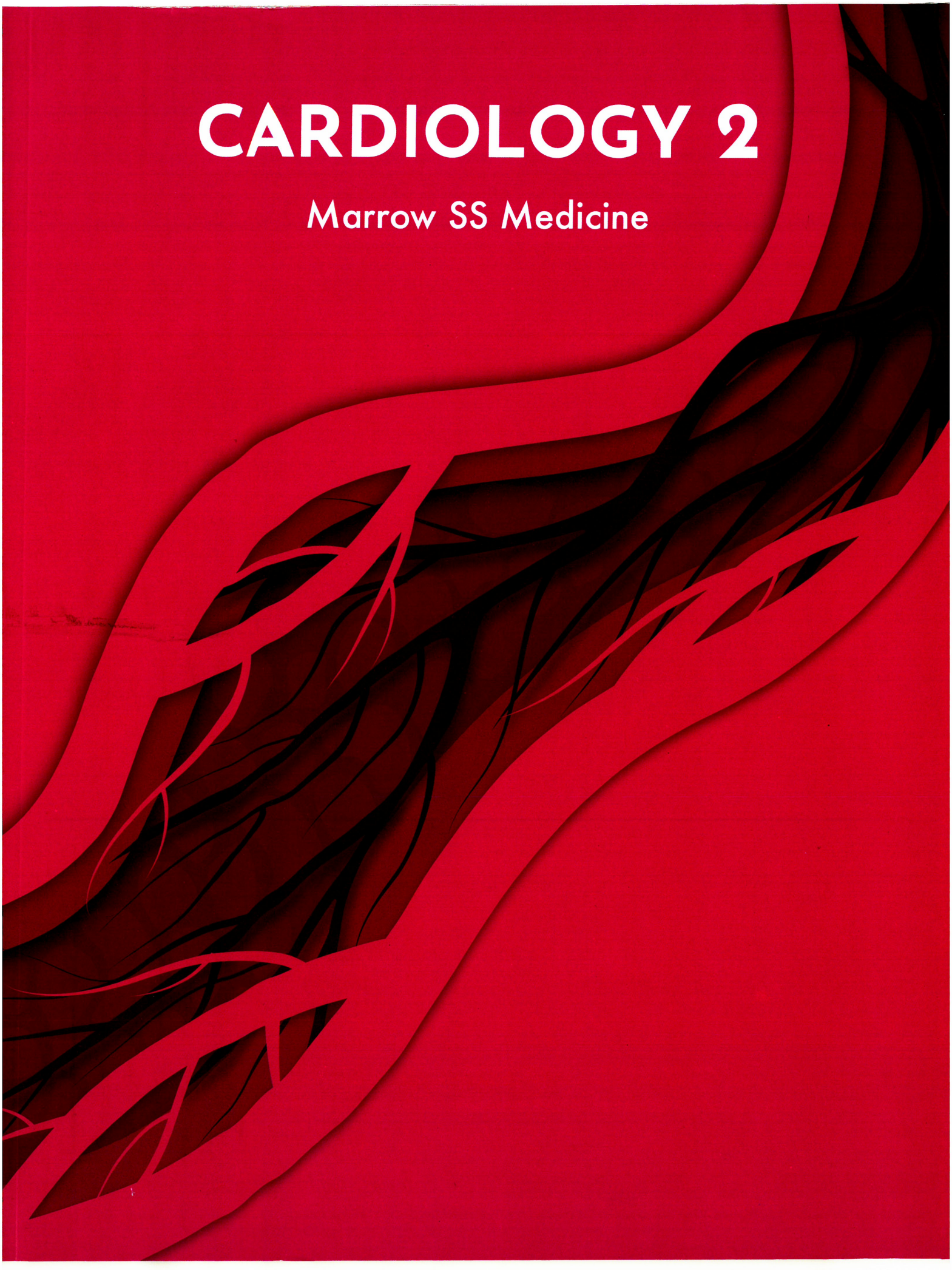
CARDIOLOGY 2

Marrow SS Medicine



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BICUSPID AORTIC VALVE

Introduction

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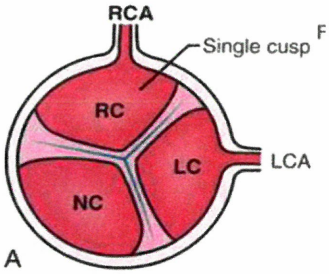
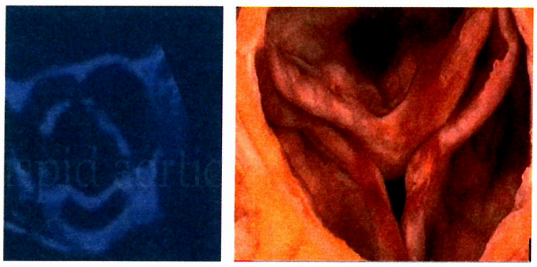
Normal aortic valve (AV) is tricuspid :

- Right cusp (RC).
- Non-coronary cusp (NC).
- Left coronary cusp (LC).



Right coronary artery (RCA) arises from RC (9 o'clock/11 o'clock position).
Left coronary artery (LCA) arises from LC (3'o clock position).



