

Valvular Aortic Heart Diseases

WE'VE REVIEWED THE ECG, CXR,
ECHO, HOLTER, NUCLEAR SCAN,
ETC. ETC.... AND STILL
NO DIAGNOSIS!

THINK WE SHOULD BREAK
DOWN AND DO A HISTORY
AND PHYSICAL EXAM?



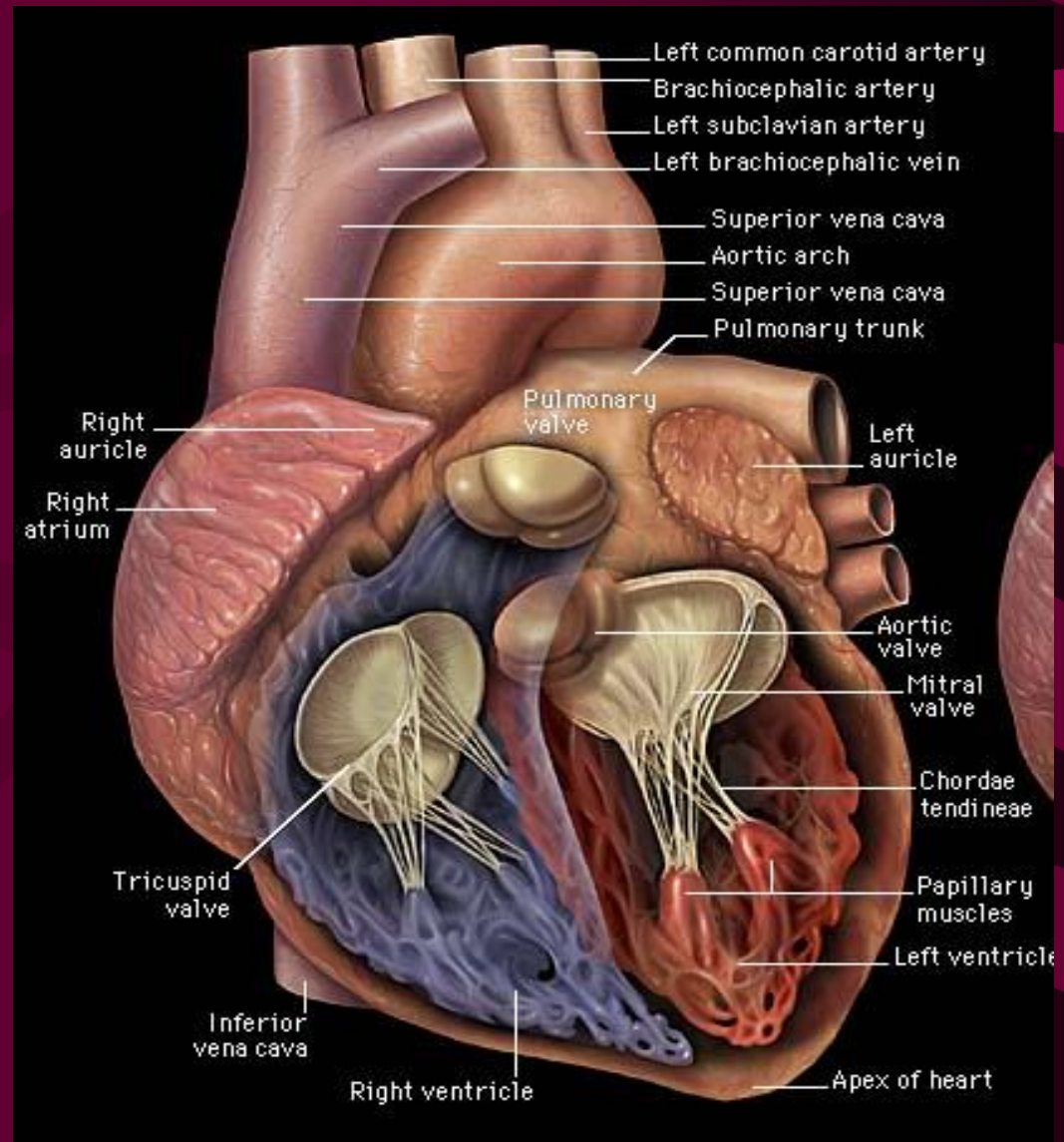
Spectrum of valve heart diseases

Aortic Valve

Mitral Valve

Tricuspid Valve

Pulmonic Valve



Spectrum of valve heart diseases

Aortic Valve

Regurg

Stenosis

Mitral Valve

Regurg

Stenosis

Tricuspid Valve

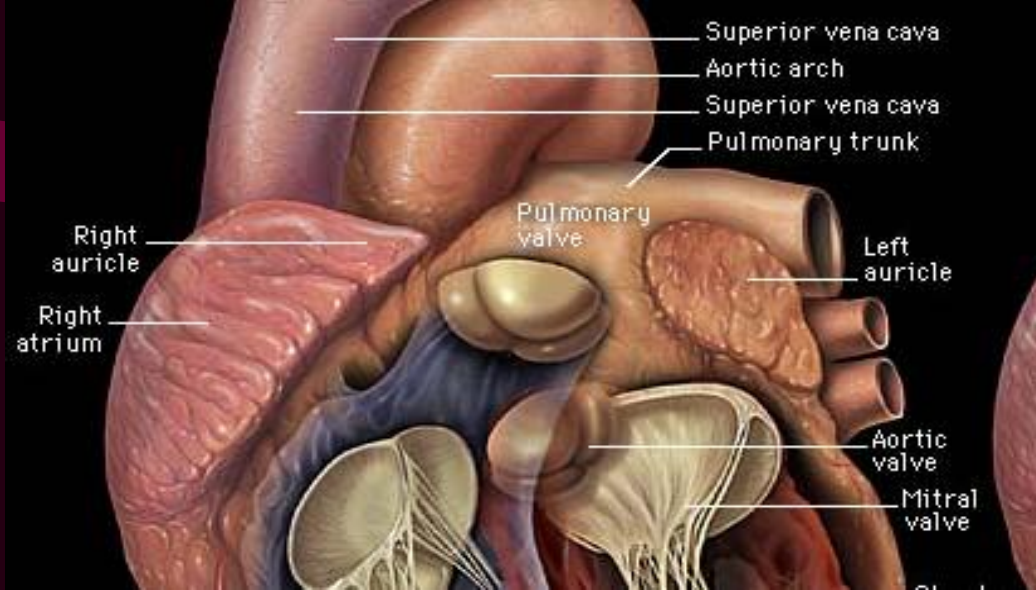
Regurg

Stenosis

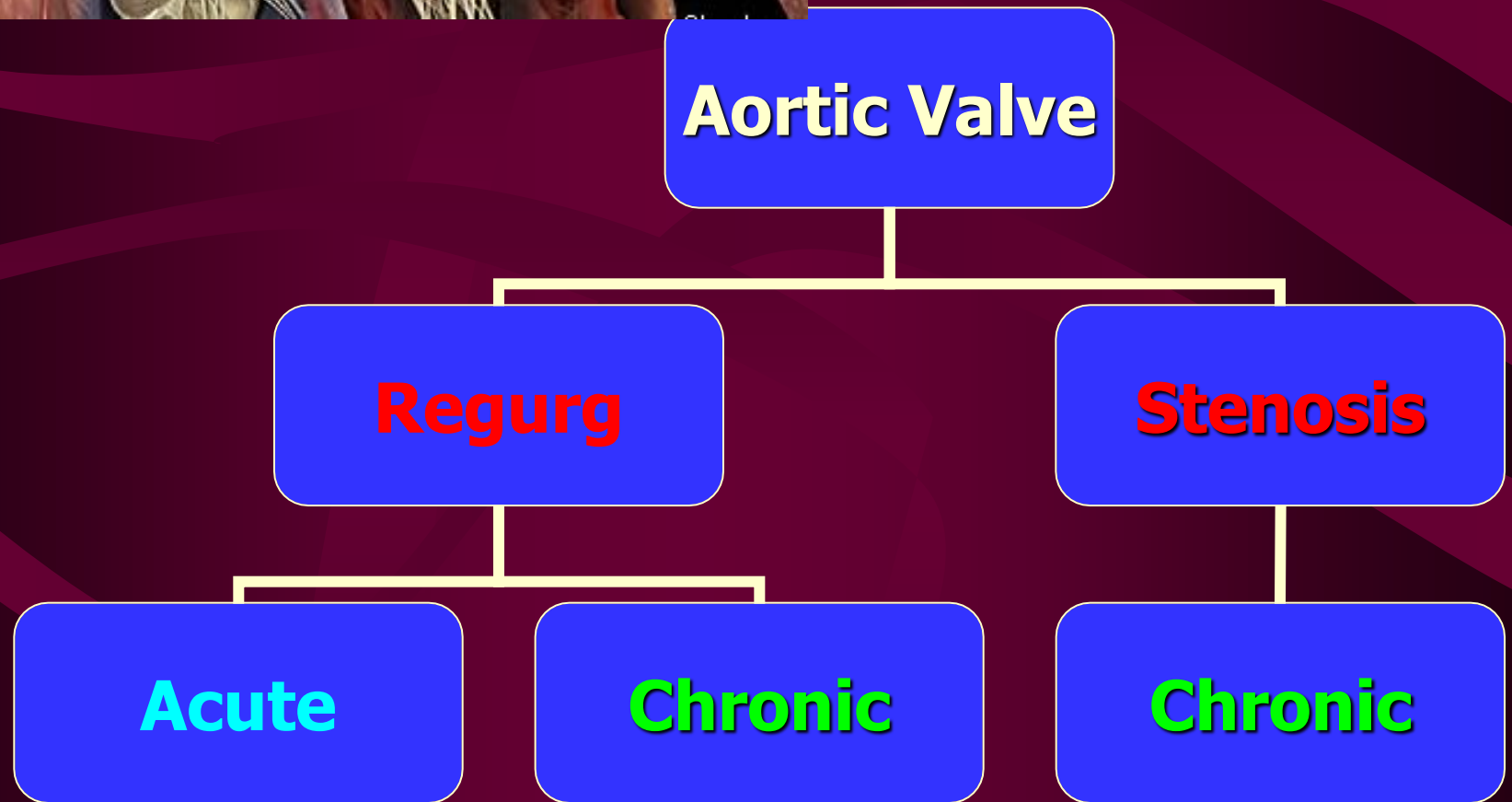
Pulmonic Valve

Regurg

Stenosis



Spectrum of aortic valve diseases





Eric Petersen / The Livingston Enterprise

Cardiac Physiology

Systole

S1-S2

AV – opens

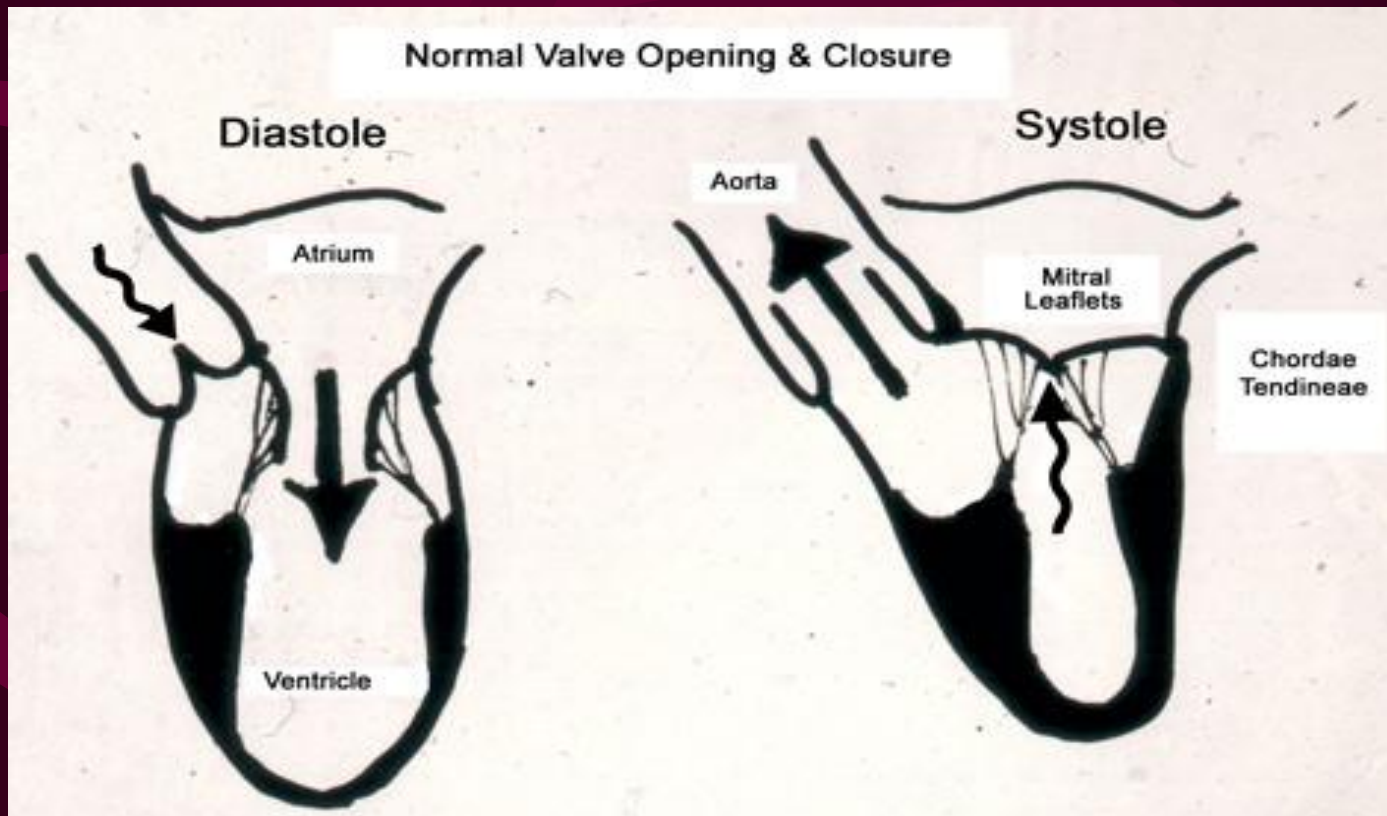
MV – closes

Diastole

S2-S1

AV – closes

MV – opens



Cardiac Physiology

(murmurs)

Stenosis – Obstruction of (**forward**) flow across an **opened** valve

Regurg/ Insuff – leaking (**backflow**) of blood across a insufficient **closed** valve

Systole

S1-S2

AV – opens----- Aortic Stenosis

MV – closes----- Mitral Regurg

Diastole

S2-S1

AV – closes----- Aortic Regurg

MV – opens----- Mitral Stenosis

*These concepts are set in stone, it can't occur any other way,
It would be anatomically impossible*

Aortic Valve

- ***Aortic Stenosis (AS)*** –

obstruction of **blood** flow across the *opened*
aortic valve in systole

- ***Aortic Regurgitation***

Aortic Stenosis

Etiologies

- ***Congenital*** ***0-30 yrs***
- ***Bicuspid*** ***30-50 yrs***
- ***Rheumatic*** ***30-60 yrs***
- ***Degenerative*** ***>60 yrs***

A rheumatic etiology of AS is favored by a history of active *rheumatic fever*.

Morphopathology

Rheumatic changes

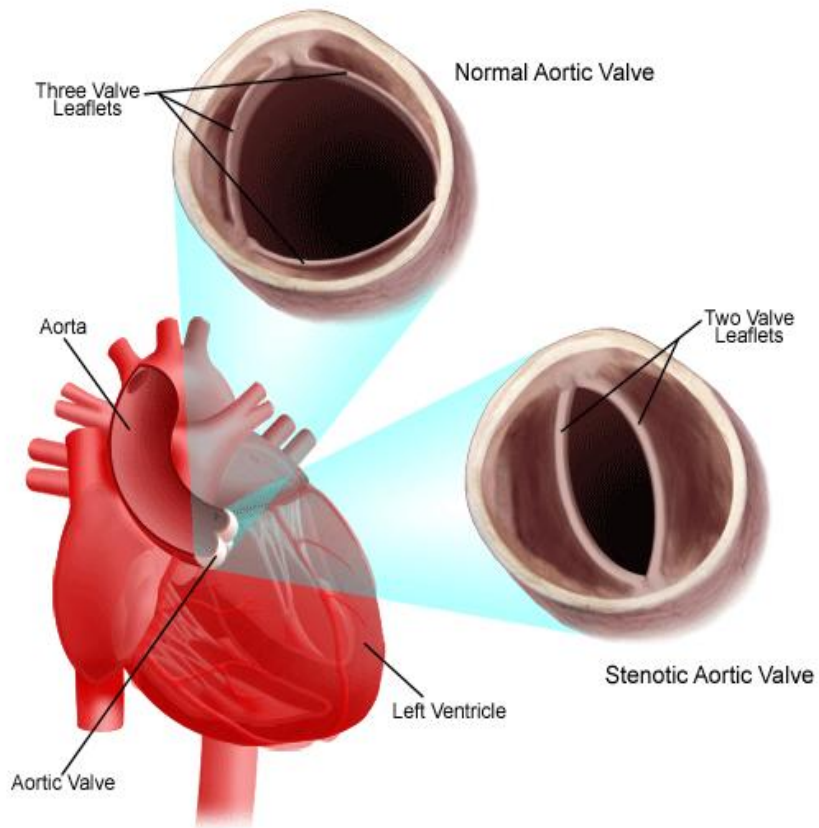
- commissural fusion, sometimes resulting in a bicuspid valve.
- leaflets are more susceptible to trauma and ultimately leads to fibrosis, calcification, and further narrowing.

Morphopathology

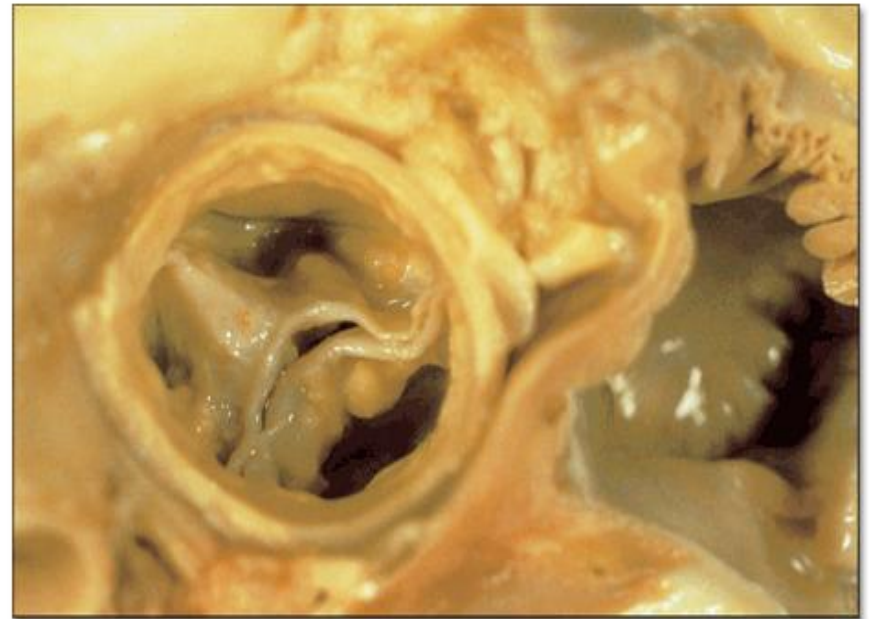
- abnormal architecture of the congenitally deformed valve makes its leaflets susceptible to hemodynamic stresses, which lead to valvular thickening, calcification and narrowing of the aortic orifice.

