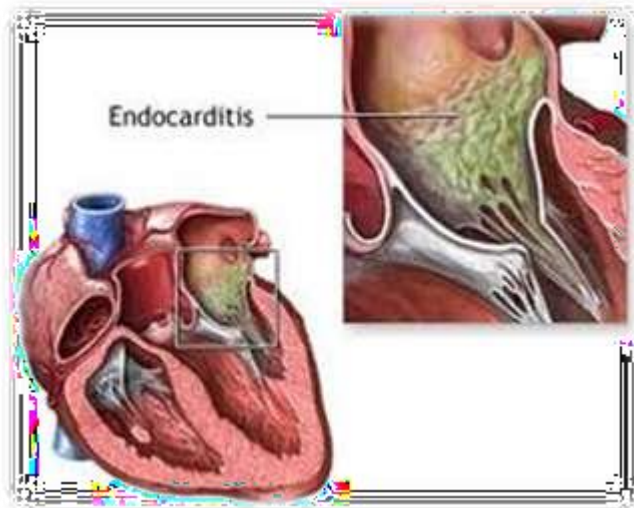
- TAVR involves the implantation of a bioprosthetic aortic valve using a catheter that is inserted peripherally, typically through the femoral artery, and implanted without requiring a median sternotomy (ie, without open heart surgery). It is being used commonly now in suitable patients.
- If MS + AR, first go for AR as correcting MS will increase LV load  $\rightarrow$  LVF.

# Prognosis

- Death usually occurs within 4 yrs after onset of angina pectoris and within 3 yrs of onset of heart failure
  - It is imperative to intervene before irreversible LV dysfunction occurs.
  - After AVR, Long term results reveal 80-90% three year survival in patients with preserved LV function, and 40- 60% three year survival in patients with poor LV function.
  - Mortality rate of AVR ranges from 3-8%
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# Prophylaxis for IE

- Antibiotic prophylaxis prior to dental procedures is **no longer routinely recommended** for all patients with AR under current ACC/AHA guidelines.<sup>[1]</sup>



*Thank You*