Tricuspid arotic valve

Developmental abnormalities

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Developmental abnormalities can cause :

- Unicuspid AV.
- Bicuspid AV.
- Quadricuspid AV.

Lesser the number of cusps, more is the predisposition to aortic stenosis (AS).
Higher the number of cusps, more is the predisposition to aortic regurgitation (AR).

Unicuspid AV/bicuspid : AS > AR.
Quadricuspid AV : AR > AS.

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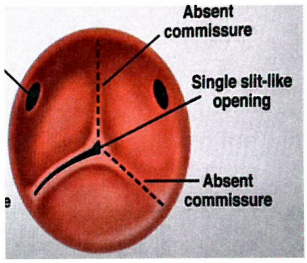
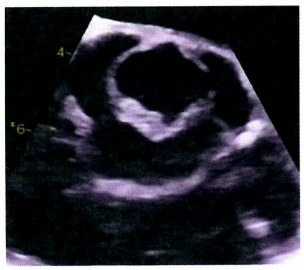
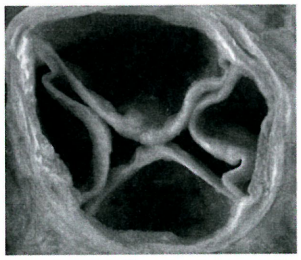
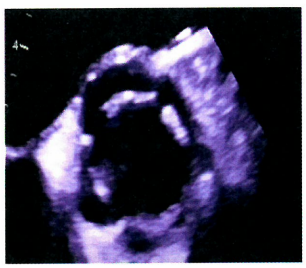
Unicuspid AV : Stenosis > regurgitation, very narrow (Slit like) opening of AV.





Quadricuspid AV : Stellate/star-shaped opening of AV.

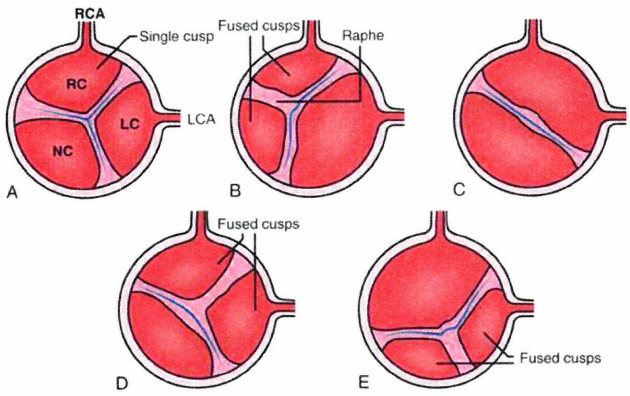




Bicuspid AV :

- m/c congenital heart disease.
- 1-2% of population
- male > Female (4 : 1).
- Autosomal dominance inheritance : NOTCH 1 gene.
- m/c cusp fusion : RC-LC fusion.

Presence of a raphe helps in the identification.



A : Normal AV.

B : RC-NC fusion.

C : RC-LC fusion (No raphe).

D : RC-LC fusion (m/c).

E : NC-LC fusion (rare).

Clinical features

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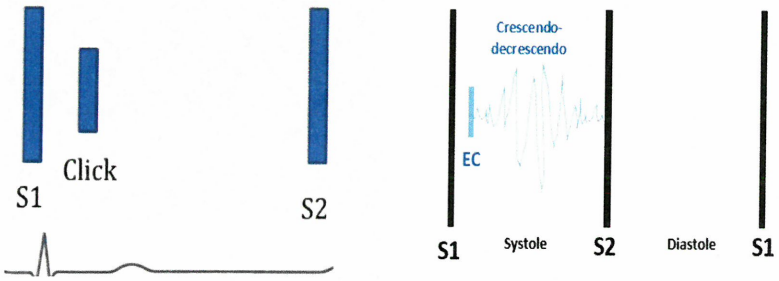
Characteristic of BAV : Ejection click.

Murmur of AS after the ejection click.



Problems of BAV : valvulopathy and aortopathy

00:05:25

Valvulopathy :

- AS : 40–50 years of age (BAV is an inherently stenotic valve).
  Mechanism of AS in BAV : Accelerated degeneration/ wear and tear due to its inability to handle the hemodynamic stress.
  Tricuspid calcific AS : Presents at 70–80 years of age.
- Mild-moderate AR : 20–30 years of age.
  Severe AR is rare, only in cases of valve IE/redundant valve leaflets.

Aortopathy :

- Aortic dissection.
- Aortic dilation.
- In all patients posted for AVR surgery, morphology of the AV should be known to rule out aortopathy.
- Aortopathy is screened by TEE/CT aortogram.
- Only 15% of patients with BAV will have a normal function in the fifth decade.

Severe AS in a BAV is different to a tricuspid valve, as BAV is associated with aortopathy :

- Entire aorta is abnormal : Especially proximal aorta and is vulnerable to accelerated degeneration of media causing aortic dilation.
- Abnormal fibrillin 1 and MMP release.