Aortic Stenosis

An Example of Aortic Stenosis



Bicuspid Aortic Valve Stenosis



Etiologie

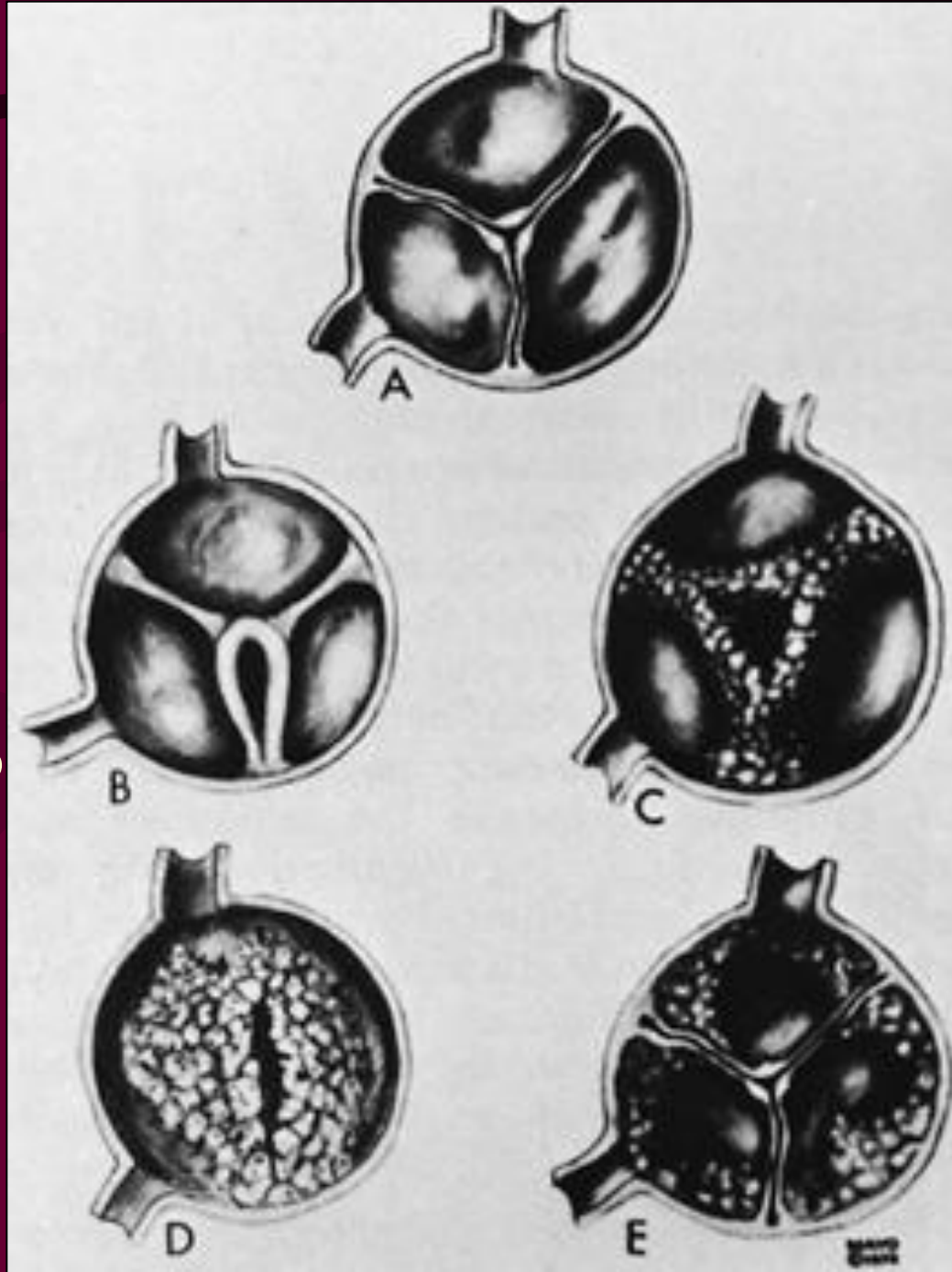
A. Normal Ao valve

B. Congenital AS

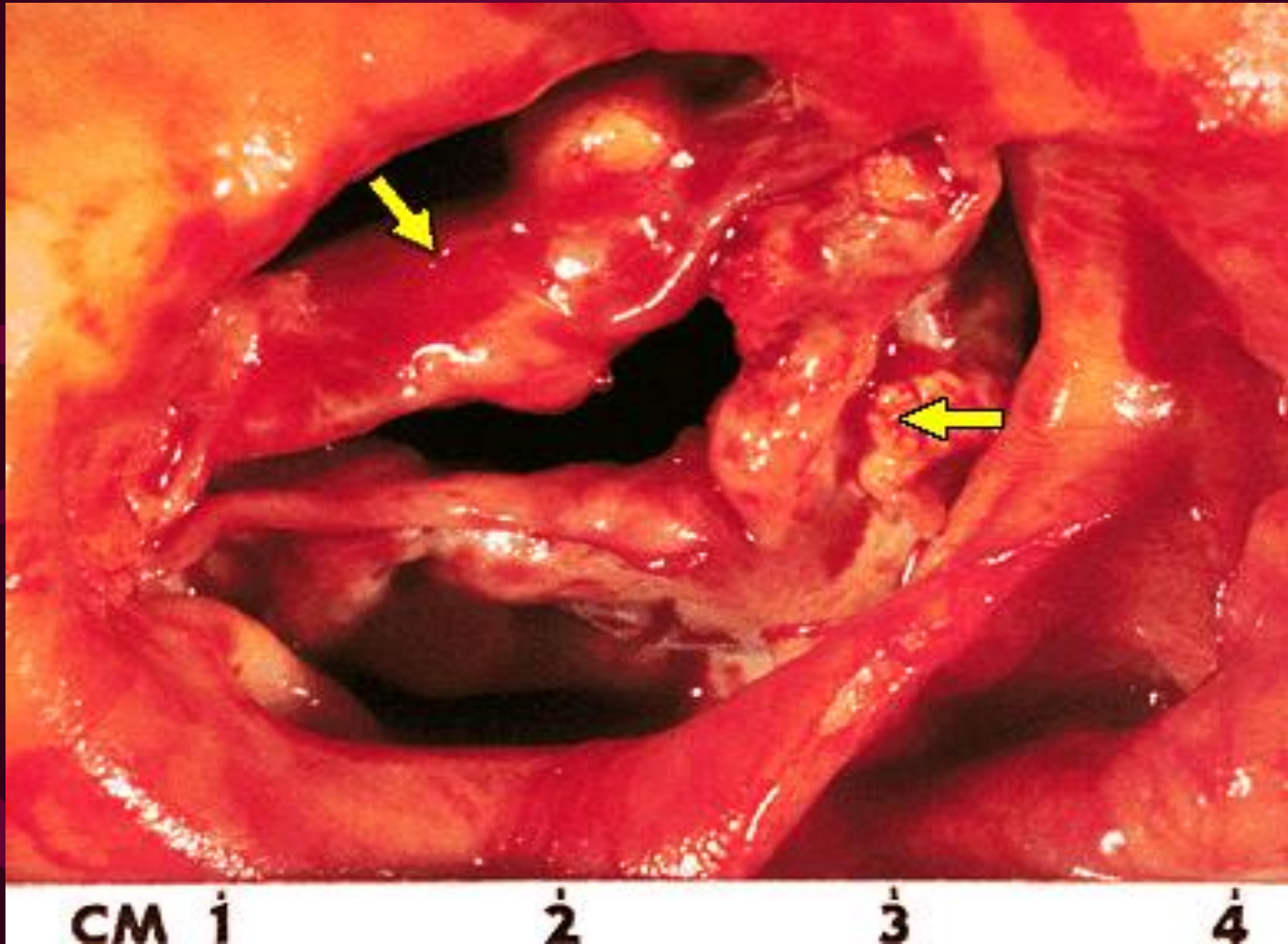
C. Rheumatic AS

D. Calcific AS

E. Degenerative AS

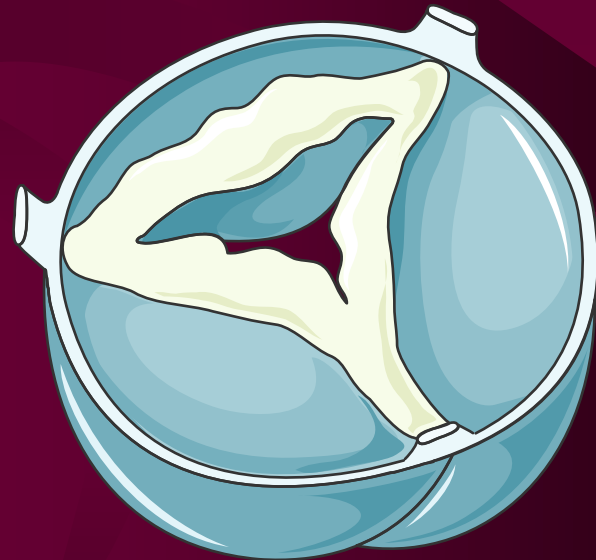
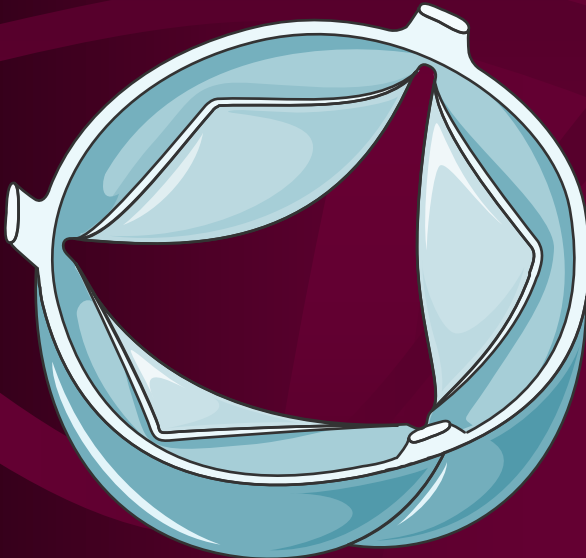


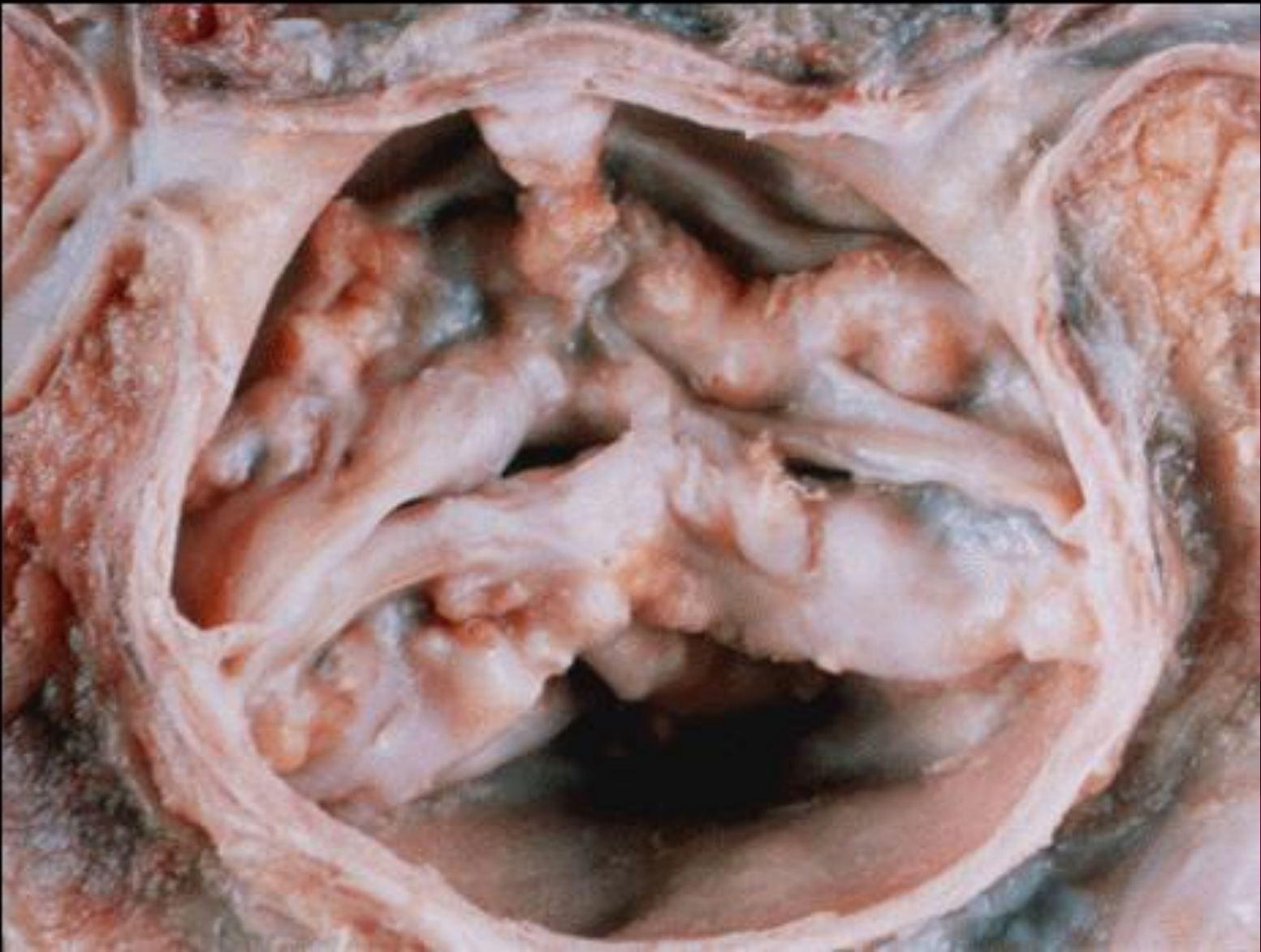
Calcific aortic stenosis



Fibro-calcific Degeneration

- the most common cause of AS in adults in America and Europe.



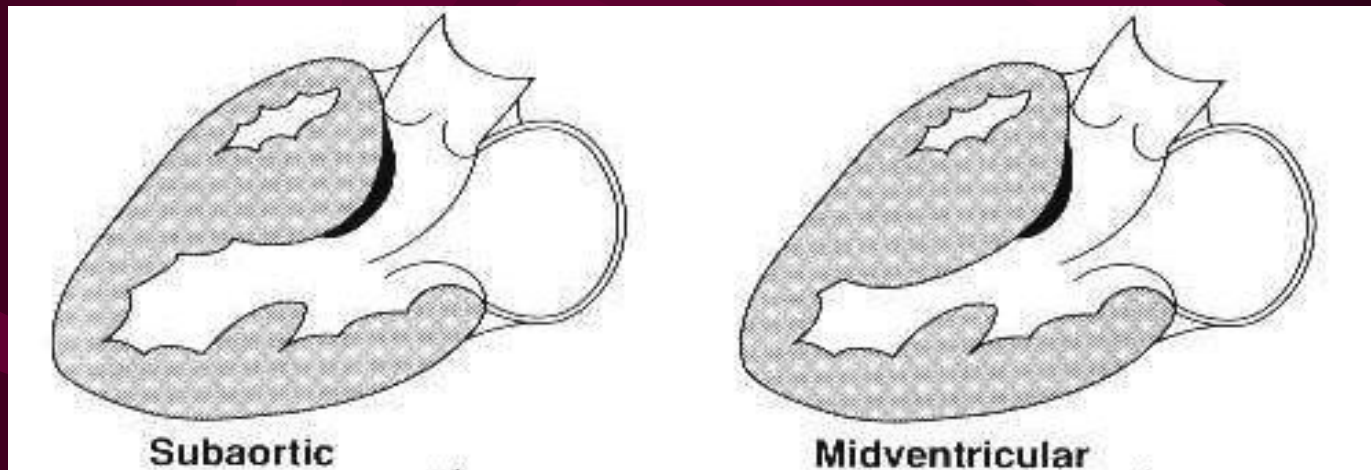
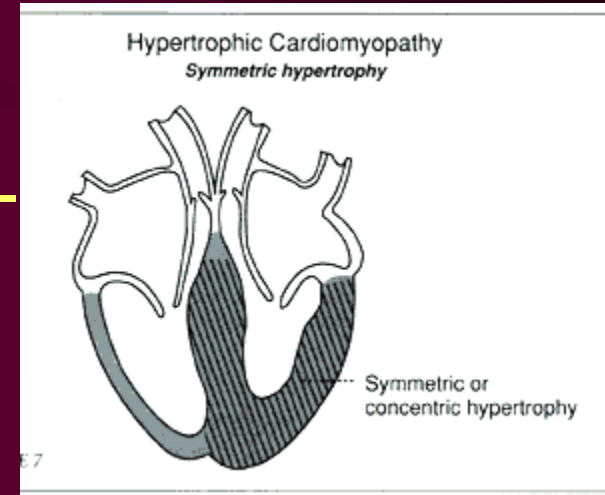


- Often is difficult to determine the etiology of the underlying process.

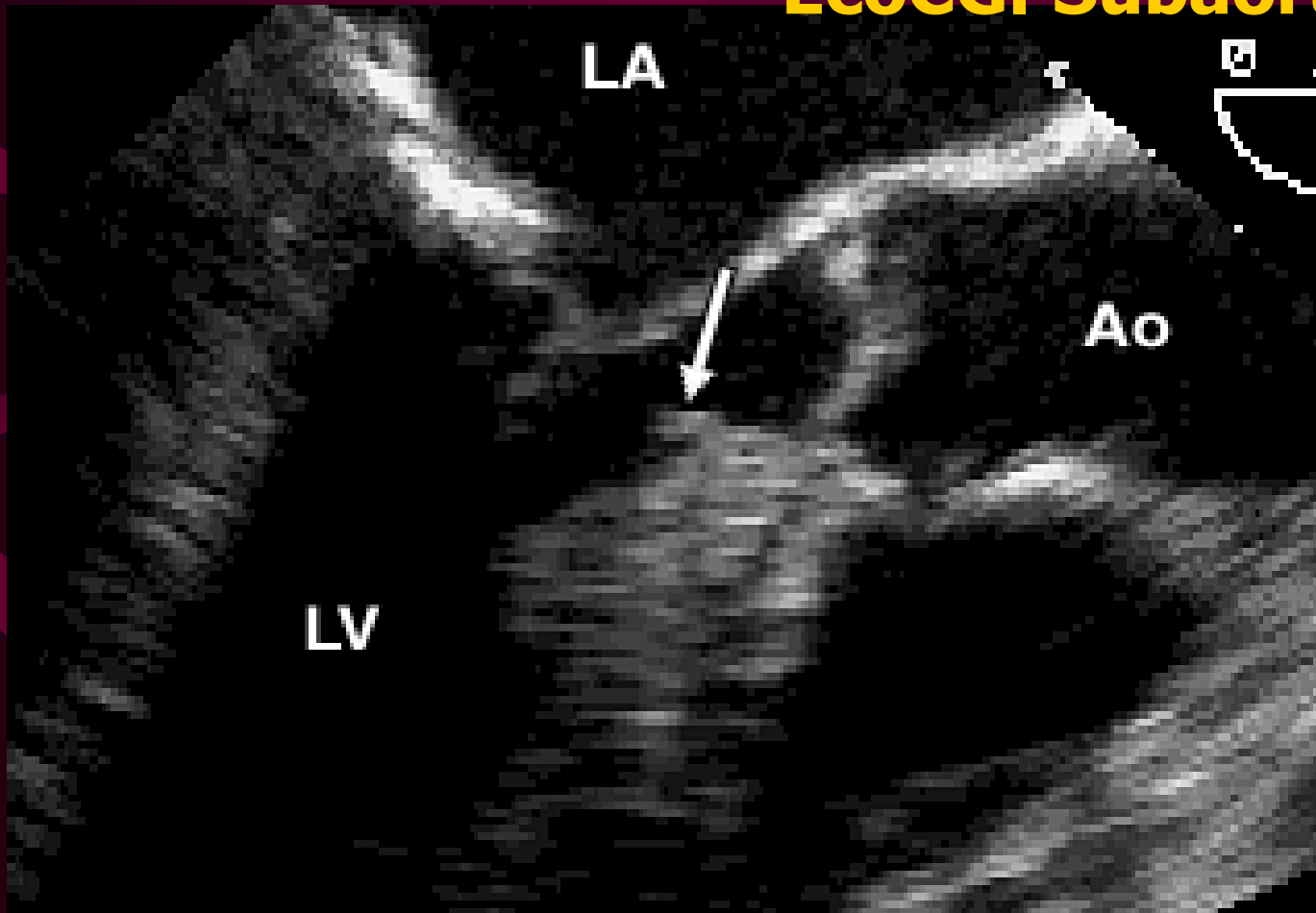
Other forms of AS

1. Subvalvular obstruction, met in :

- Hypertrophic cardiomyopathy -
 - marked hypertrophy of the LV which involves interventricular septum.



- **Congenital anomaly :**
a membranous diaphragm below the
aortic valve or a fibrous ridge just below
the aortic valve. **EcoCG. Subaortic membrane**



2. Supravalvular AS - a congenital anomaly - is produced by:

- narrowing of the ascending aorta or
- a fibrous diaphragm with a small opening just above the aortic valve.

Aortic Stenosis

(normal = 2.4 - 3.0 cm²)

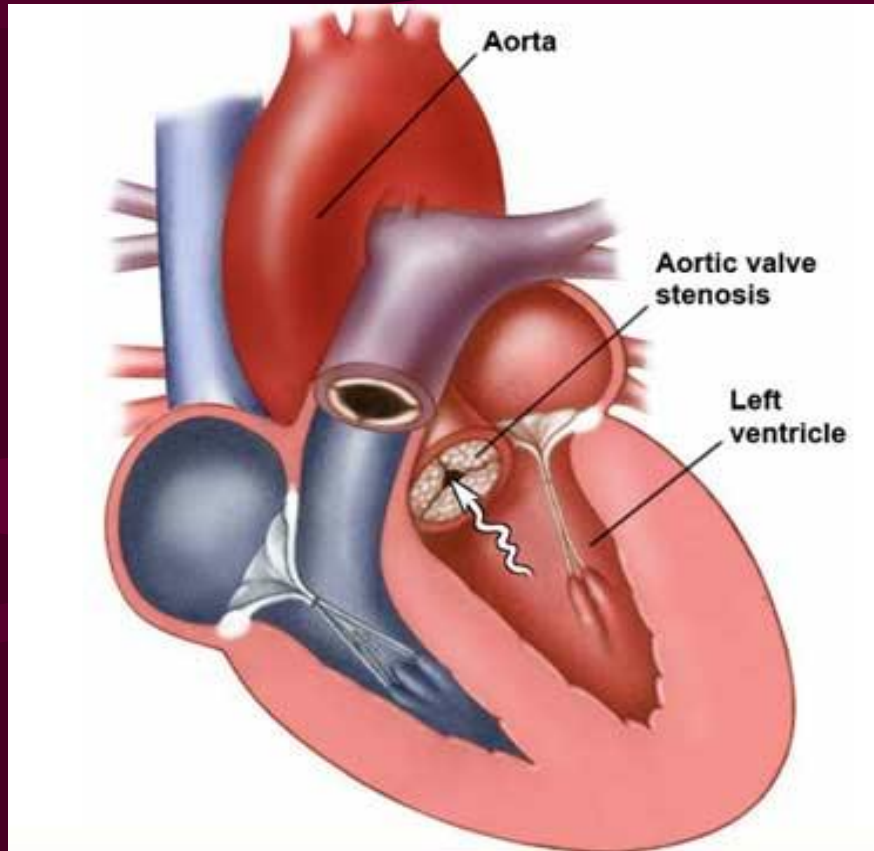
In general:

- Mild Aortic Stenosis = 1.5-2.0 cm²
- Moderate Stenosis = 1-1.5 cm²
- Severe Aortic Stenosis = <1.0 cm²
- Critical Aortic Stenosis = <0.8 cm² (1/3 of the normal)

EPIDEMIOLOGY of AS

- AS occurs in about 25 % of all patients with chronic valvular heart disease;
- About 30% of persons > 65 years exhibit aortic valve sclerosis,
- 2% of them exhibit frank stenosis.

Aortic Stenosis. PATHOPHYSIOLOGY(1)



1. AS produces an *obstruction to LV outflow in systole*;
2. Usually obstruction increases gradually - many years;
3. Obstruction to LV outflow causes an increased *systolic pressure gradient between the LV and aorta*;

AS. PATHOPHYSIOLOGY (2)



Normal



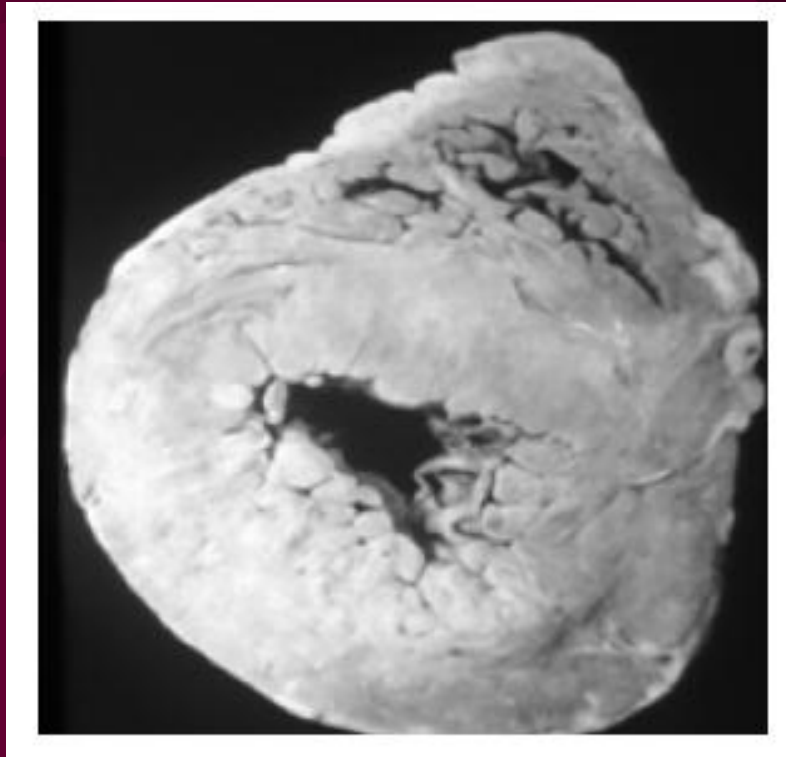
Overstraining of the LV

↓
Concentric
Hypertrophy

4. Systolic pressure gradient between the LV and aorta induce LV overstraining, which leads to concentric **Left Ventricle hypertrophy** (LVH);

AS. PATHOPHYSIOLOGY (3)

- 5. LVH is maintaining LV output.
- 6. LV **ejection time increase**, leading to bradycardia.

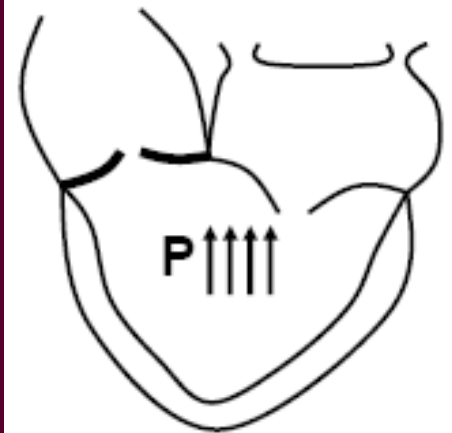


7. Late in the course, after a long period of compensation, LV decompensation occurs - **LV failure**.

8. LV failure first signs are the reduction of cardiac output (**CO↓**), **LV dilatation** and **↓ Ejection Fraction**.

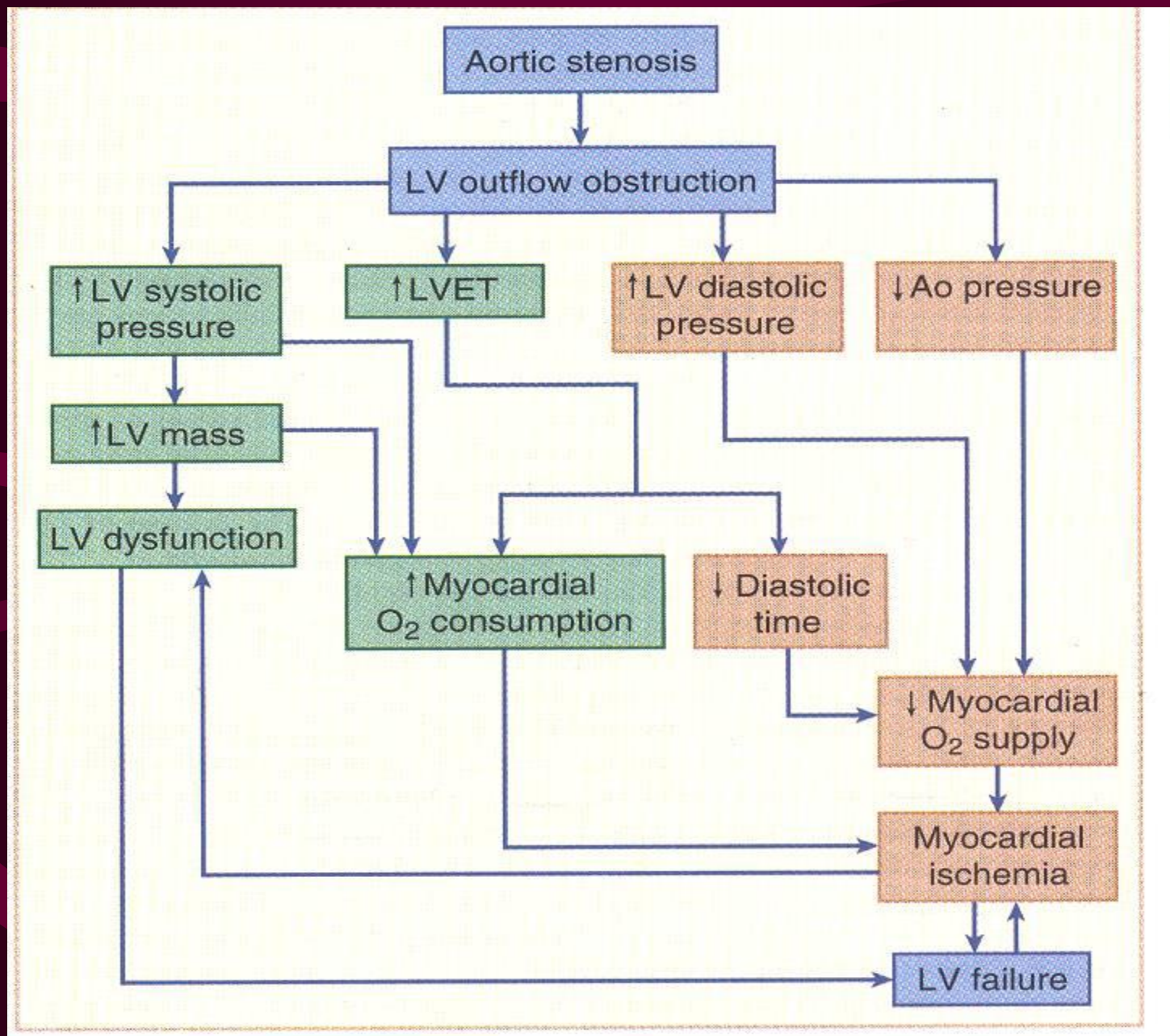
9. The CO decreases, and the following mean pressures rise :
LVEDp, LA pressure,
later - PA and RV pressures.

- **Concentric hypertrophy**
 - Reduces wall stress
 - Reduces ventricular compliance
 - LVEDp increases
 - LAp increases



AS. PATHOPHYSIOLOGY (4)

- A severe AS may exist for many years without complications and clinical signs.



Aortic Stenosis

Symptoms

- *Angina pectoris*
- *Syncope*
- *Congestive Heart Failure (CHF)*

NB !

- **Angina** - specific chest pain, caused by insufficient blood supply to myocardium through the coronary arteries,

Aortic Stenosis

Symptoms of Aortic Stenosis

- **Angina**
 - Imbalance between supply and demand
 - Elevated LVEDp decreases perfusion pressure
 - Myocardial hypertrophy increases demand
- **Syncope with exertion**
 - Inability to increase cardiac output and meet reduced SVR demands
- **Congestive heart failure**
 - Elevated LVEDp = elevated LAp = pulmonary venous congestion

CHF expression is exertion dyspnea

Exertional syncope

- may result from a decline in arterial pressure caused by vasodilatation in the exercising muscles in the face of a fixed CO

All these Clinical symptoms

- Appear only in critical AS.
- Even critical AS may exist for many years (until 60-80 years) without producing any symptoms (LVH maintains CO).
- Often there is a history of a slow progression of fatigue and dyspnea.

Late in the course of the disease

- marked fatigability, weakness,
- Pale skin (manifestation of a low CO).
- **LV failure** - Orthopnea, nocturnal dyspnea, pulmonary edema.
- **RV failure** with hepatomegaly, peripheral edema,

Aortic Stenosis

Prognosis with Symptomatic Aortic Stenosis

Clinical Symptoms	Median Survival
Angina	5 years
Syncope	3 years
CHF	2 years

Aortic Stenosis

PHYSICAL EXAM

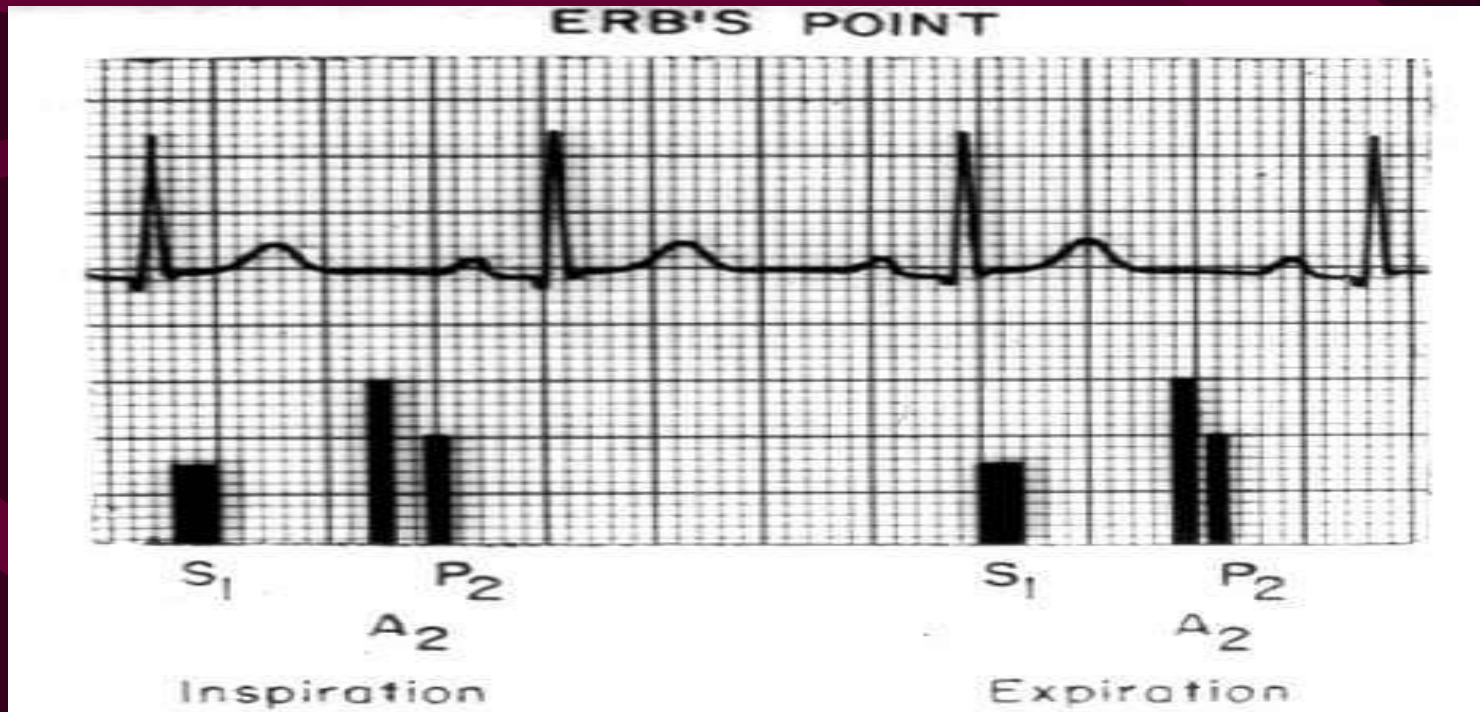
AS. Palpation of heart region

- The apex beat (LV impulse) - usually powerful, may be displaced laterally, reflecting the presence of LV hypertrophy and dilatation.
- Systolic thrill ("cat's purr") may be present in the II intercostal space, mostly right one, jugular notch, along the carotid arteries.

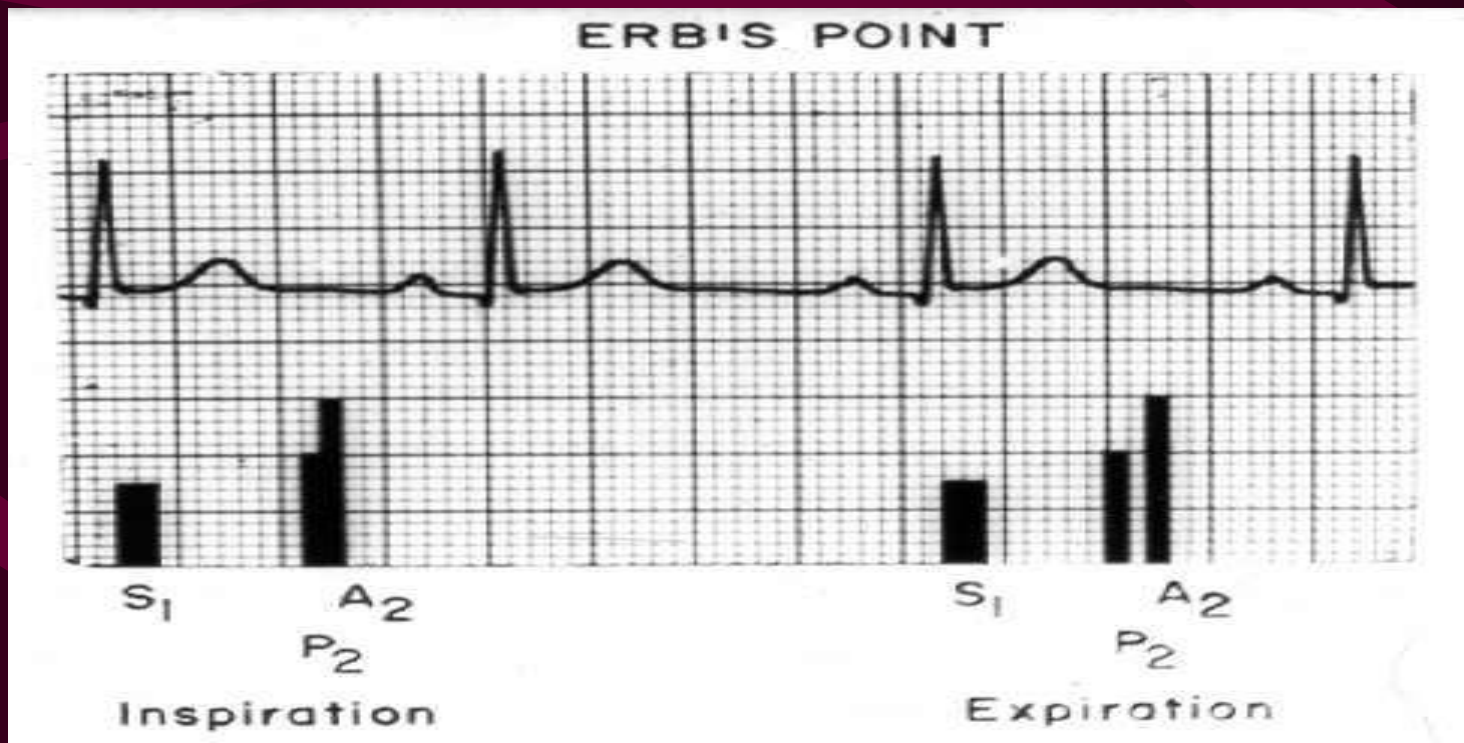
AS. Auscultation in Aortic area

- **S2 diminished** usually, due to pronounced valve calcification (+ arterial hypotension late in the course of disease).
- S4 or S3 gallop may be audible at the apex, when the LV dilates.
- Paradoxical splitting of the S2 second sound .

- The **normal** influence of inspiration on S2: the pressure within the thorax $\downarrow$, venous return to RV $\uparrow$, so emptying time of RV to be delayed, PV closure more later.

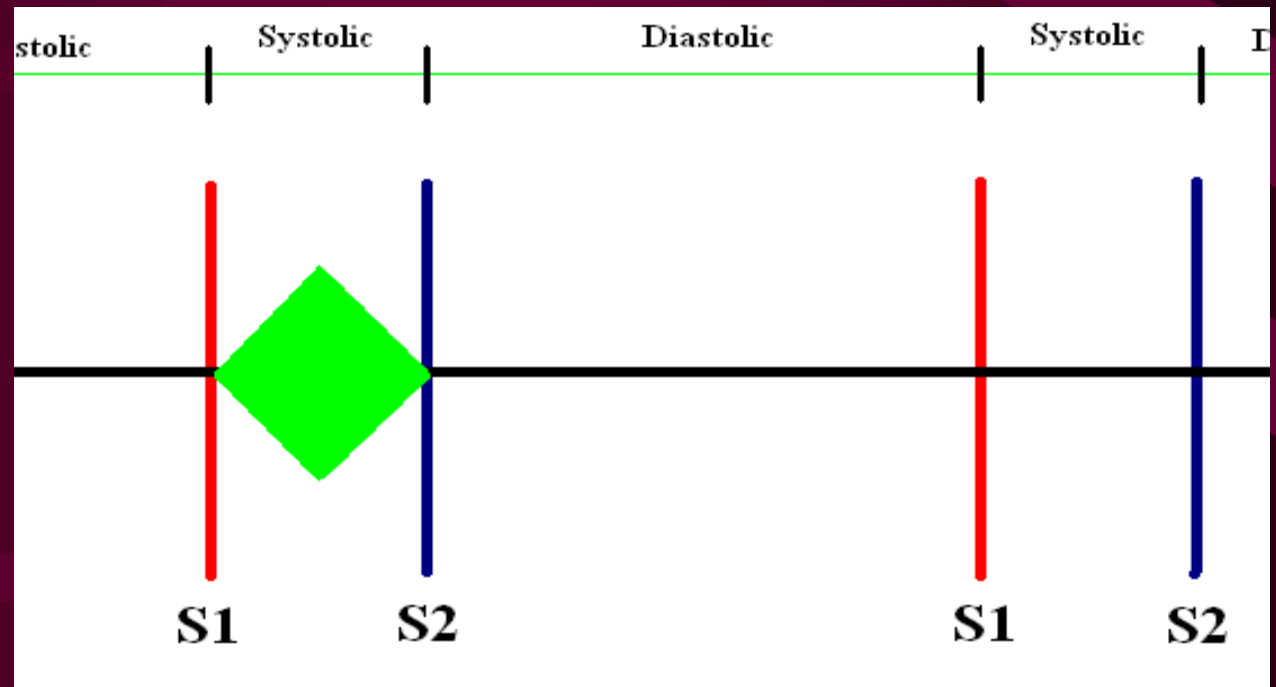


- *Paradoxical splitting of S_2 in AS*
 - LV systole become prolonged ,
 - the emptying time of LV is delayed, the order of valve closure is reversed. In inspiration, the two components then move closer together or may be single.



AS. Auscultation (murmurs)

- An ejection (mid) systolic murmur shortly after the S1, increases in intensity toward the middle of ejection, and ends just before aortic valve closure.



- It is characteristically low-pitched, rough in character, loudest at the base of the heart, most commonly in the second right intercostal space.
- It is transmitted upward along the carotid arteries.
- Better heard in orthostatic position, the patient bending forward, post-expiratory apnea.

INSPIRATION

- INSPIRATION **DECREASES** VENOUS RETURN TO THE LEFT HEART
- INSPIRATION WILL **INCREASE** THE VENOUS RETURN TO THE RIGHT HEART
- MURMURS ON THE LEFT SIDE WILL GENERALLY DECREASE WHILE THOSE ON THE RIGHT SIDE OF THE HEART WILL INTENSIFY (TR – CARVALLO'S SIGN)

Heart rate

- Sinus bradycardia (FCB < 55 b/min), which is compensatory - LV systole becomes prolonged for a fuller ejection through a narrowed lumen.