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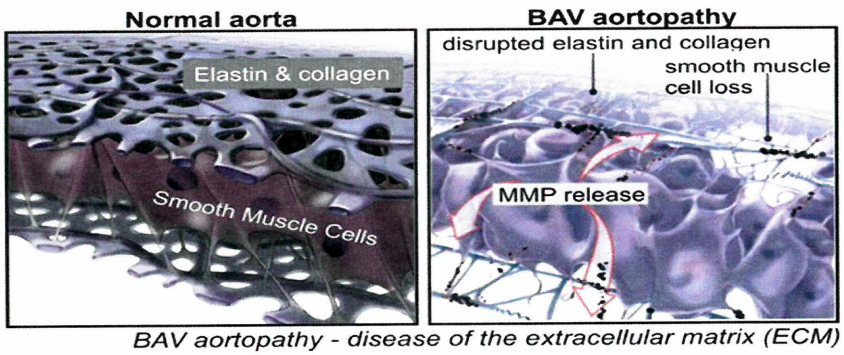
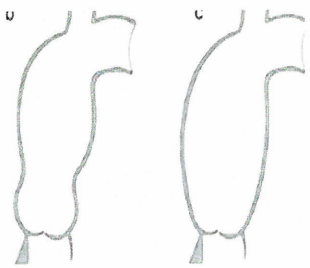
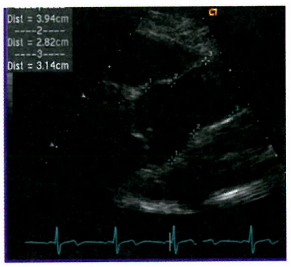


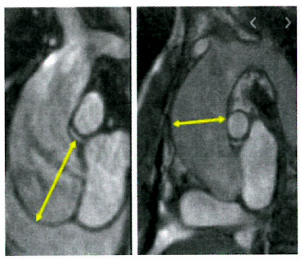
BAV aortopathy - disease of the extracellular matrix (ECM)

- Aortic dissection : < 4%.
- Aortic aneurysms : Proximal aorta.



Dilated aortic root





Proximal ascending aorta dilation

Aortopathy :

1. Increase in aortic diameters :
   - Sinuses.
   - Ascending aorta.
2. Increase in aortic stiffness.
3. Increase in rate of aortic enlargement.
4. Increase risk of aortic dissection.

Does BAV affect Quality of life (QOL)/Exercise capacity, life expectancy?

No

Management

00:11:47

In all cases of BAV look for aortopathy : TEE/CT aortogram/Cardiac MRI.

Only BAV : Does not change life expectancy.

Other presentations :

- BAV + Severe AS.
- BAV + Severe AR (Rare).

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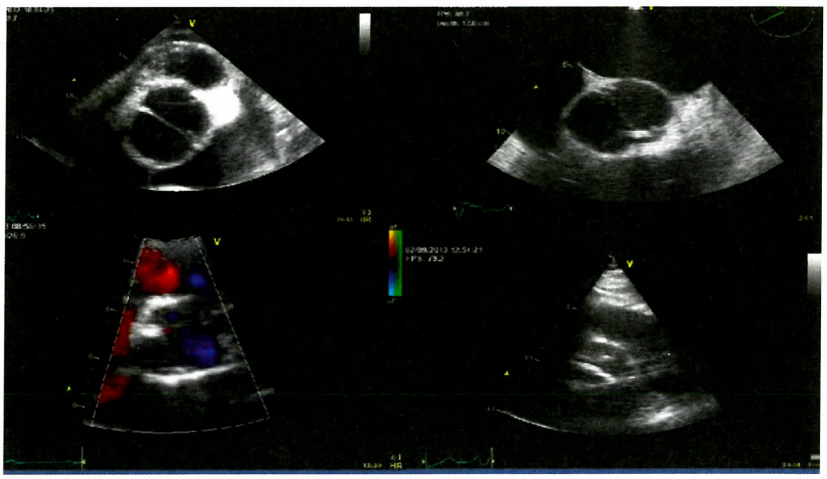
Aortic root diameter	Indication for elective aortic root surgery
≥ 55 mm	All- class I in ACC/AHA guidelines and 2a in ESC guidelines.
50-54 mm	If associated with any of : • Family history of dissection or unexplained sudden death. • Rapid growth progression of aortic dilatation. • Others (systemic hypertension, associated coarctation, female patient seeking pregnancy.
≥ 45 mm	If concomitant valve surgery is indicated (Bental procedure).

Case : 50 year old BAV on follow up :

ECHO :

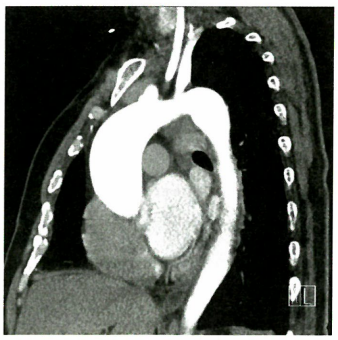


Right cusp + left coronary cusp fusion (Year 2000)
more restriction of valve (Year 2010)
marked calcification of valve (Year 2019)

CT aortogram :

Proximal aorta is markedly dilated

Ascending aorta 6 cm.



CT Aortogram

Management :

Patient undergoes AVR + Aortic root replacement (Bental procedure)

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Associations of BAV

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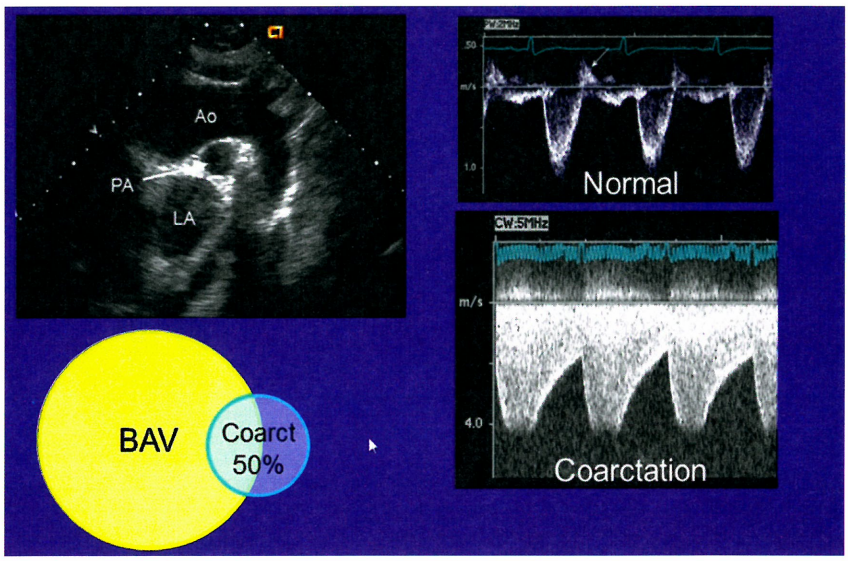
Condition	Incidence of BAV (%)	Comments
Coarctation of aorta (COA)	50	BAV confers increased risk of aortic complications.
Turner syndrome	30	Most frequent cardiac abnormality; right-left cusp fusion most common.
Supravalvular aortic stenosis	30	Usually part of William syndrome.
Subvalvular aortic stenosis	23	May result in significant aortic regurgitation.
Patent ductus arteriosis	Unknown	Usually diagnosed in childhood/ infancy.
Sinus of Valsalva aneurysm	15-20	Frequently asymptomatic; most commonly involves right coronary sinus.
Ventricular septal defect	30	May result in significant aortic regurgitation.
Shone complex	60-85	Series of left-sided aortic regurgitation.
Ascending aortic dilatation	Common	BAV is one of the most common associates.

BAV & COA : 50 % cases of COA have BAV.



AORTIC STENOSIS

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Introduction 00:00:21

3 types :

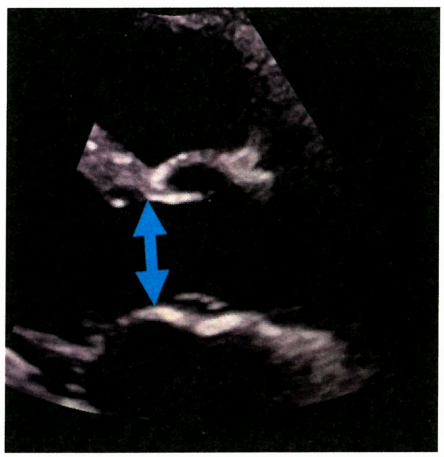
Based on site :

- Valvular AS.
- Subvalvular .
- Supravalvular AS.

Most common to least common occurance : Valvular AS > Subvalvular AS > Supravalvular AS.

Normal AV area- 3-4 cm^2 → SCTIMSTI8.

No significant pressure gradient across valve.



Aortic valve completely open : 2 leaflets parallel to each other
LV pressure equal to Aortic systolic pressure

Afterload :

- Resistance against which ventricle ejects .
- Normal → no much contribution to afterload by aortic valve.
- In aortic stenosis : LV struggles to eject against the valve → LV pressure increases → Pressure gradient +.
  - more the severity of stenosis more the pressure gradient.
  - Valve is the site of maximum resistance.
  - Time of peak ejection is increased.
  - Increased velocity of ejection as large pressure gradient called as AV_{max}.

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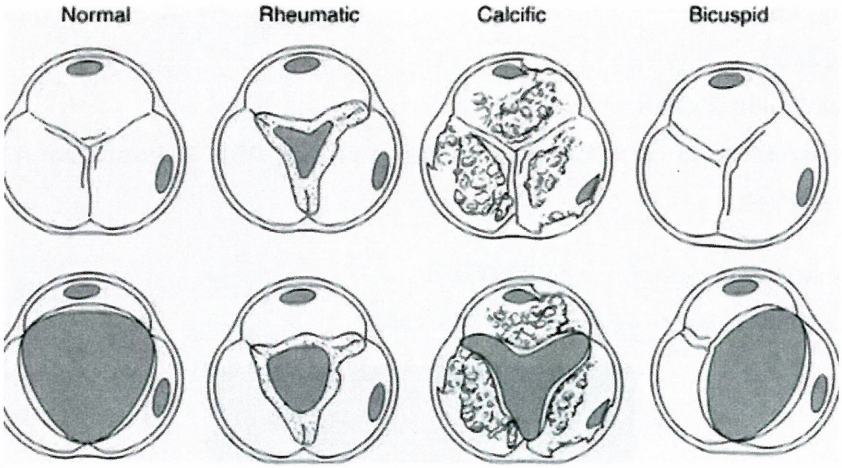
Causes

Degenerative/Calcific :

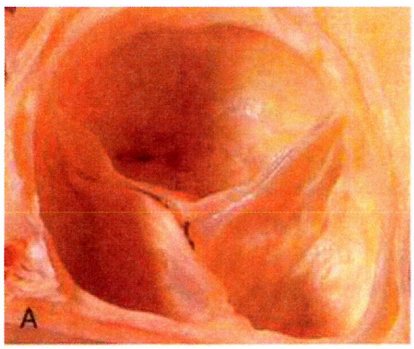
- Tricuspid / Bicuspid aortic valve-MC.
- Calcification prevents valve opening.