Arterial pressure

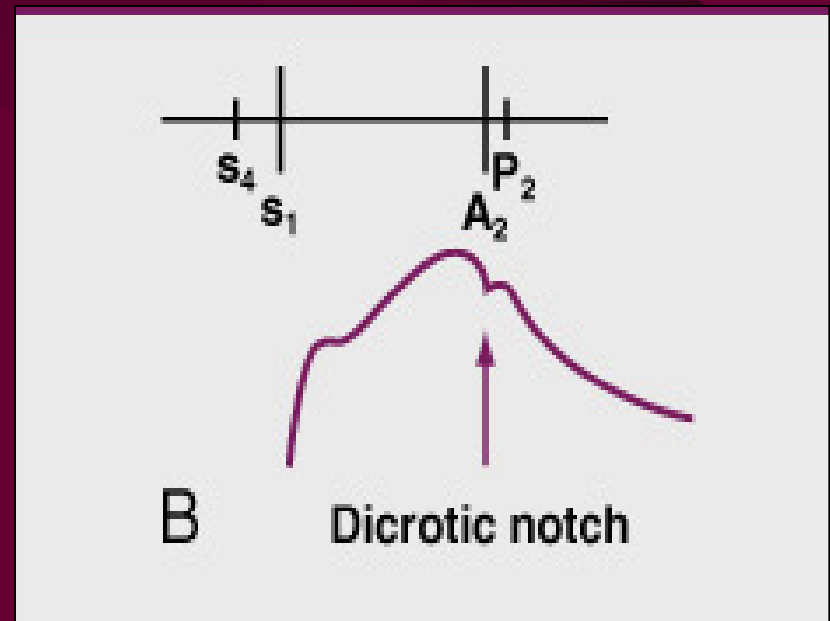
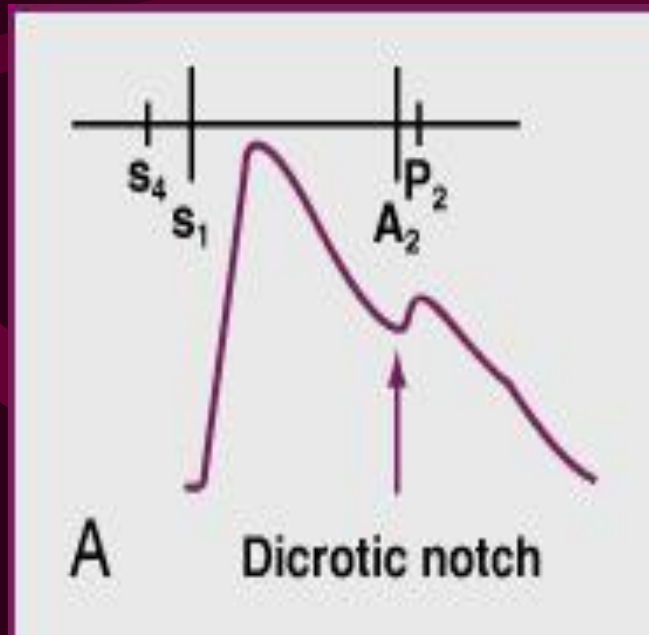
- is usually within normal limits.
- When stroke volume declines, the systolic pressure may decrease (ex - 100/80) and the *pulse pressure narrow*.

AS. Peripheral arterial pulse

- **Pulsus parvus et tardus** – a small, slow pulse - it rises slowly, to a delayed peak; may be palpated on the carotid or radial arteries.

In the elderly, the stiffening of the arterial wall may mask this important physical sign.

When the pulse pressure is reduced, the pulse amplitude may be so small that may become difficult to be appreciated.

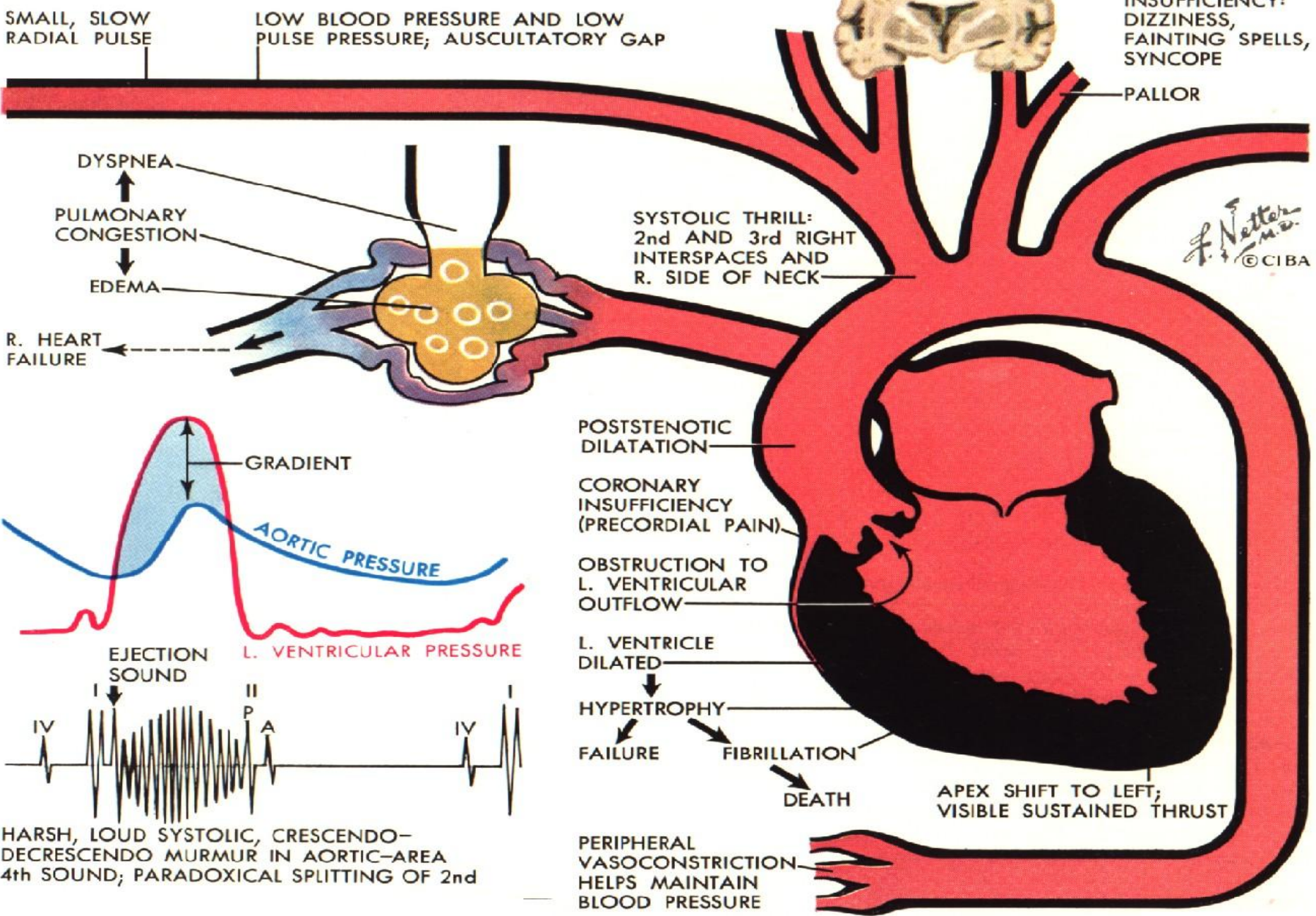


Aortic Stenosis

Physical Exam, summary

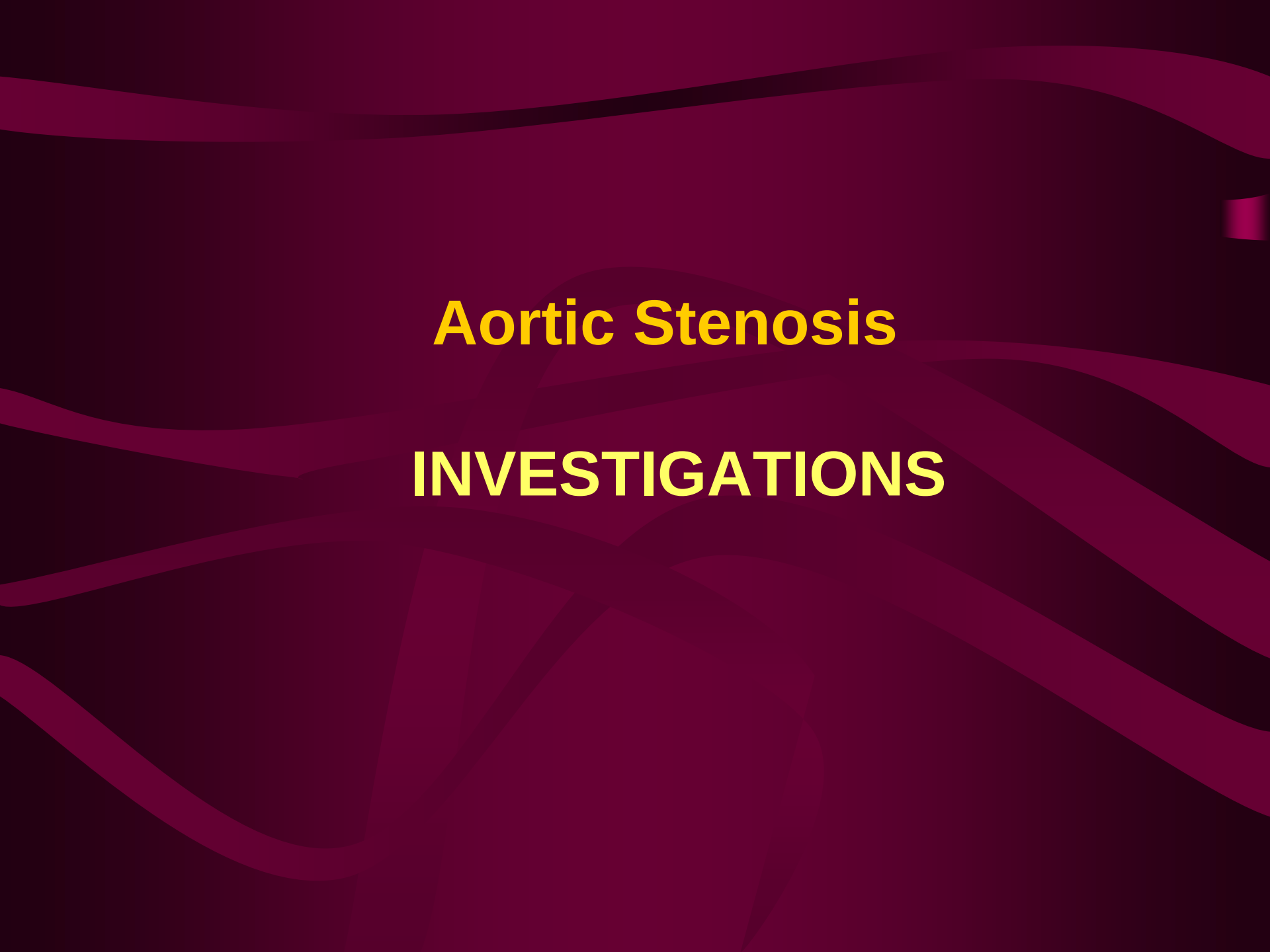
- *Harsh Systolic Ejection Murmur – late peaking*
- *S4 or S3 gallop may be (from LVH)*
- *Sustained apex beat (LV impulse, from LVH)*
- *Pulsus Parvus et Tardus*

AORTIC STENOSIS



HARSH, LOUD SYSTOLIC, CRESCENDO-DECRESCENDO MURMUR IN AORTIC-AREA
4th SOUND; PARADOXICAL SPLITTING OF 2nd

PERIPHERAL VASOCONSTRICTION HELPS MAINTAIN BLOOD PRESSURE



Aortic Stenosis

INVESTIGATIONS

Diagnosis

- *ECG – LVH, LAE*
- *Echo 2D/color doppler – test of choice*
- *Cardiac Catheterization – helpful, confirmatory, needed if the pt is older – look at the coronaries*

ECG -Electrocardiogram

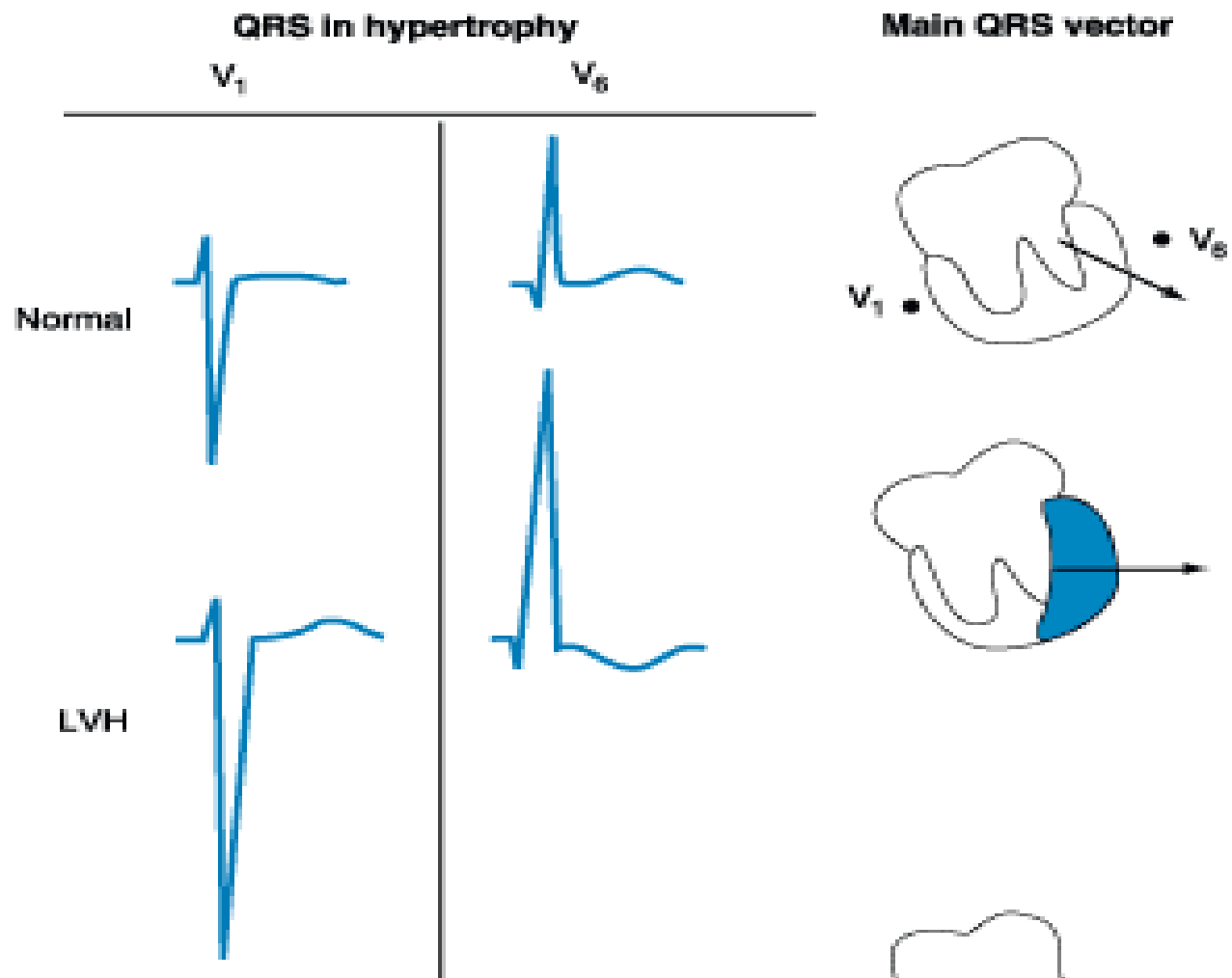
LV hypertrophy (LVH)-

- The main finding in most patients with severe AS

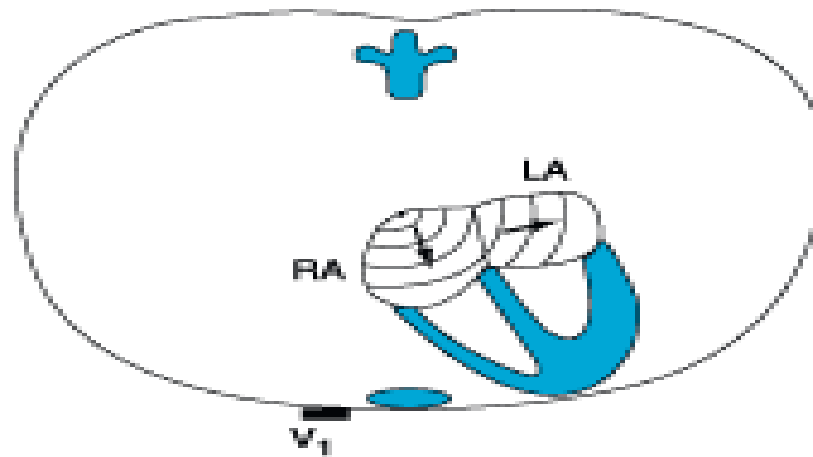
In advanced cases:

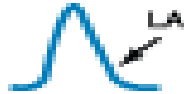
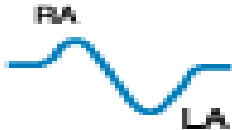
- ST-segment depression and T-wave inversion (LV "strain") *in the left leads* (I, aVL , V5-V6).
- Signs of LA enlargement (LAE)