Rheumatic :

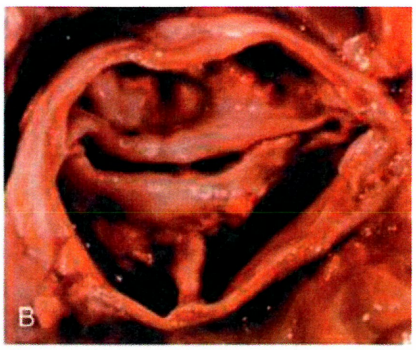
- Commissural fusion prevents valve opening.

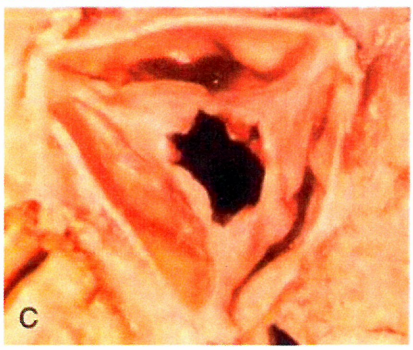


Normal

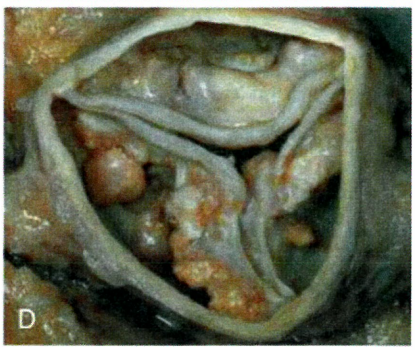


Bicuspid aortic valve with calcification





Rheumatic aortic Stenosis with Commissural fusion



Tricuspid aortic valve with calcification. No Commissural fusion

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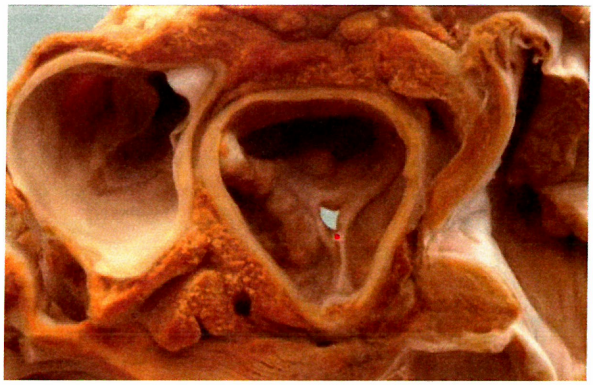
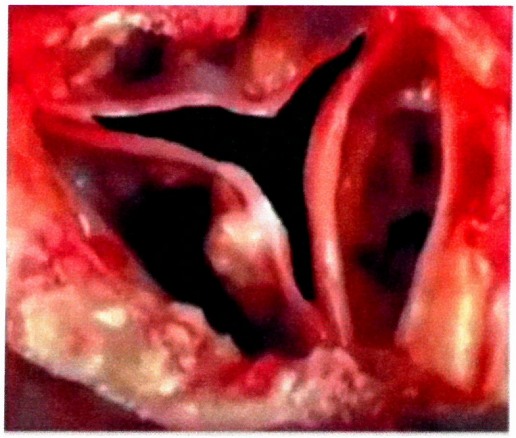
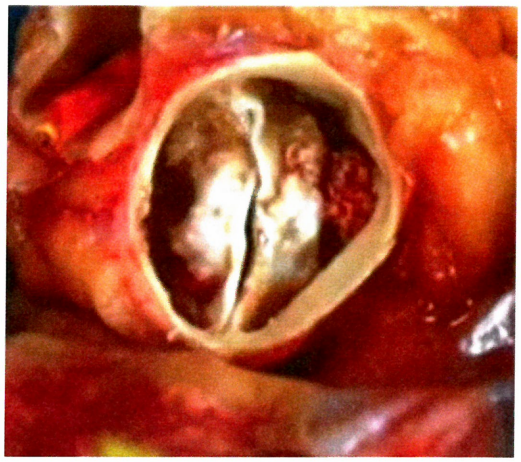
Severe AS due to Rheumatic



Senile/ Calcific / Degeneration In Tricuspid :



Senile/ calcific / degenerative in bicuspid :



Rare Etiologies :

- Congenital AS.
- Homozygous Type II hyperlipoproteinemia.
- Rheumatoid arthritis.
- Alkaptonuria/Ochronosis.

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Hemodynamics of AS

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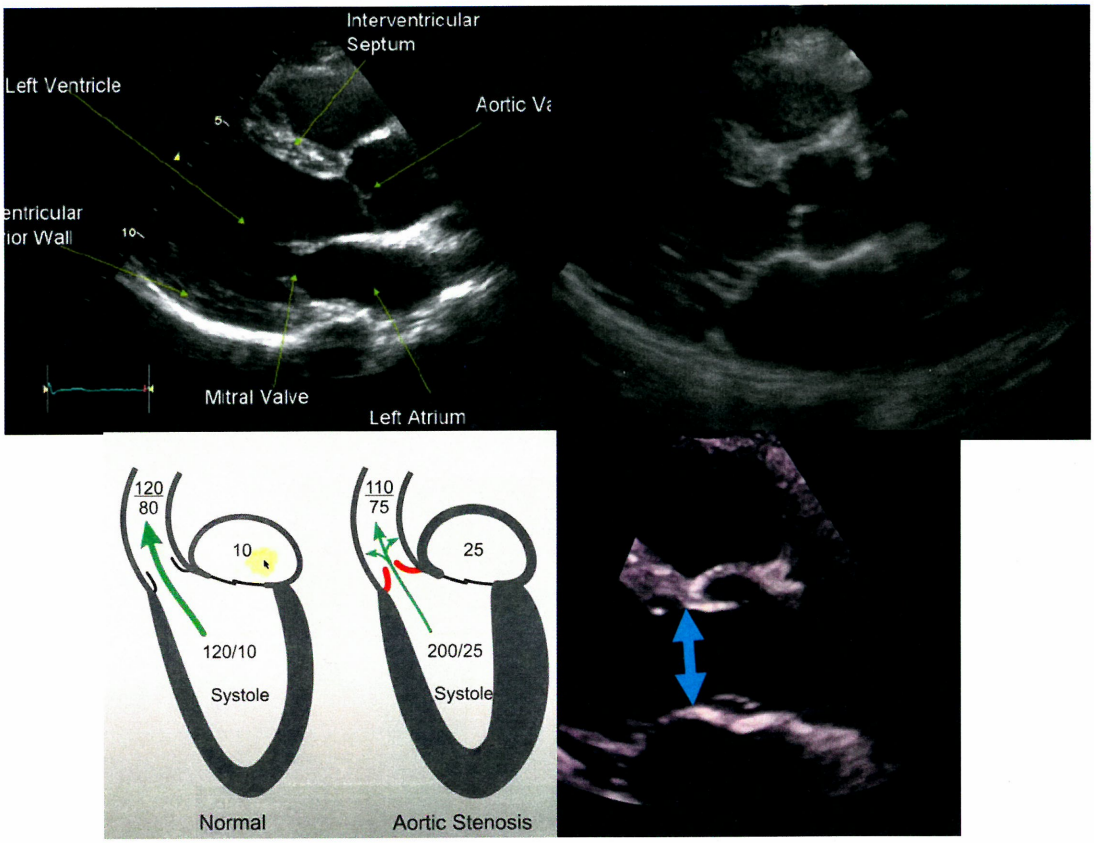

Note :

Increase in Pressure gradient(PG) as severity of AS increases.

- LVEDP = LAP = Pulmonary cappillary wedge presssure
  - LA pressure increases.
  - Normal : LA contribute 20-25 % of cardiac output .
  - In AS : LA contributes 40-50% of cardiac output → manifested as LV S4.
  - LVEDP increase → LAP increases → force of contraction increases as per Frank Starling law.

If this patient develops :

- AF → Hypotension /shock/ cardiac failure.
- Systemic HTN → mask AS by decreasing pressure gradient.
  - Hence Comment about the severity of AS only after controlling blood pressure.
- LVF → decreases pressure gradien further called as classical Low flow low gradient AS.
- Anemia → increased velocity across all chambers.

Note : ECHO does not estimate pressure gradient.

Increase in velocity indirectly aids in assessing pressure gradient.

Pressure gradient = $4V^2$.