LVH

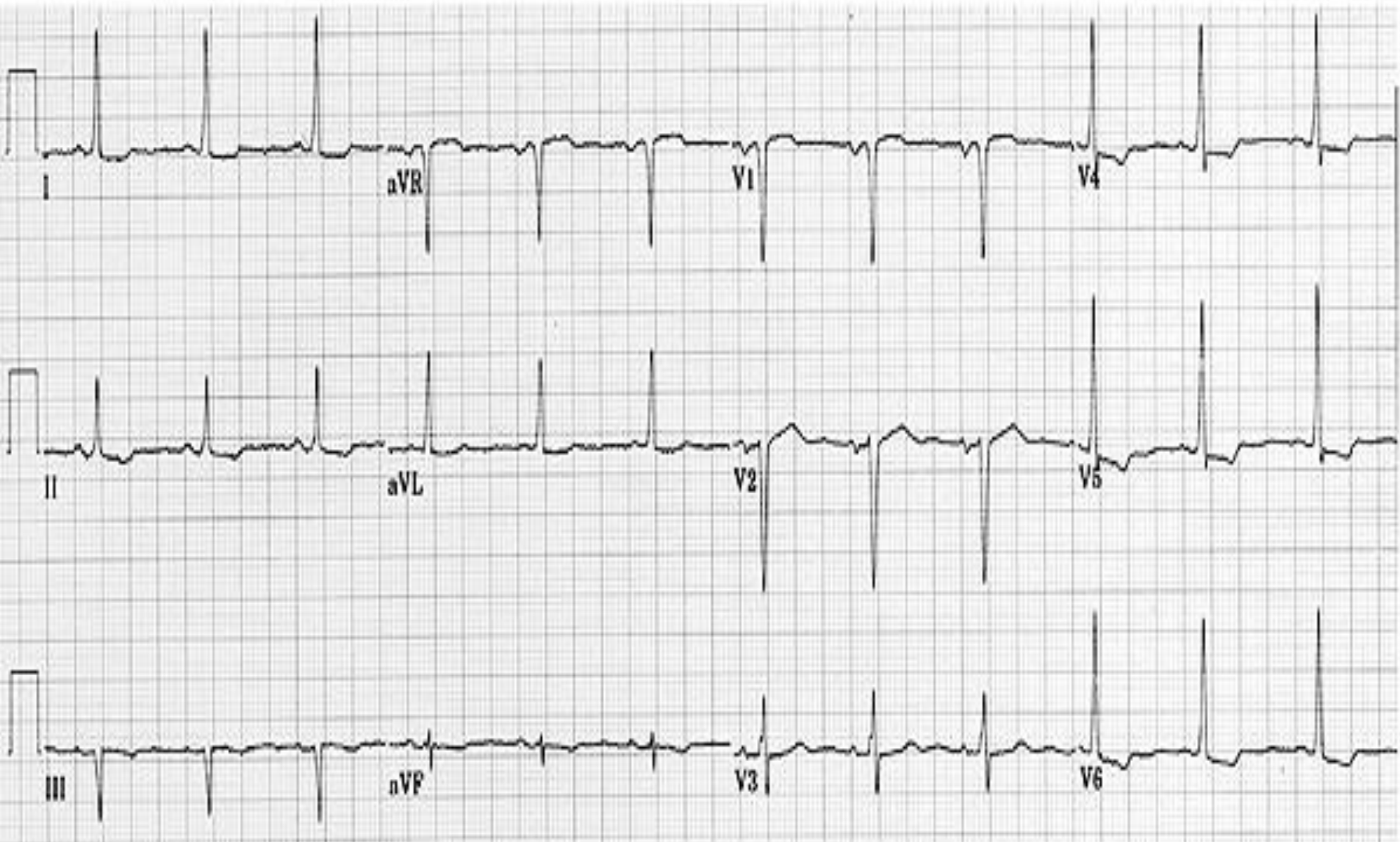


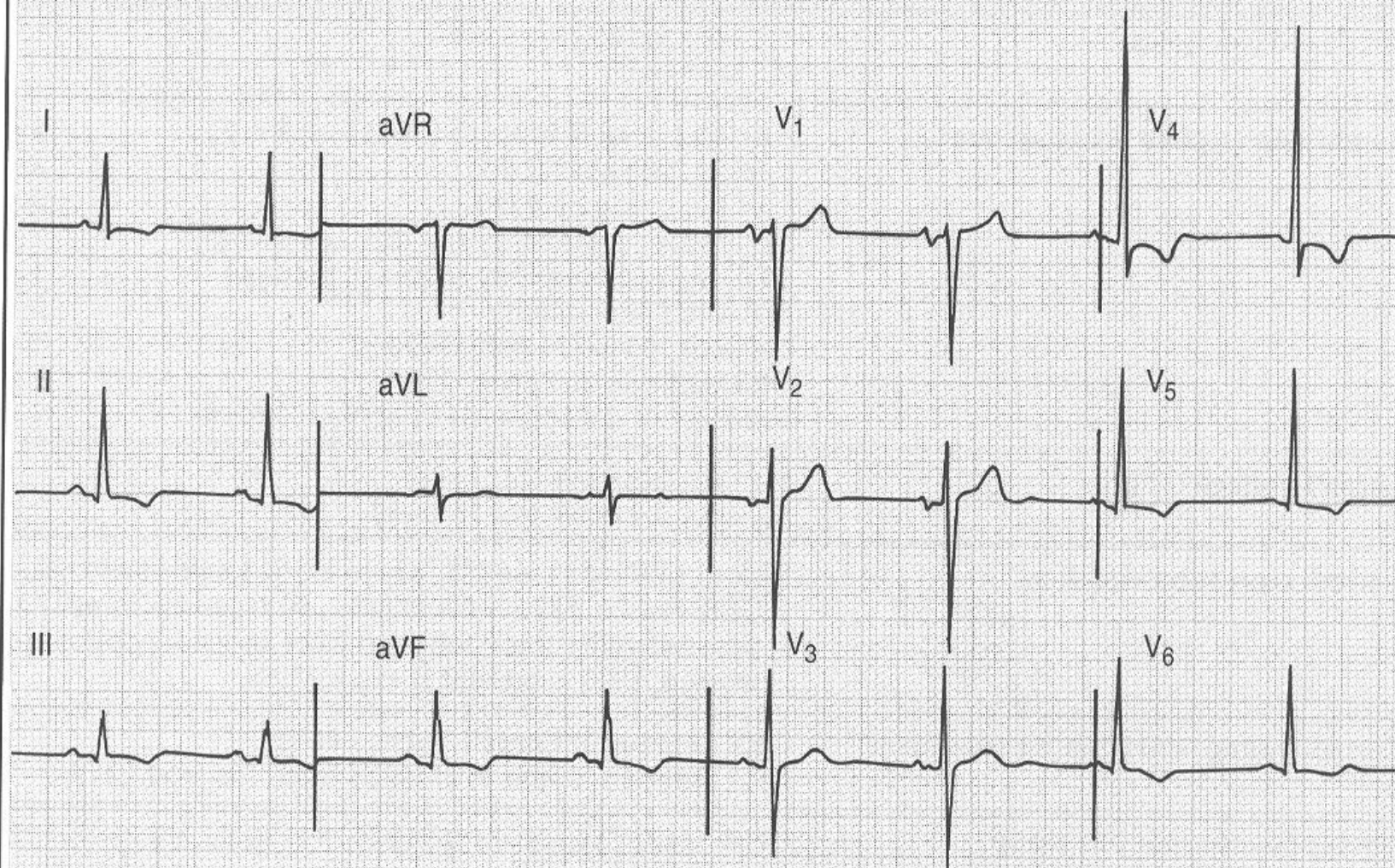
LAE – P wave changes



	Normal	Right	Left
II			
V ₁			

ECG - LVH





- However, there is no close correlation between the ECG findings and the hemodynamic severity of AS

EchoCG - key findings

- **LV hypertrophy**
- **Valvular calcification - multiple, bright, thick, echoes from within the aortic root;**
- **Congenitally bicuspid valves.**
- **Decreased opening of aortic valve < 8 mm**
- **aortic orifice < 0,8cm²**
- **transaortic pressure > 50 mmHg**

EchoCG

Ejection fraction (EF) =
= $\text{LVeDV} - \text{LVeSV} / \text{LVeDV} =$
= Stroke Volume / LVeDV in %

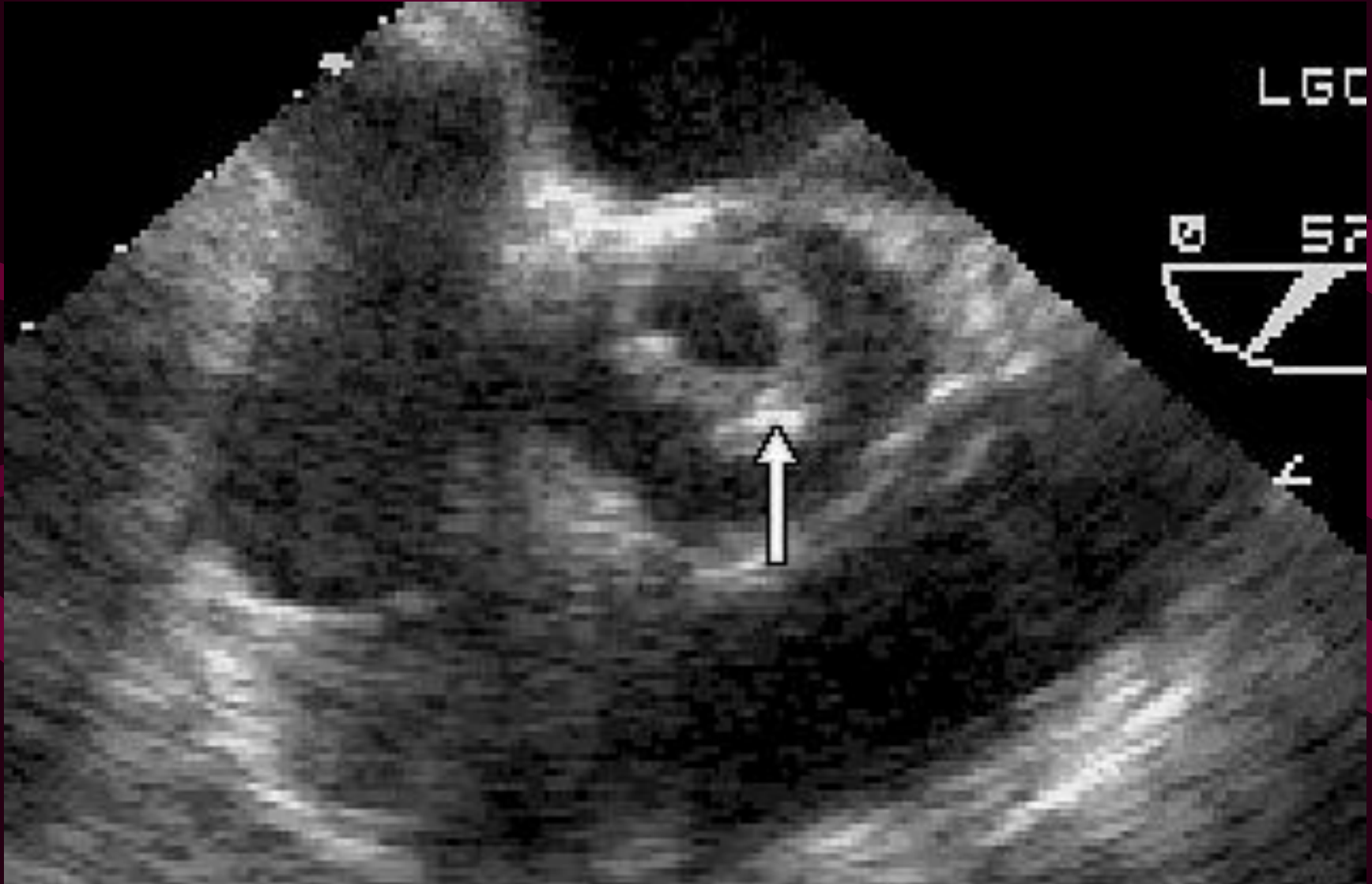
First sign of LV failure = decreased ejection fraction.

Transesophageal EchoCG

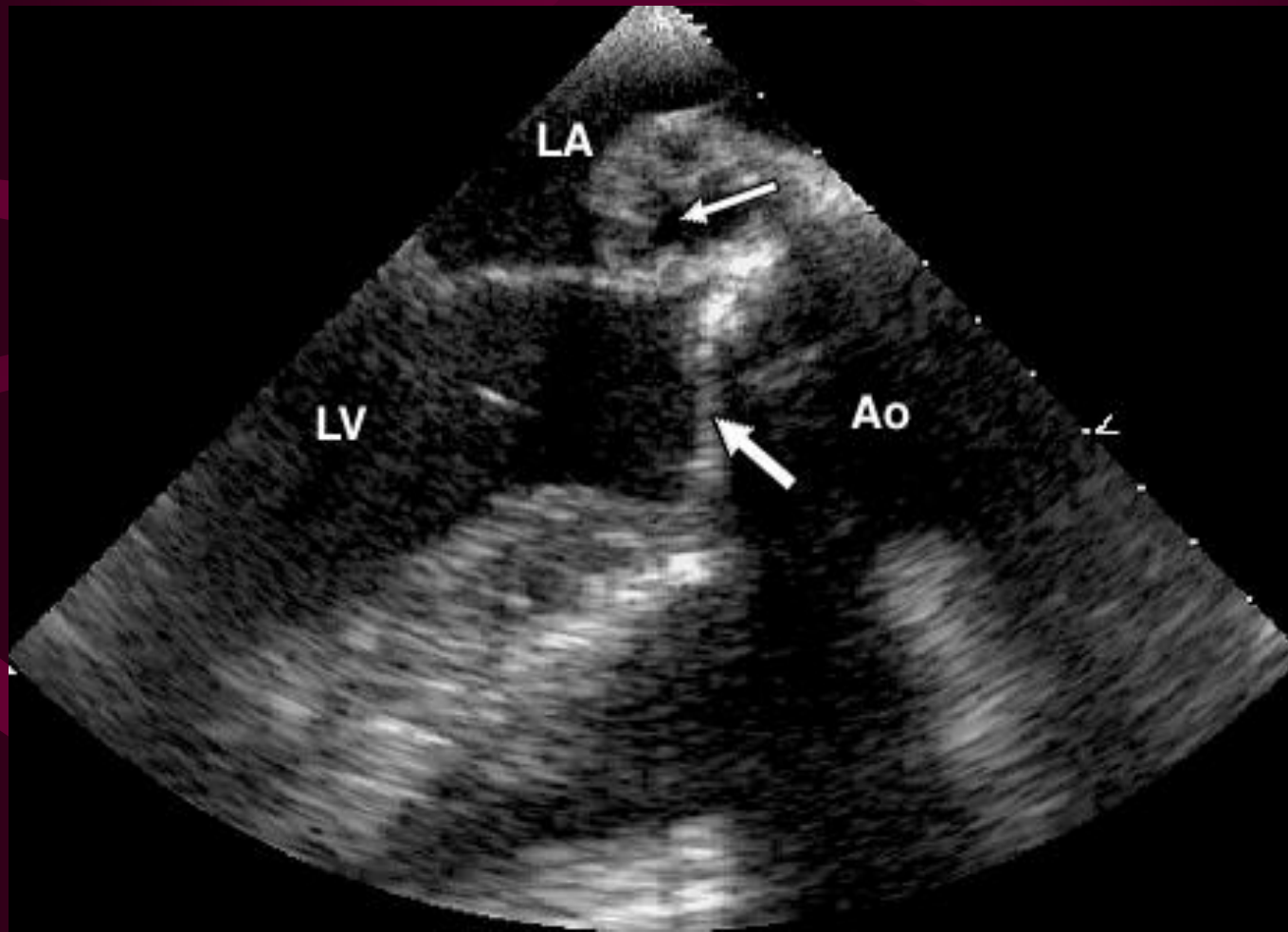
EchoCG with Doppler identify:

- Valvular pressure gradient
- Valvular abnormalities

Aortic stenosis with one cusp aortic valve (arrow- single commissure)



Transesophageal echo of the aortic valve
abscess seen in the inner annular fibrosa;
thin arrow- abscess cavity,
thick arrow- aortic valve



Chest X-ray

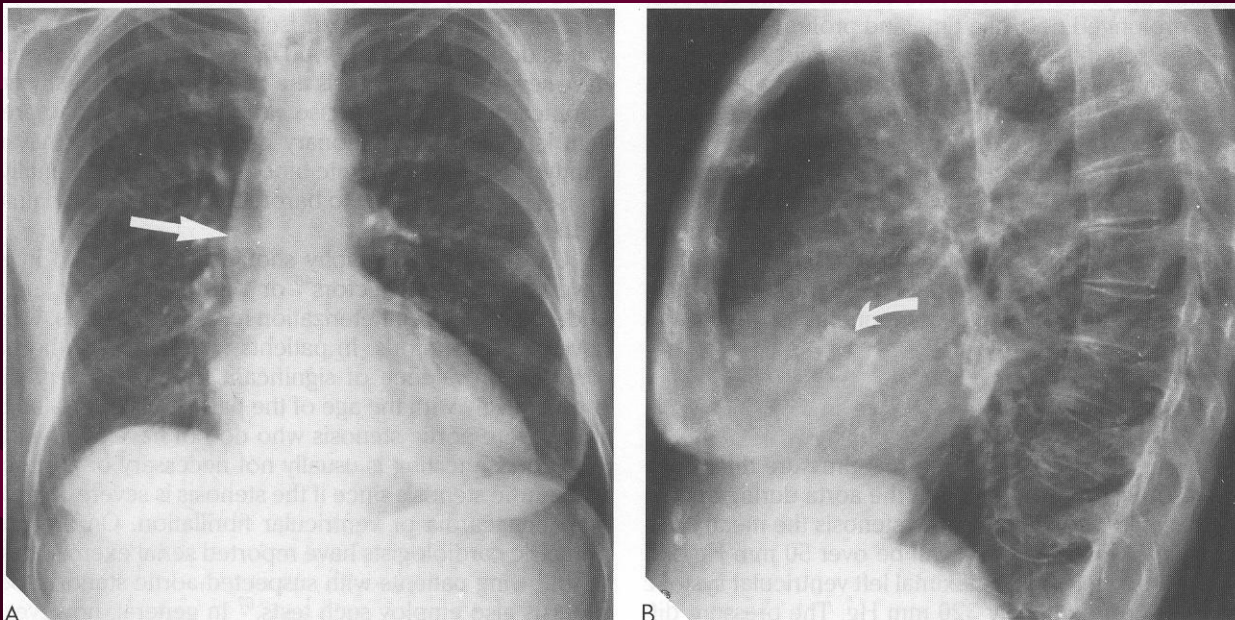
- Little cardiac enlargement (for many years).
- Poststenotic dilatation of the ascending aorta (in critical AS).
- Aortic calcification.

In later stages as the LV dilates, there is evidence of :

- LV enlargement;
- pulmonary congestion;
- enlargement of the LA, PA.

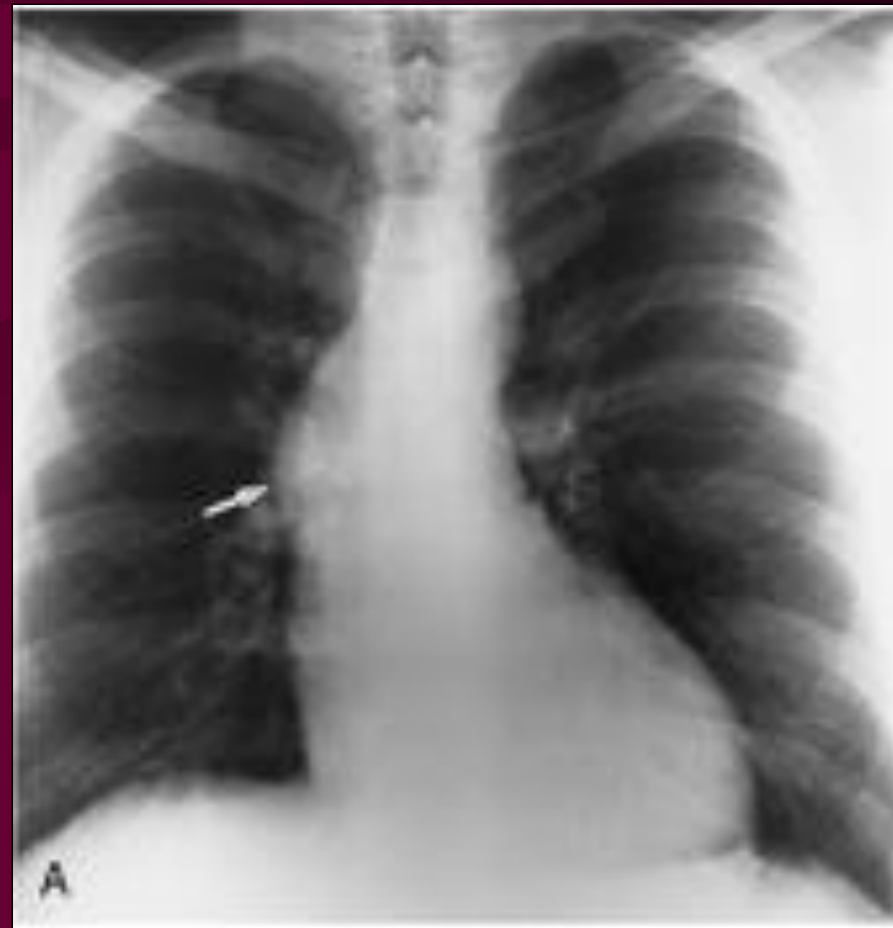
Chest X-ray

- **Poststenotic dilatation of the ascending aorta (in critical AS).**
- **Aortic calcification.**



Chest X-ray

- **Poststenotic dilatation of the ascending aorta**
- **LVH**
- **Pulmonary congestion**
- **More expressed heart waste (aortic configuration)**



X-ray of Heart

-LVH

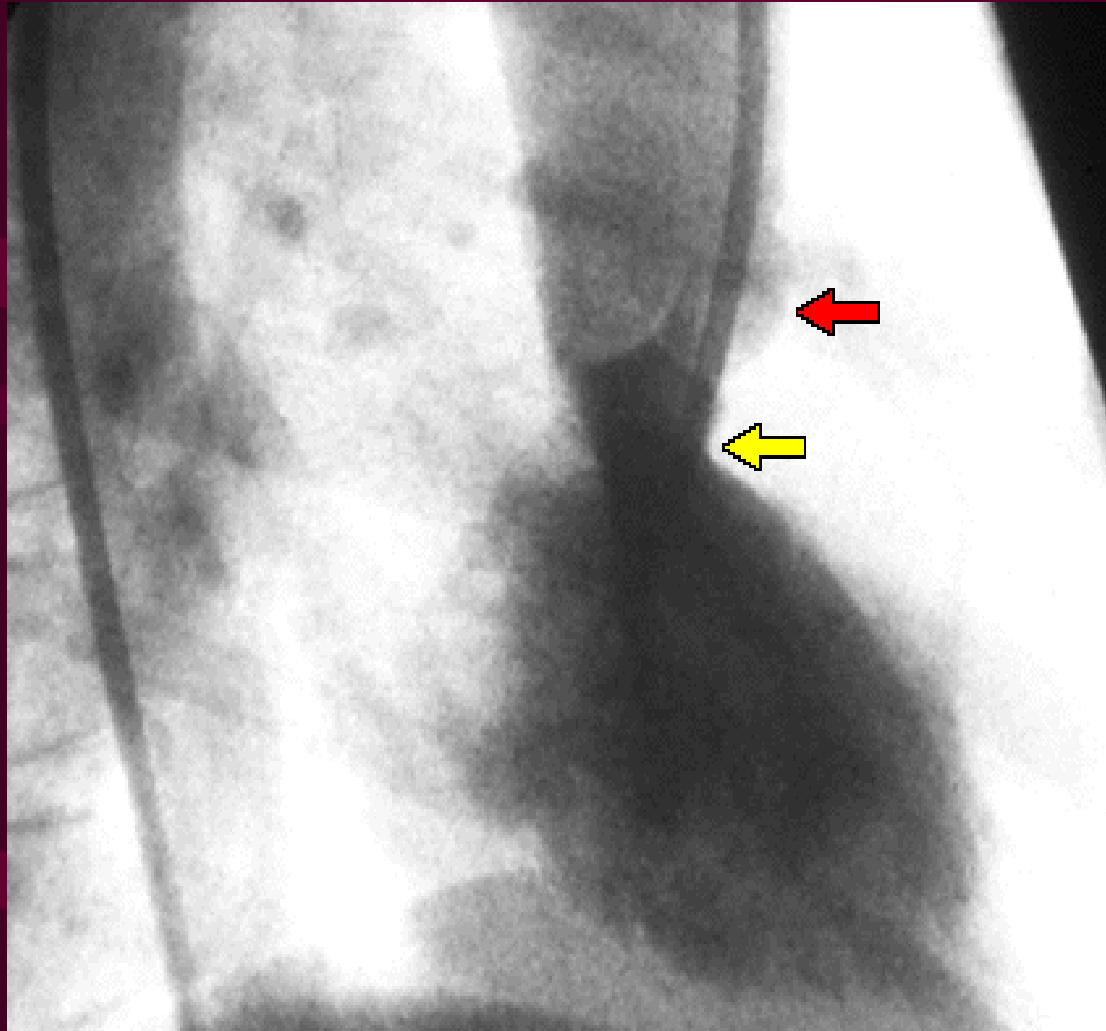
- Pulmonary congestion



Catheterization

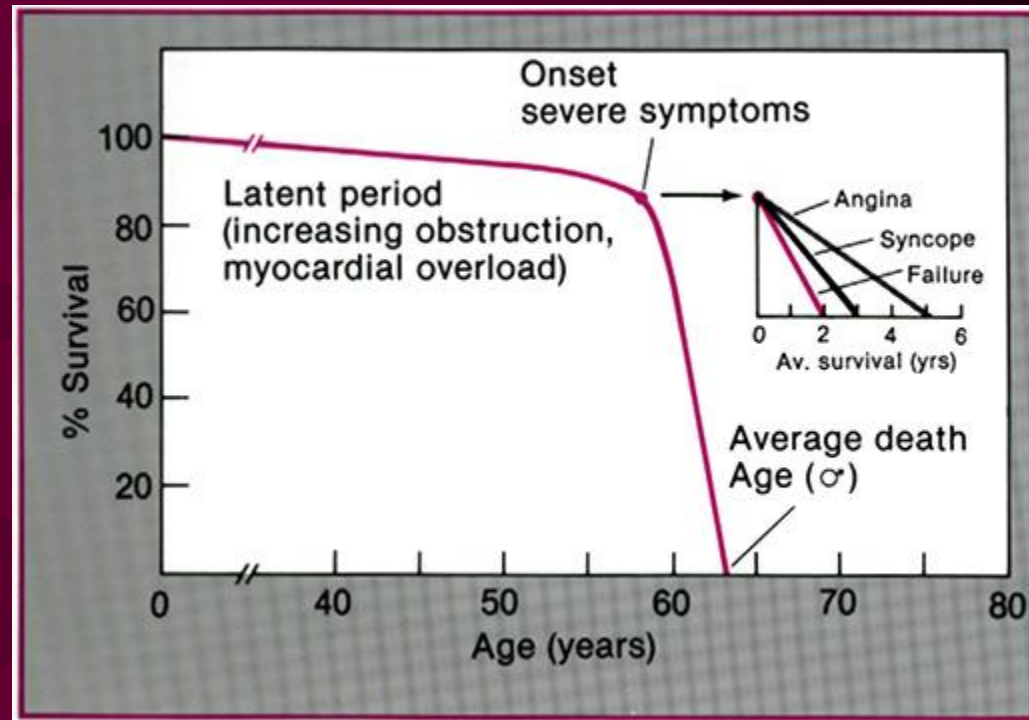
- Catheterization of the left side of the heart
 - coronary angiography
- should be carried out in patients suspected of having severe AS who are being considered for operative treatment.