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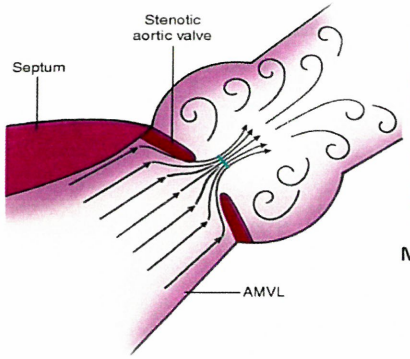
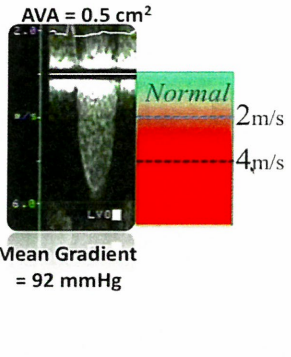
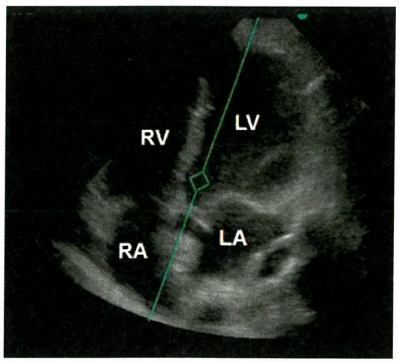
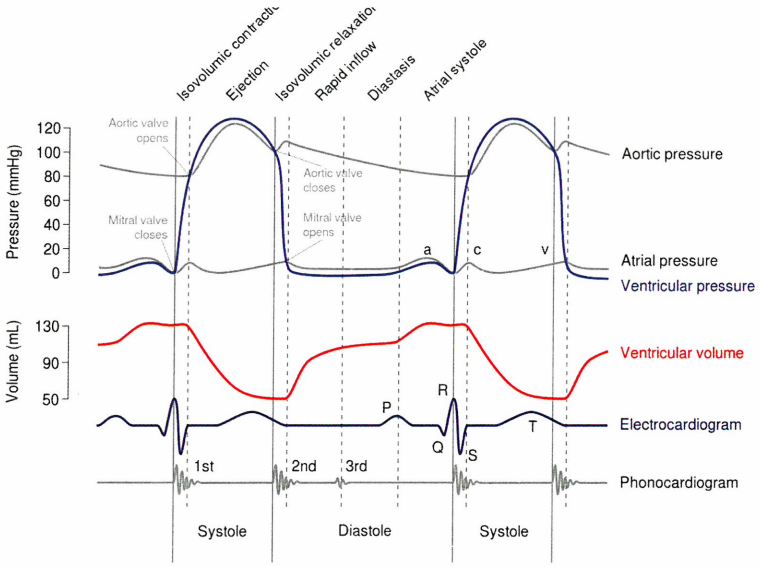


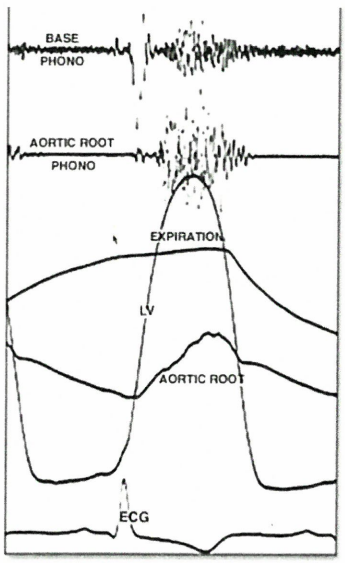




Severity of AS : severity is flow dependent.

	Mild	Moderate	Severe
Peak velocity (m/s)	2–2.9	3–4	>4
Mean gradient (mmHg)	<20	20–40	>40
Valve area (cm^2)	>1.5	1–1.5	<1
Indexed valve area (cm^2/m^2)	>0.85	0.60–0.85	<0.60
Velocity ratio	>0.50	0.25–0.5	<0.25





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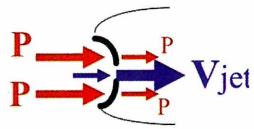
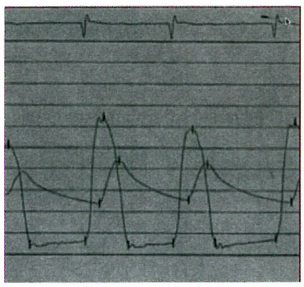
Note :

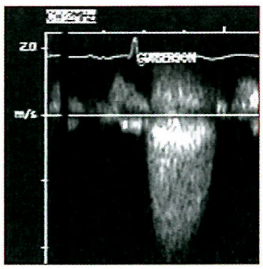
- Pulses parvus et-tardus : Low volume slow rising pulse.
- Heaving apex : higher amplitude + sustained apex beat.
- Longer late peaking murmur : more severe AS the later the peak & longer murmur.
  - Indicates severity.

Summary :

- Velocity increases as blood passes through the stenosis
- Pressure Gradient = $4V^2$

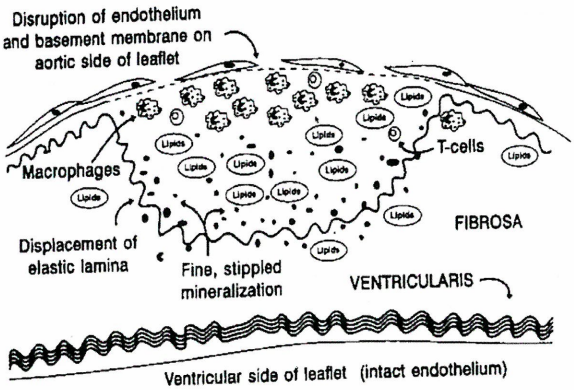




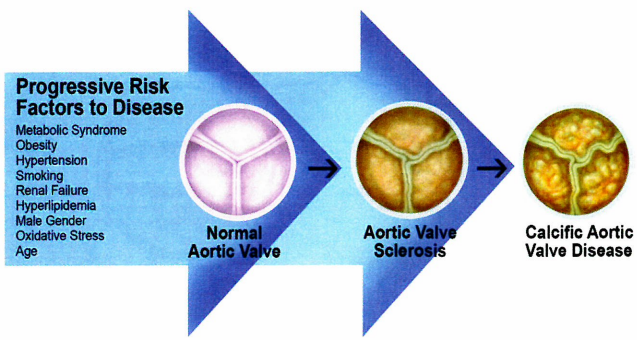


Pathogenesis 00:20:45

- Similar to atherosclerosis in pathogenesis & risk factors.
- statins given in severe AS : not useful.