Subvalvular stenosis (yellow arrow - AS ; red arrow - aortic valve)



NATURAL HISTORY

- In > 80% of patients who died with AS, symptoms had existed for < 4 years.
- Congestive heart failure was considered to be the cause of death in 1/2 - 2/3 of patients.



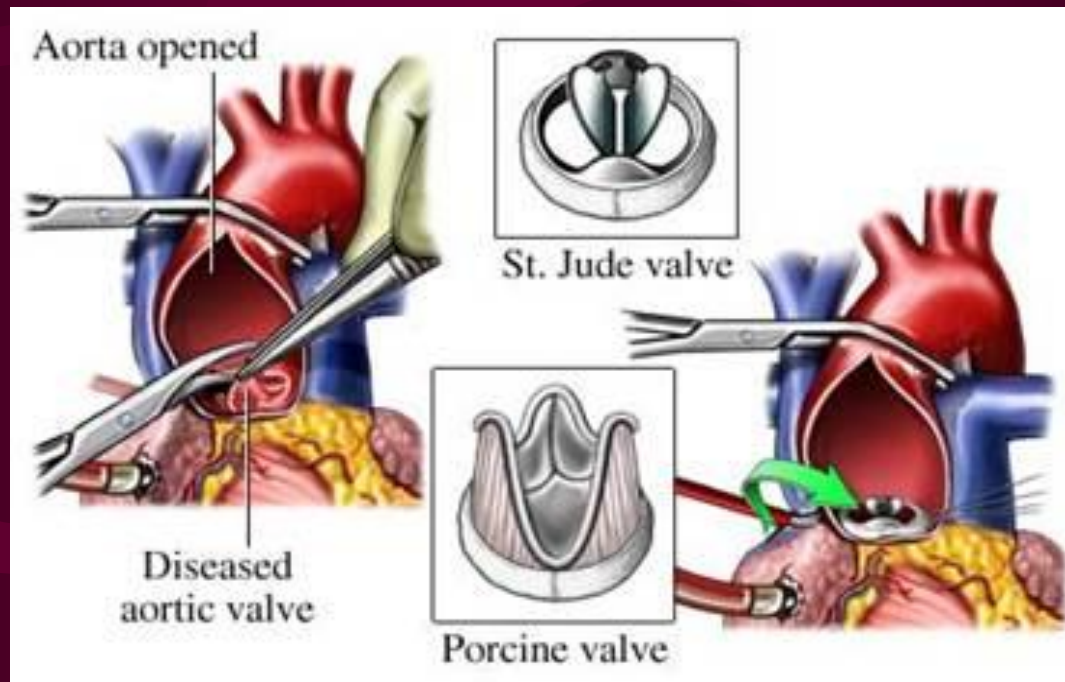
Aortic Stenosis

Treatment of Symptomatic Aortic Stenosis or Decreased LV Function

Medical Therapy – treats the symptoms not the cause

Aortic Valve Replacement

Bioprosthetic vs Mechanical AVR



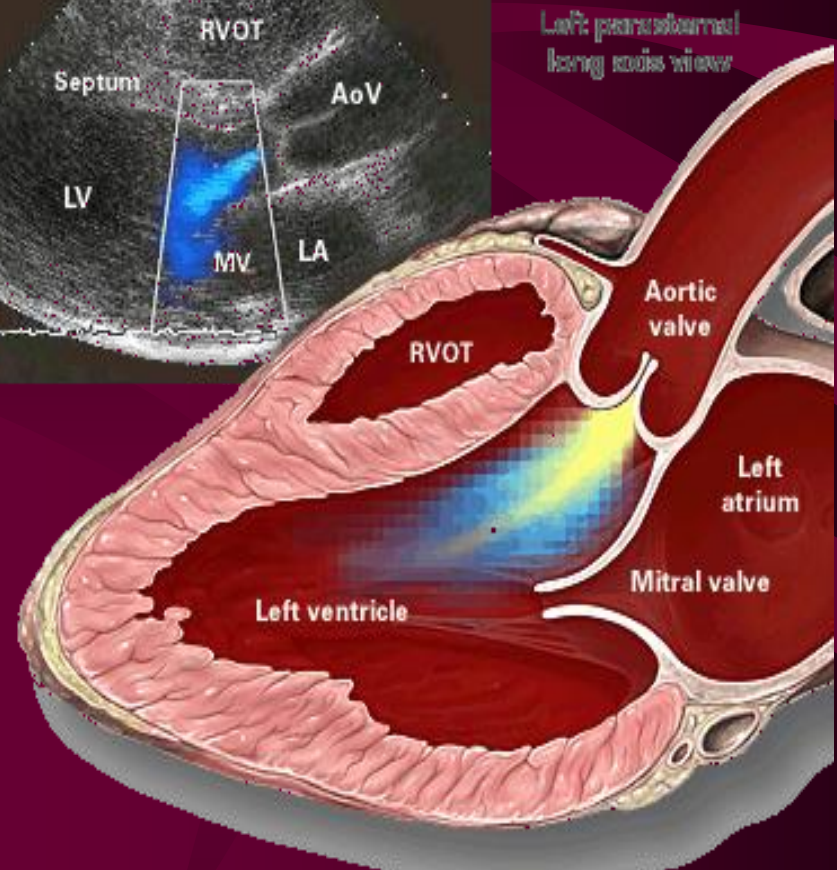
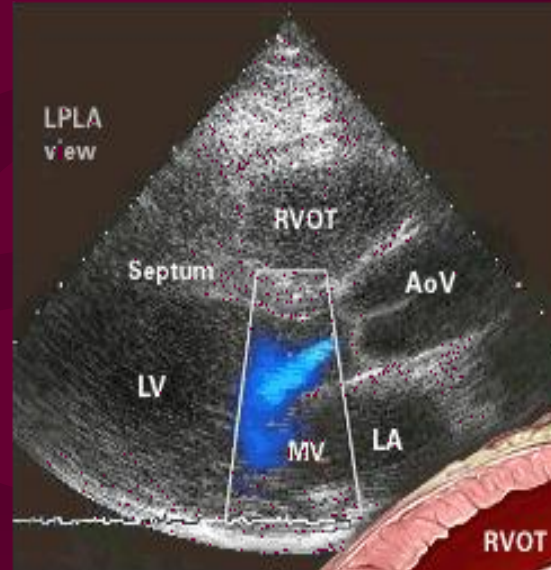
Aortic Valve

- *Aortic Stenosis*

- *Aortic Regurgitation (AR)* -

backflow of blood across the *incomplete closed* aortic orifice in diastole

Aortic Regurgitation



Aortic Regurgitation

Etiologies

- **Abnormalities of the Leaflets**
 - *Rheumatic, Bicuspid, Degenerative*
 - *Endocarditis*
 - *Congenital fenestrations of the aortic valve*
 - *Traumatic rupture of the aortic valve*
- **Dilation of the Aortic Orifice**
 - *Aortic Aneurysm / Dissection / severe AHT*
 - *Inflammatory* (Syphylis, Giant Cell Arteritis, Vasc Dis - Ankylosis Spondylitis, Reiters)
 - *Inheritable* (Marfans, Osteogenesis Imperfecta)

❖ Chronic AR

- 2/3 of cases are due to rheumatic fever,
- Congenital bicuspid aortic valves;
- Prolapse of an aortic cusp, resulting in progressive chronic AR, occurs in approximately 15% of patients with ventricular septal defect.
- *Pathomorphology*: thickening, deformity, and shortening of the aortic valve cusps, that prevent their closure during diastole.

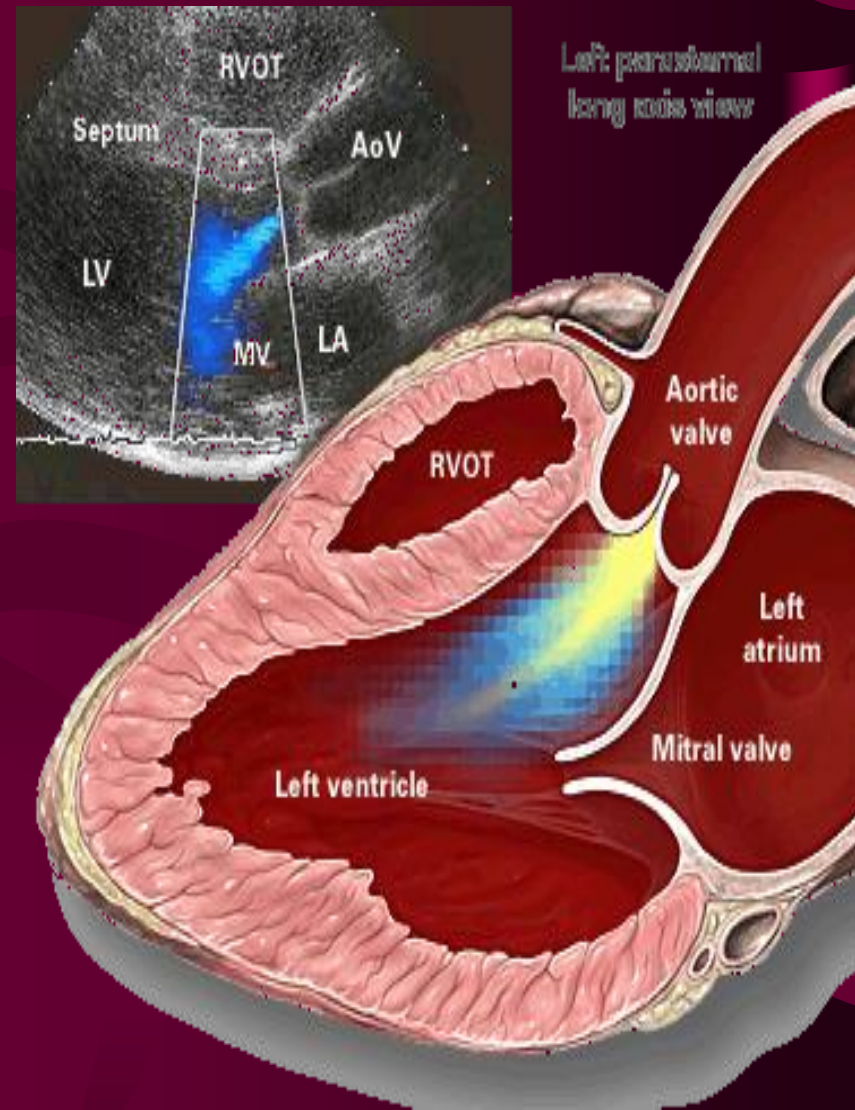
Acute AR

Etiology :

- Infective endocarditis,
- Traumatic rupture of the aortic valve (nonpenetrating cardiac injuries).
- *Pathomorphology:* perforation or erosion of the leaflets.

Aortic Regurg –PATHOPHYSIOLOGY (1)

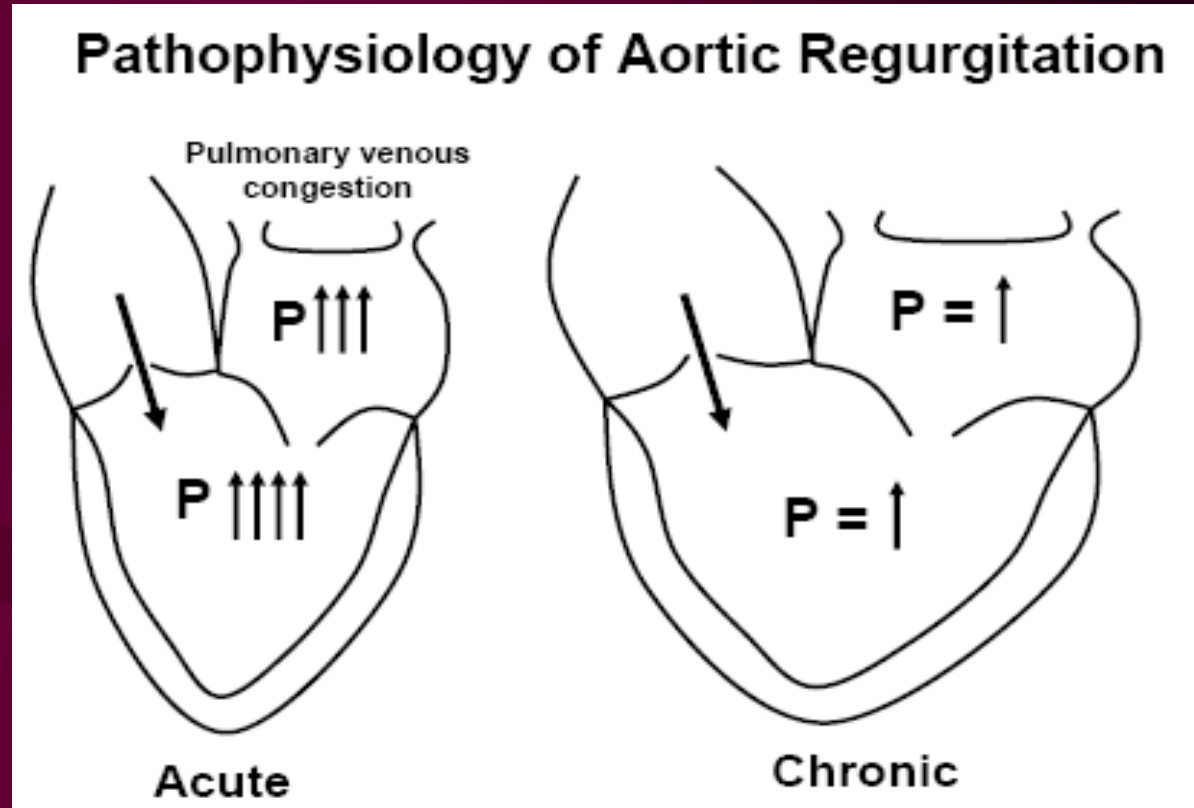
- During diastole, blood is delivered into the LV not only from the LA, but also from aorta due to regurgitation through incomplete closed aortic valve;
- Amount of blood at the end of diastole increases;
- LV volume increases (end-diastolic);
- Stroke LV volume increases (amount of blood pushed in aorta);



AR-PATHOPHYSIOLOGY (2)

Compensatory mechanisms:

- Dilatation of LV (is getting overfilled);
- The dilatation of the LV allows this chamber to eject a larger stroke volume.
- Intensified work of LV causes LV hypertrophy;



- Sinus Tachycardia (which reduces diastole)
- Aortic dilatation

Left Ventricular Hypertrophy



Normal



Overstrained LV
(AS)

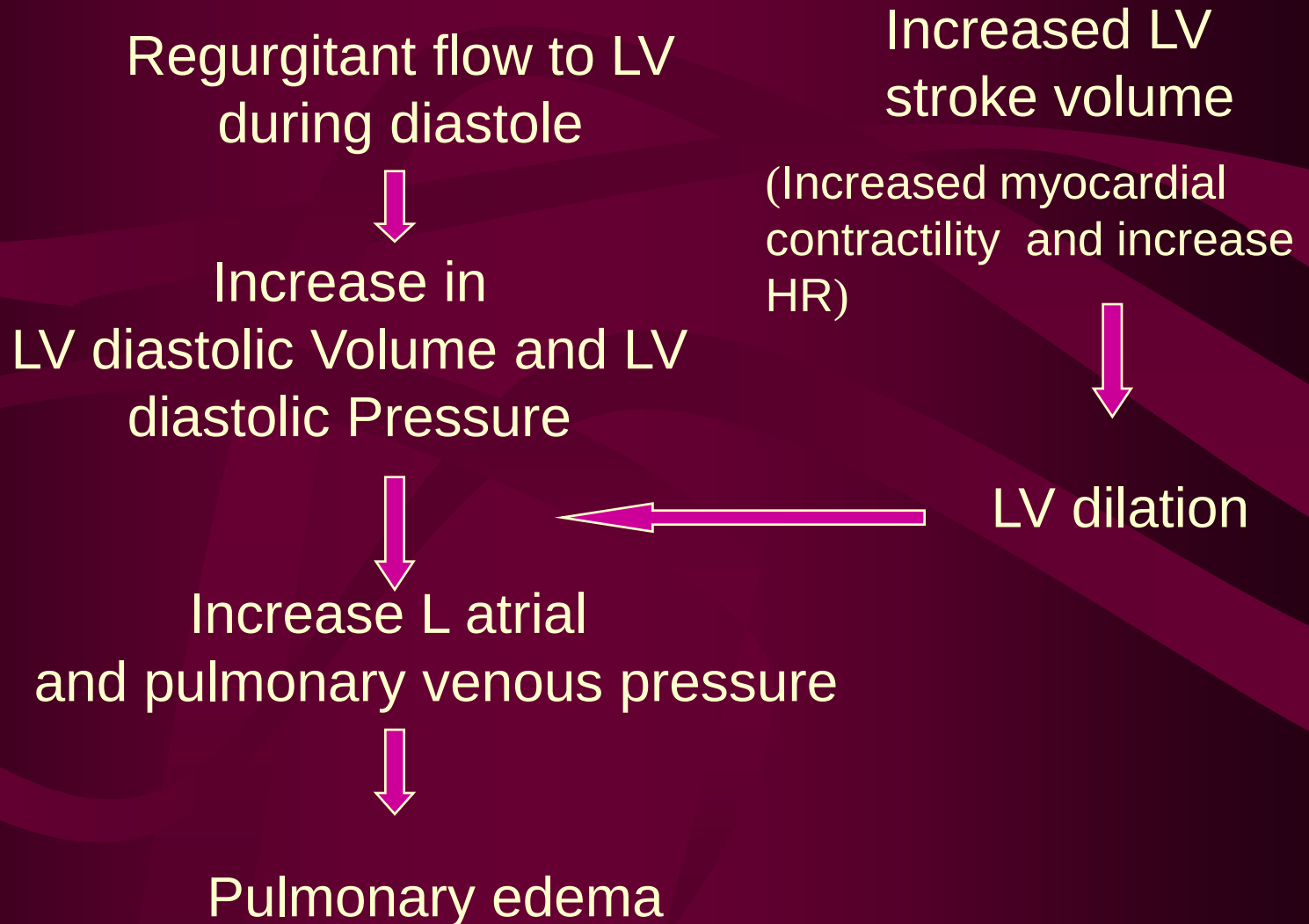
***Concentric
Hypertrophy***



Overfilled LV
(AR)

***Excentric
Hypertrophy***

Acute AR-PATHOPHYSIOLOGY (3)



AR-PATHOPHYSIOLOGY (4)

- In patients with severe AR, the effective forward CO often fails to rise normally during exertion.
- The strong muscle of LV can compensate the disease for a long time – 10-20 years.

AR-PATHOPHYSIOLOGY (5)

- High systolic blood pressure
- Low diastolic blood pressure

Example: 160/50

- **Widened pulse pressure**
 - Stroke volume increased (high SBP)
 - Regurgitant volume increased (low DBP)

AR-PATHOPHYSIOLOGY (6)

- Imbalance between myocardial supply and demand
 - Decreased DBP = decreased perfusion pressure = decreased supply
 - Increased LV size (and thus wall stress) = increased demand
- As a result = angina pectoris

AR-PATHOPHYSIOLOGY (7)

When LV Decompensation occurs

- LV failure :

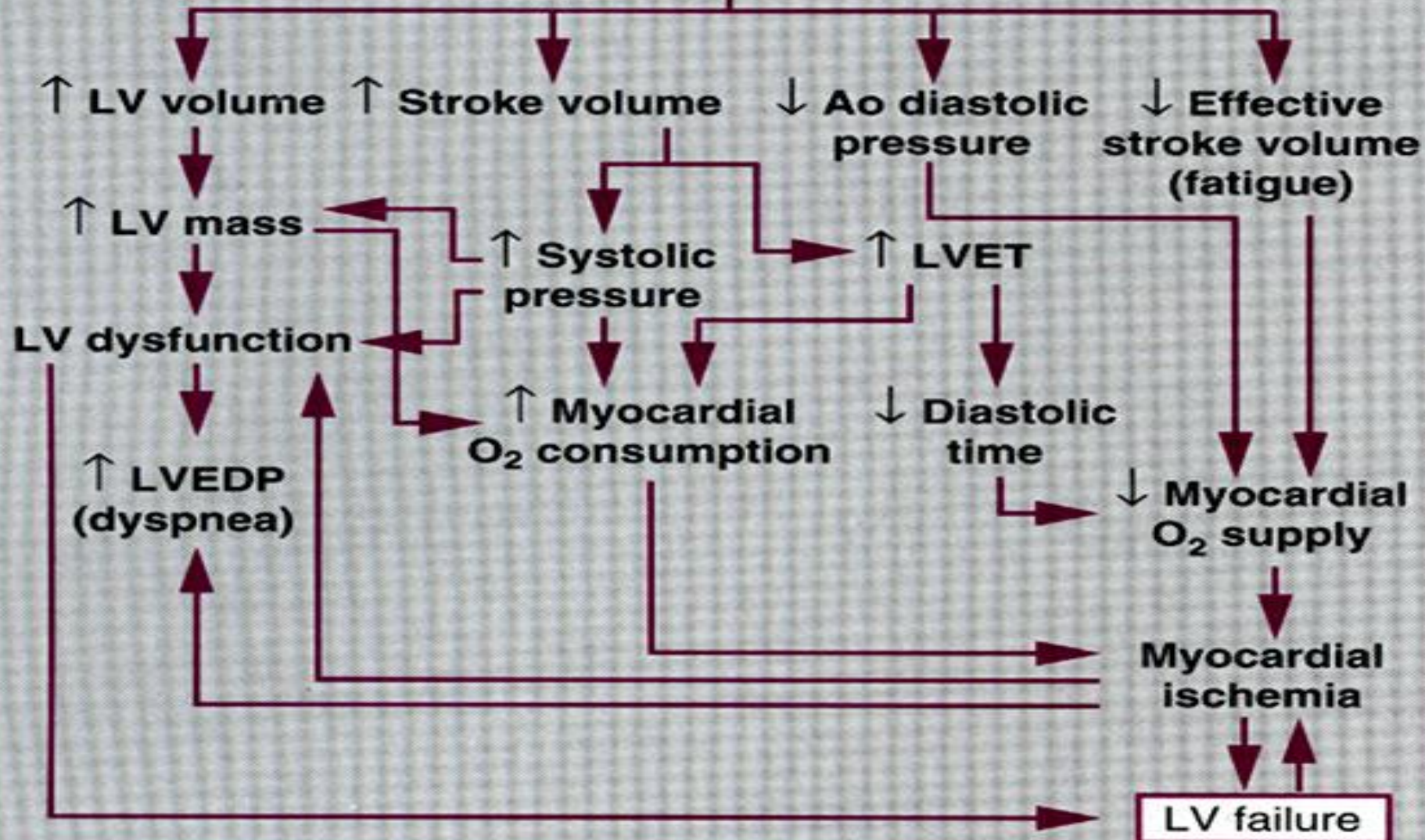
- the end-diastolic volume rises
- the CO at rest decline.

The earliest sign: decreased ejection fraction (EF), determined by EcoCG.

- elevation of the pressure in LA,
- PA wedge pressure increases,
- PA and RV pressures elevate

Aortic regurgitation

Diastolic regurgitation



AR. Complaints

- **Palpitations** due to sinus tachycardia during exertion or emotion,
- **Angina pain** may develop at rest as well as during exertion

Later complaints

- **Pulmonary venous congestion**
 - Dyspnea on exertion
- **Inadequate cardiac output**
 - Fatigue
 - Diminished exercise tolerance

The **angina episodes** can be prolonged and often do not respond to sublingual nitroglycerin.

Mechanism of angina : increased oxygen demand and reduced supply

Increased oxygen demand : LV dilatation and hypertrophy.

Reduced supply: the major portion of coronary blood flow occurs during diastole, when arterial pressure is subnormal, reducing coronary perfusion pressure

PHYSICAL FINDINGS in severe AR,

caused by the increased stroke volume and high pulse pressure (big difference between systolic and diastolic arterial pressures)

- the jarring of the entire body (jumping of the leg which is put on the other leg);
- **Head bobbing** - bobbing motion of the head with each systole (*de Musset symptom*);
- the abrupt distention and collapse of the larger arteries are easily visible – “**carotid shudder**”;
- **Muller sign** - pulsations of the uvula;
- Changes in **pupil dimensions** together with systole/diastole.
- **Quinke's pulse** (capillary pulsation when pressing the nail).

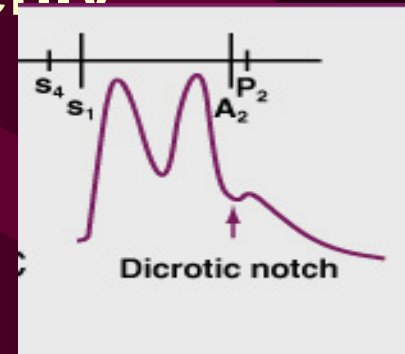
The examination should be directed toward the detection of conditions predisposing to AR, such as :

- the Marfan syndrome,
- rheumatoid spondylitis,
- ventricular septal defect.

Arterial Pulse in free AR

- Corrigan's pulse = high pulse and rapidly increasing pulse = “**pulsus altus et seller**”

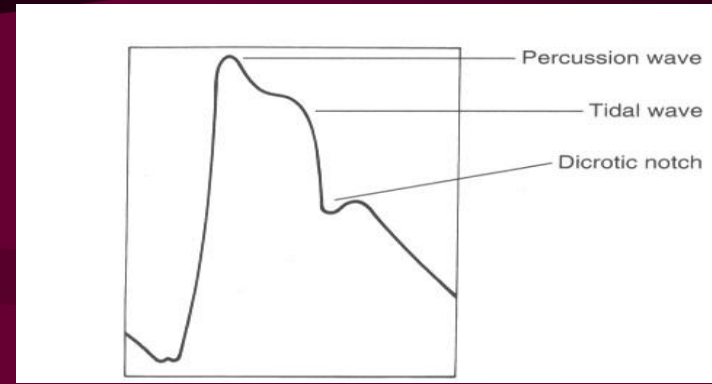
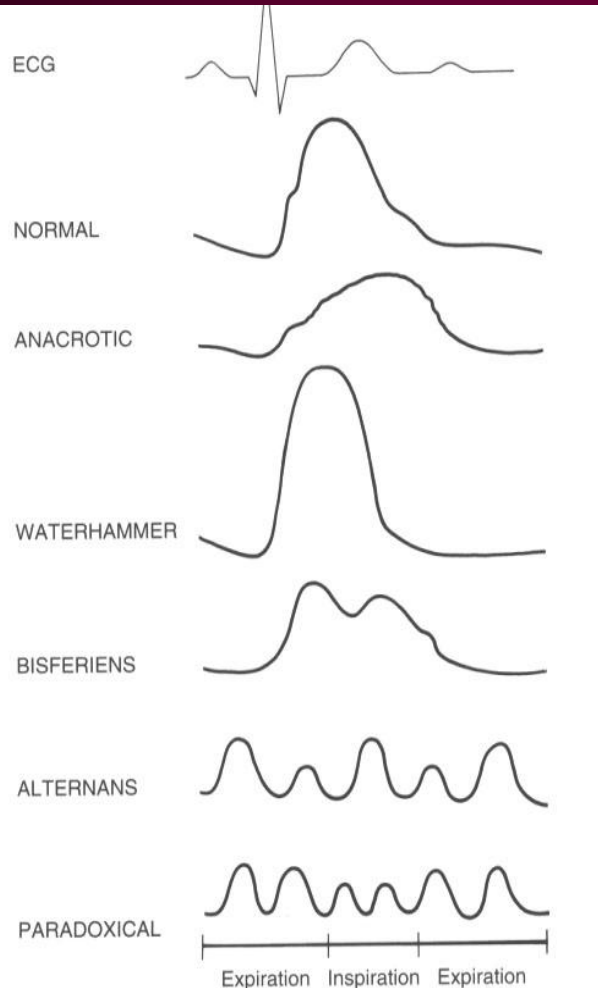
This is a rapidly rising pulse, like "water-hammer", which collapses suddenly as arterial pressure falls rapidly during late systole and diastole;



- ***Pulsus bisferiens*** - bounding, visible and forceful peripheral pulse, with two systolic waves.



ABNORMALITIES OF THE ARTERIAL PULSE



AORTIC STENOSIS

AORTIC REGURGITATION

AORTIC REGURGITATION

CONGESTIVE HEART FAILURE

CARDIAC TAMPONADE

- *Quinke's pulse* - an systolic blushing and diastolic blanching of the nail skin when pressure is applied gently to the tip of the nail.

Signs in the femoral arteries

- ***Duroziez's sign*** - systolic + diastolic bruit (murmur) produced by compression of the femoral a. with a stethoscope;
- ***Traube's sign or Pistol-shot sound*** - sharp sound like a gunshot.

Arterial pressure

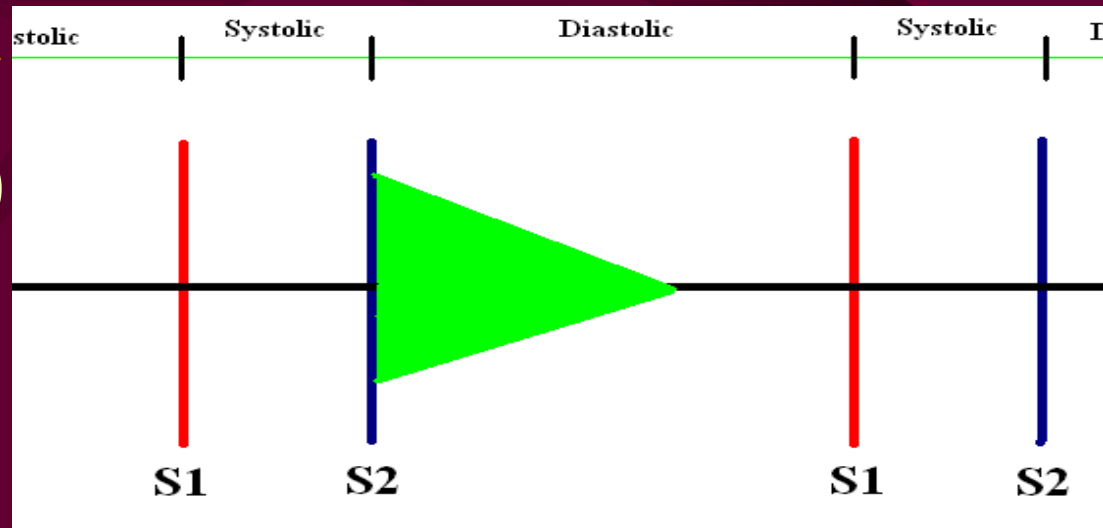
- The systolic pressure is elevated
- the diastolic pressure is decreased,
- widening of the arterial pulse pressure
(ex – 160/40; 170/30 mmHgcol).

AR. Palpation in the heart region

- The LV impulse (apex beat) is forced and displaced laterally and inferiorly.
- A diastolic thrill (“cat’s purr”) may be palpable along the left sternal border,
- In many patients with pure AR, the carotid arterial pulse is bisferiens, i.e., with two systolic waves.

AR. Auscultation

- S2 is diminished;
- S3 or S4 may be audible.
- Diastolic murmur, *decrescendo*, high-pitched, blowing, heard best in the 2nd right and 3rd left ICS (Erb point), immediately after S2;
- *Austin Flint murmur* (diastolic MV murmur)



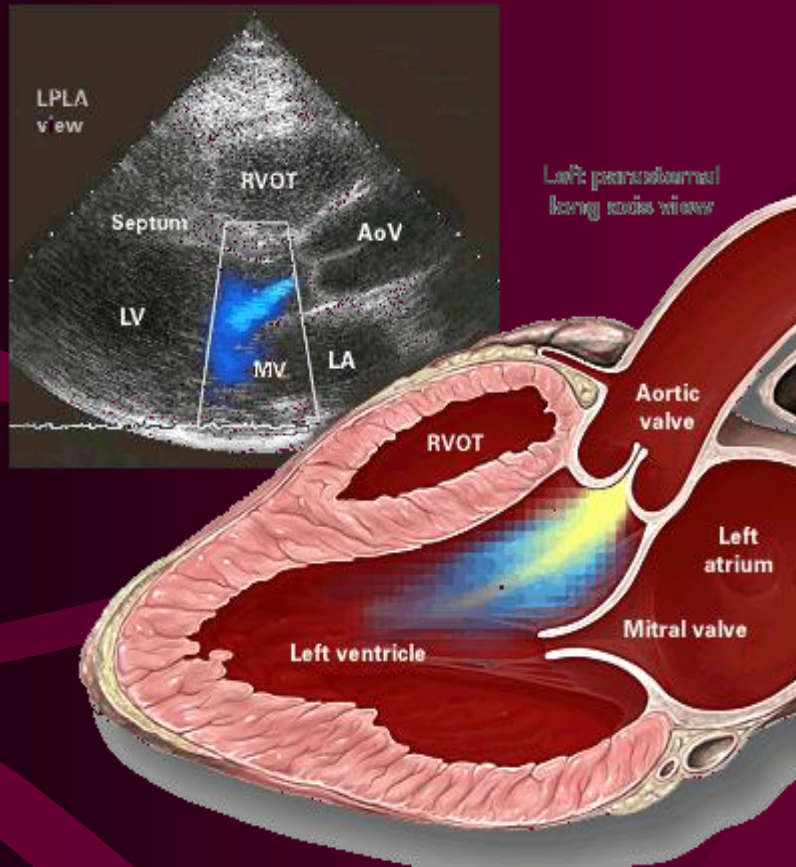
Murmur of AR

Accentuated by:

- sitting up,
- leaning forward,
- and with the breath held in forced expiration.
- isometric exercise such as handgrip.

Aortic Regurg

– Austin Flint Murmur –
a second murmur
frequently heard in
patients with severe AR –
a low-pitched
diastolic bruit,
similar to that heard in
MS.



Due to (1) anterior displacement of the mitral valve by the aortic regurgitant stream = **relative mitral stenosis** ;
(2). vibration of the anterior leaflet of the MV as it is buffeted simultaneously by the blood jets from the LA and the aorta

Both the Austin Flint murmur and the diastolic murmur of Mitral Stenosis are loudest at the apex, but :

- the murmur of MS is usually accompanied by a loud S1
- immediately follows the OS of the mitral valve,
- is longer in duration than Flint murmur.

*If you're not confused,
you're not paying
attention.*

Tom Peters

Marked LV dilatation lead to functional Mitral Regurgitation

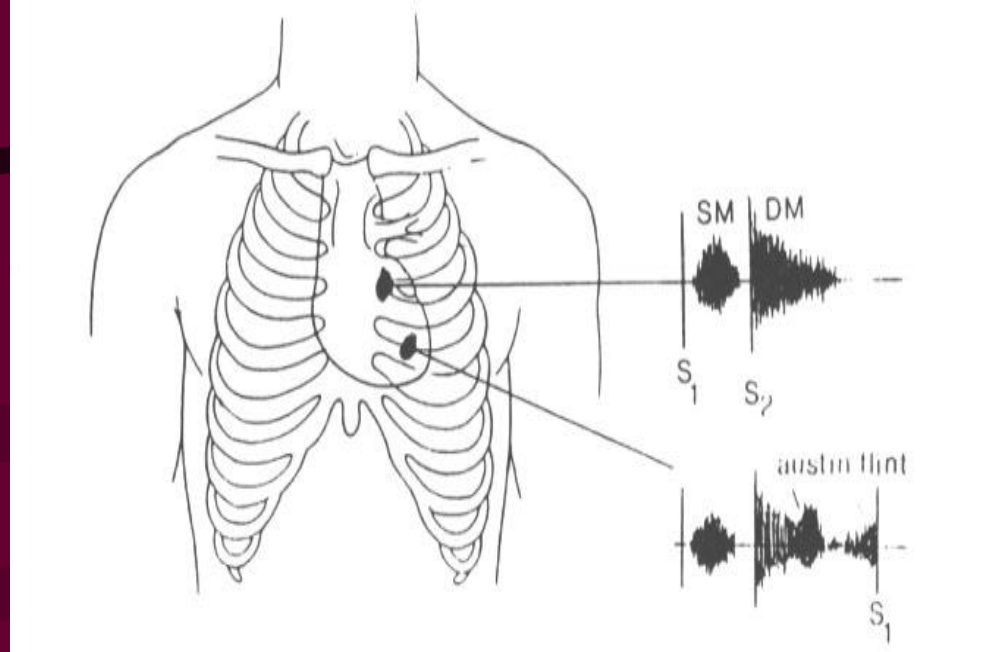
- Because the mitral orifice is get dilated
- Appears a blowing holosystolic murmur at the apex, which is transmitted to the axilla.

Aortic Regurgitation

Physical Exam.

TRADITIONAL PHYSICAL FINDINGS. Summary

- ***Dilated apical impulse***
- ***Diastolic Decrescendo Murmur***
- ***S4, S3 Gallop - advanced AR***
- ***Apical Rumble – “Austin Flint Murmur”, which may resemble MS***
- ***Wide pulse pressure***



Aortic Regurgitation

Diagnosis

Diagnosis

- *ECG – LVH, LAE*
- *EchoCG 2D/color doppler – test of choice*
- *Cardiac Cath – helpful, confirmatory, needed if the pt is older – look at the coronaries*

ECG

- In patients with mild AR, there may be no electrocardiographic abnormalities, but with severe, chronic AR - signs of **LV hypertrophy** become manifest.
- ST-segment depression and T-wave inversion in leads I, aVL, V5, and V6 ("LV strain").

ECG - LVH





I



II



III



VR



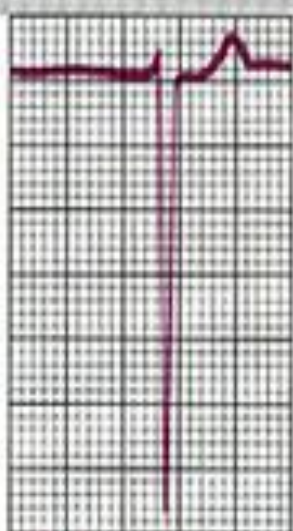
VL



VF



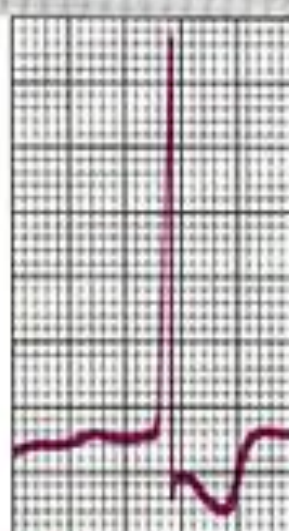
V₁



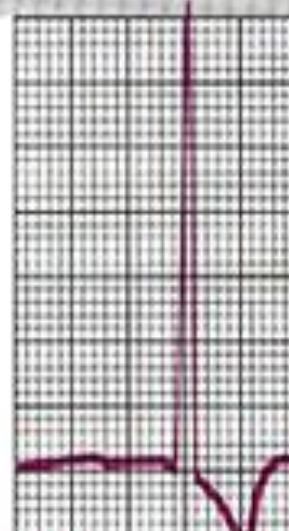
V₂



V₃



V₄



V₅



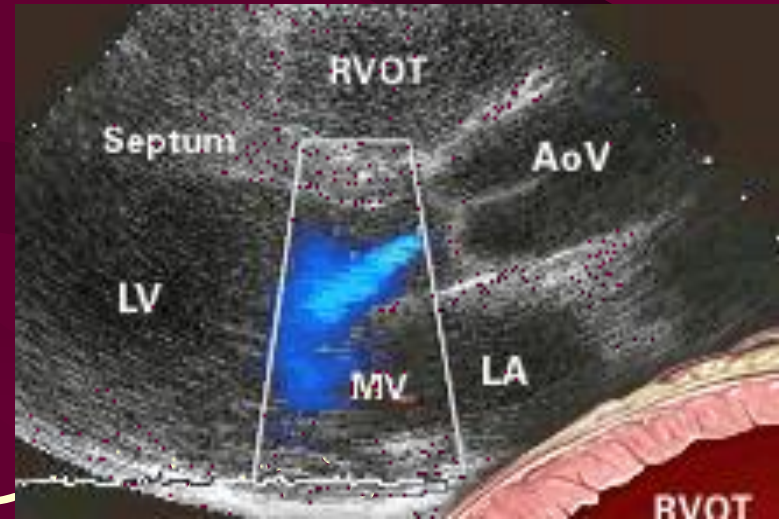
V₆

ECHOCG SIGNS

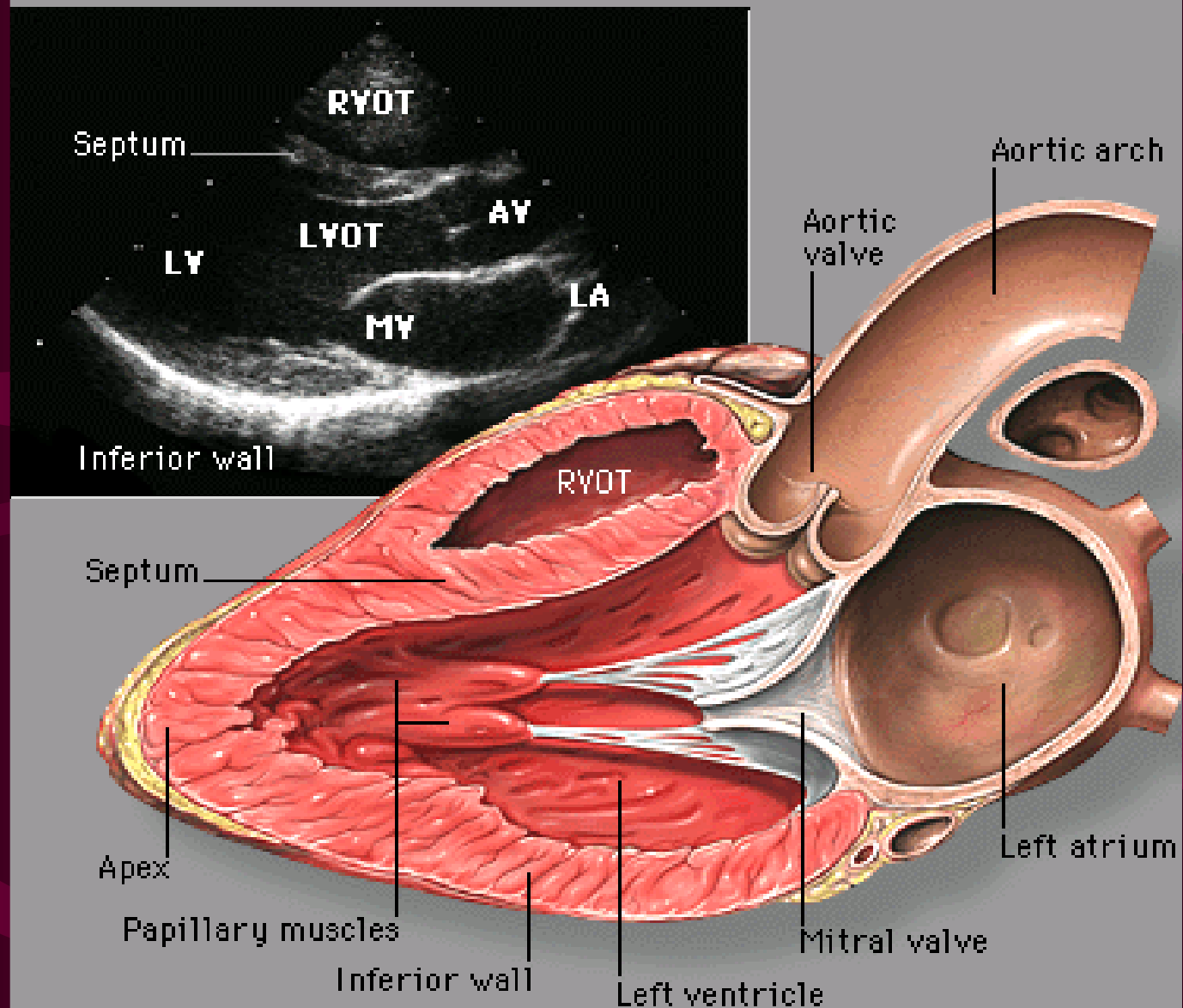
- A rapid fluttering of the anterior mitral leaflet produced by the impact of the regurgitant jet is a characteristic finding.
- Thickening and failure of the aortic leaflets closing also may be noted.
- Dilatation of the aortic annulus.
- LV performance is evaluated
 - Ejection Fraction

$$LVeDV - LVeSV / LVeDV = SV / L$$

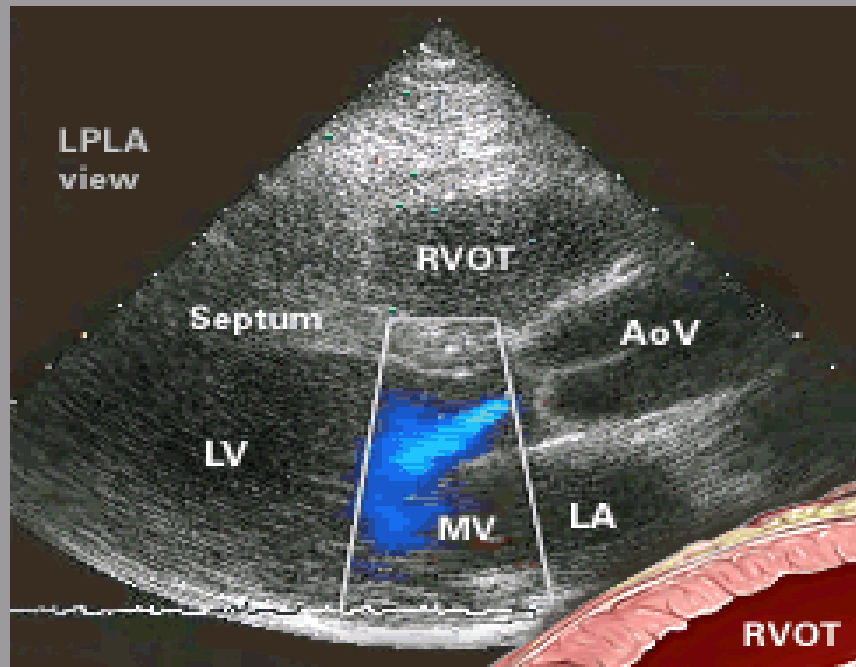
Color flow Doppler echocardiographic imaging is very sensitive in the detection of AR (blue jet) and its severity.



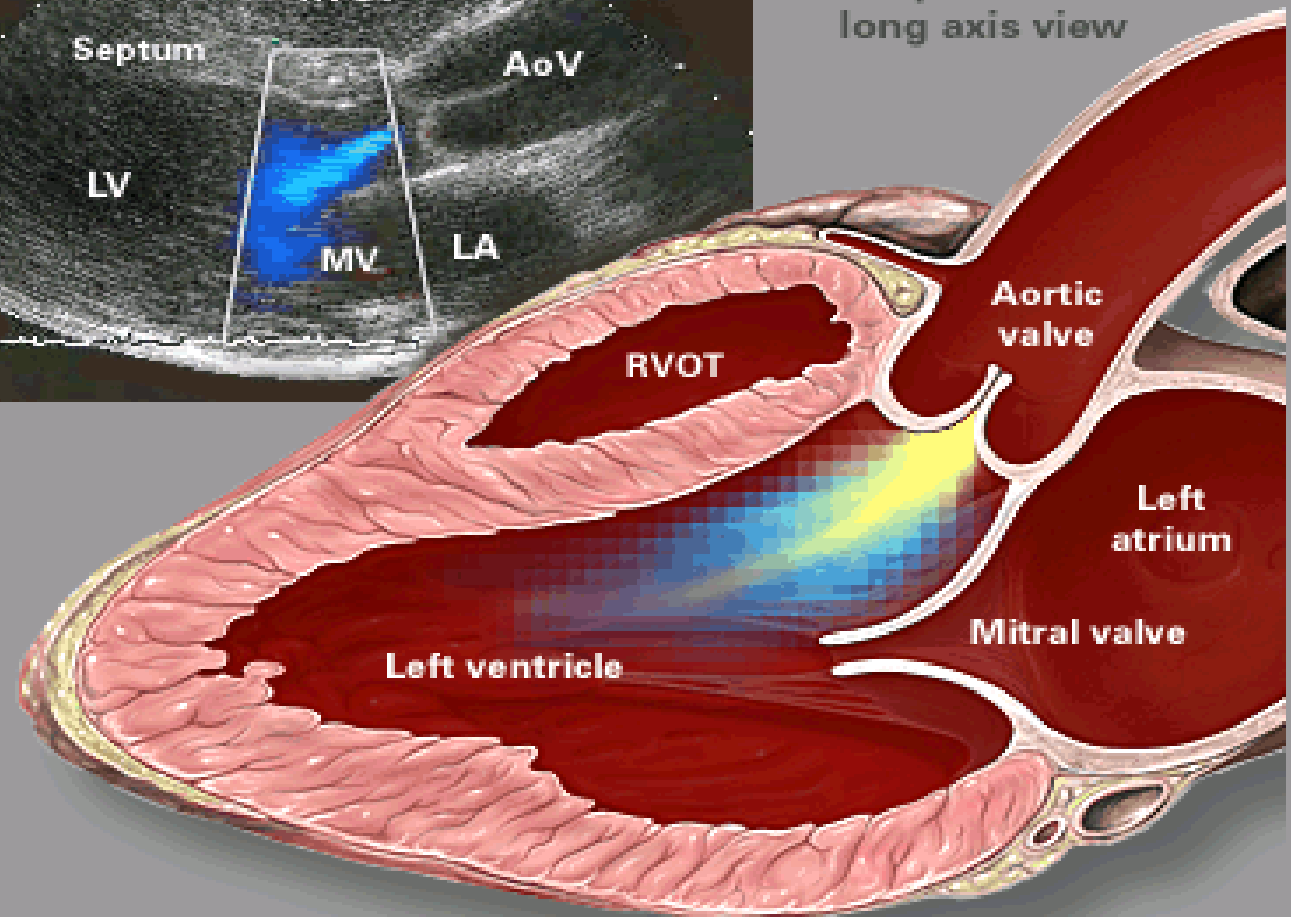
Left parasternal long axis view



Aortic regurgitation



Left parasternal long axis view



EcoTransesofageala.

Aorta este dilatata, disectie de aorta
+ regurgitatie aortica severa.

Sageata – aorta ascendenta dilatata

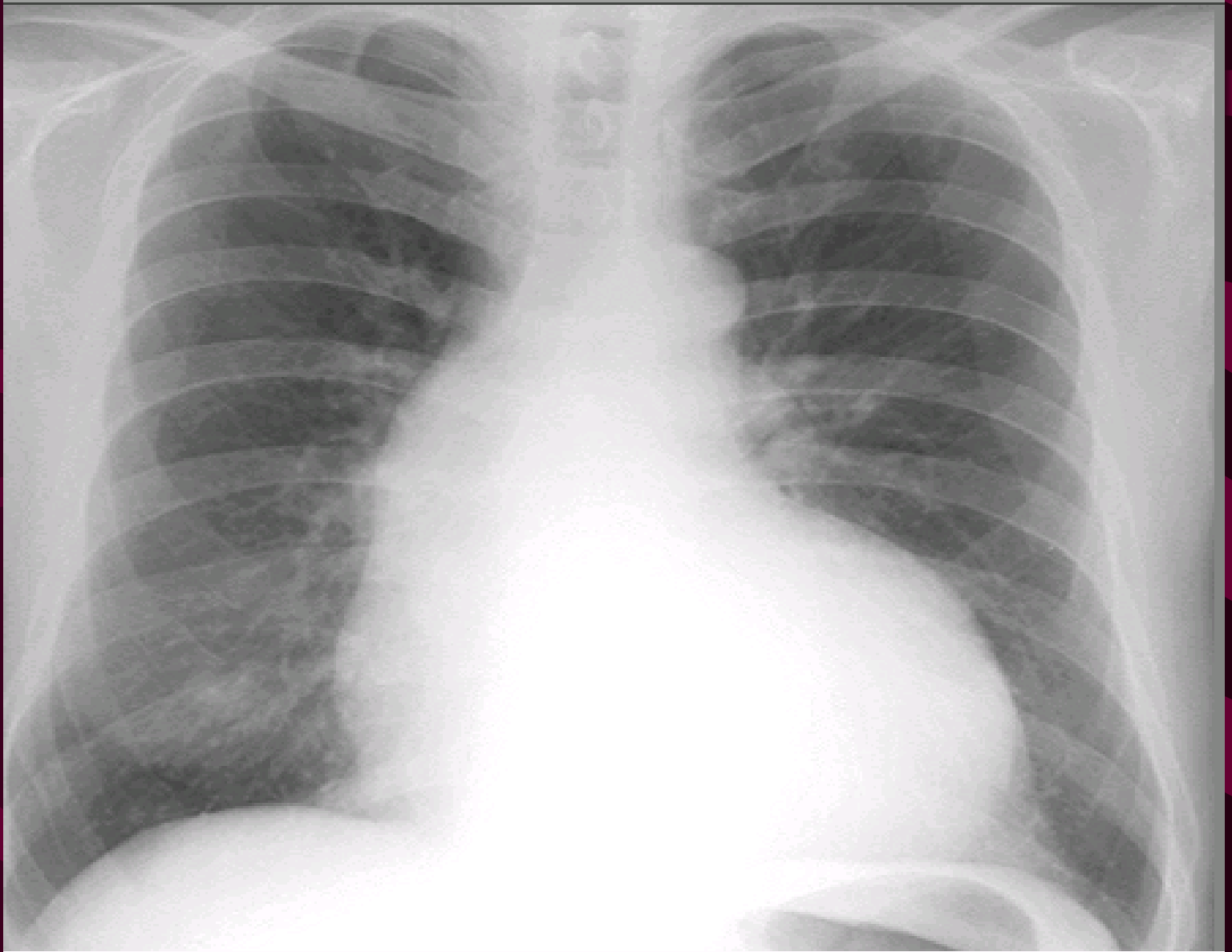


X-RAY

In severe chronic AR:

- the apex may be displaced downward and to the left in the frontal projection,
- the cardiac shadow extends below the left diaphragm.
- ascending aorta may be moderately dilated.
- aneurysmal dilatation of the aorta may be noted.
- Aortic configuration of the heart

Aortic regurgitation



CARDIAC CATHETERIZATION AND ANGIOGRAPHY

- is providing an accurate confirmation of the magnitude of regurgitation
- the LV performance,
- the condition of the coronary arterial bed .

Aortic Regurgitation

Treatment of Asymptomatic Aortic Regurg

Medical Therapy – treats the symptoms not the cause

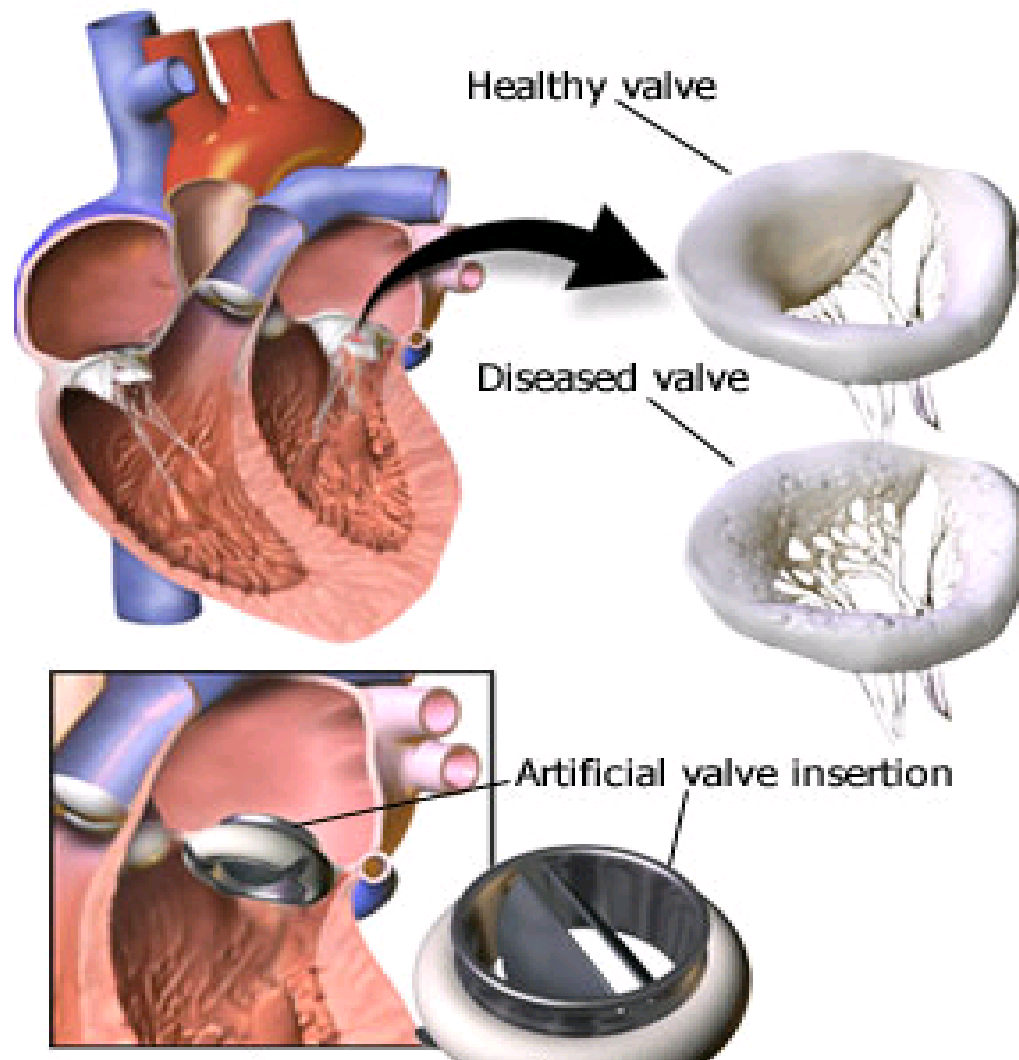
- **Serial Check ups with Echos** (eval EF, Severity AR)
- **SBE Prophylaxis**
- **Vasodilators** (Nifedipine, ACE-I)
- **Diuretics**

Treatment of Symptomatic Aortic Regurg

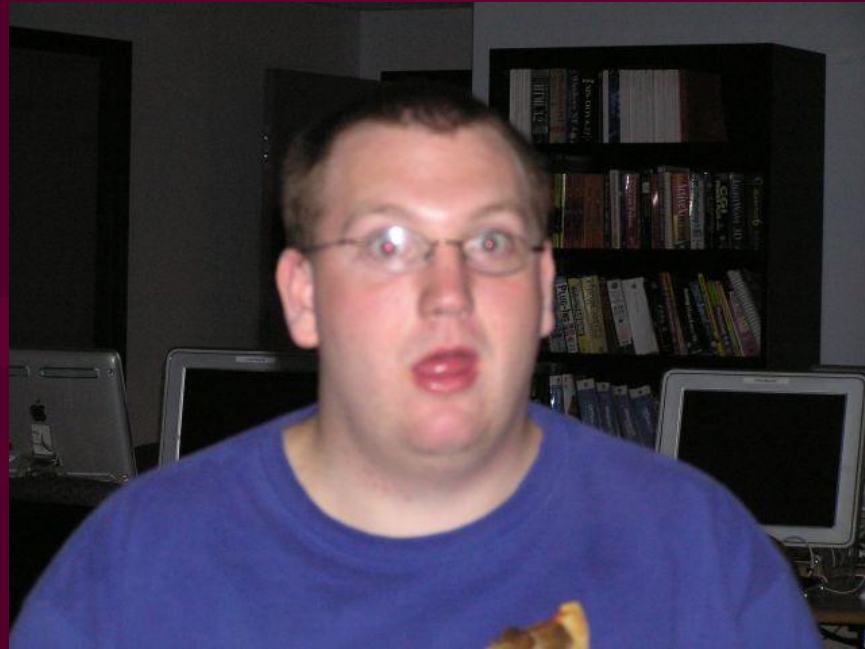
Aortic Valve Replacement

Bioprosthetic vs Mechanical AVR

Artificial Heart Valve



- Operation is to be undertaken at the optimal time, i.e., after the onset of LV dysfunction but prior to the development of severe symptoms.
- Operation should be carried out even in asymptomatic patients with progressive LV dysfunction and an $EF < 55\%$.



Spectrum of VHD – Right Sided VHD

Tricuspid Valve

Endocarditis

– IV drug abusers or inpt with IVs

Carcinoid HD - classically TS

TR – common, benign, may be secondary to
Pulm HTN

Pulmonic Valve

Pediatrics – **Pulm Stenosis**

Rheumatic HD – PI (Graham Steel Murmur)

**Right sided valvular lesions change in intensity with
inspiration**

Deep inspiration: thorax pressure↓
venous return↑, pulmonary circulation↑
clockwise rotation of heart make
murmur of TI, TS, PI↑

Expiration:

- Valsalva maneuver: thorax pressure↑
venous return↓ M of IHSS↑