Authors: Otto CM, Kuusisto J, Reichenbach DD, et al



Stages

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Stage	Definition	Description
A	At risk	Patients with risk factors for development of VHD
B	Progressive	Patients with progressive VHD (mild-to-moderate severity and asymptomatic)
C	Asymptomatic severe	Asymptomatic patients who have the criteria for severe VHD: C1: Asymptomatic patients with severe VHD in whom the left or right ventricle remains compensated. C2: Asymptomatic patients with severe VHD, with decompensation of the left or right ventricle.
D	Symptomatic severe	Patients who have developed symptoms as a result of VHD

Key: VHD = valvular heart disease.

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Stages of Valvular Aortic Stenosis (AS)

STAGE	DEFINITION	VALVE ANATOMY	VALVE HEMODYNAMICS	HEMODYNAMIC CONSEQUENCES	SYMPTOMS
A	**At risk of AS**	Bicuspid aortic valve (or other congenital valve anomaly) Aortic valve sclerosis	Aortic Vmax <2 m/sec	None	None
B	**Progressive AS**	Mild to moderate leaflet calcification of a bicuspid or trileaflet valve with some reduction in systolic motion *or* Rheumatic valve changes with commissural fusion	***Mild AS:*** Aortic Vmax 2.0-2.9 m/sec or mean ΔP <20 mm Hg ***Moderate AS:*** Aortic Vmax 3.0-3.9 m/sec or mean ΔP 20-39 mm Hg	Early LV diastolic dysfunction may be present. Normal LVEF	None
C	**Asymptomatic severe AS**				
C1	Asymptomatic severe AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	***Severe AS:*** Aortic Vmax ≥4 m/sec or mean ΔP ≥40 mm Hg AVA typically is ≤1 cm^2 (or AVAi ≤0.6 cm^2/m^2) Very severe AS is an aortic Vmax ≥5 m/sec, or mean ΔP ≥ 60 mm Hg	LV diastolic dysfunction Mild LV hypertrophy Normal LVEF	None Exercise testing is reasonable to confirm symptom status
C2	Asymptomatic severe AS with LV dysfunction	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	Aortic Vmax ≥4 m/sec or mean ΔP ≥40 mm Hg AVA typically is ≤1 cm^2 (or AVAi ≤0.6 cm^2/m^2)	LVEF <50%	None
D	**Symptomatic severe AS**				
D1	Symptomatic severe high-gradient AS	Severe leaflet calcification or congenital stenosis with severely reduced leaflet opening	***Severe AS:*** Aortic Vmax ≥4 m/sec, or mean ΔP ≥40 mm Hg AVA typically is ≤1 cm^2 (or AVAi ≤0.6 cm^2/m^2), but may be larger with mixed AS/AR	LV diastolic dysfunction LV hypertrophy Pulmonary hypertension may be present.	Exertional dyspnea or decreased exercise tolerance Exertional angina Exertional syncope or presyncope
D2	Symptomatic severe low-flow, low-gradient AS with reduced LVEF	Severe leaflet calcification with severely reduced leaflet motion	AVA ≤1 cm^2 with resting aortic Vmax <4 m/sec, or mean ΔP <40 mm Hg Dobutamine stress echo shows AVA ≤1 cm^2 with Vmax ≥4 m/sec at any flow rate	LV diastolic dysfunction LV hypertrophy LVEF <50%	HF Angina Syncope or presyncope
D3	Symptomatic severe low-gradient AS with normal LVEF or paradoxical low-flow severe AS	Severe leaflet calcification with severely reduced leaflet motion	AVA ≤1 cm^2 with aortic Vmax ≤4 m/sec, or mean ΔP <40 mm Hg AVAi ≤0.6 cm^2/m^2 Stroke volume index <35 mL/m^2	Increased LV relative wall thickness Small LV chamber with low stroke volume	HF Angina Syncope or presyncope

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Progression of AS determined by :

- LVOTO vs RVOTO :
  - LVOTO : mild AS always progress to severe AS.
  - RVOTO : mild PS may not worsen for years.
- Aortic jet velocity AVmax :
  - Strongest determinant of progression.
  - Higher velocity → faster progression.
- Severity of AV calcification :
  - Severity increases → rapid progression.
- Uncontrolled risk factors.

Rate of progression :

- Highly variable and difficult to predict in a given individual.
- Aortic sclerosis : 2.5% progress to severe AS over 8 years
- Annual decrease in AVA : 0.12 cm^2/yr.
- Annual increase in jet velocity : 0.32 m/sec/yr
- Annual increase in mean gradient : 7 mm Hg/yr.

Clinical features

00:27:12

Usually in seen in older age : Pseudosymptomatic.
Identified by tread mill test.

Symptoms

- Asymptomatic : rare
- Classical symptoms
  - Angina : survives for 5y
  - Syncope : survives for 3y
  - Dyspnea : survives for 2y.

  } referrable to LV outflow tract obstruction
  - Stroke/MI : calcium emboli
  - IE : rare.

GI bleed in setting of severe AS is due to :

- Aspirin induced gastritis.
- Colonic diverticulosis.
- Colonic polyps.
- Colonic angiodysplasia : Heyde's syndrome.

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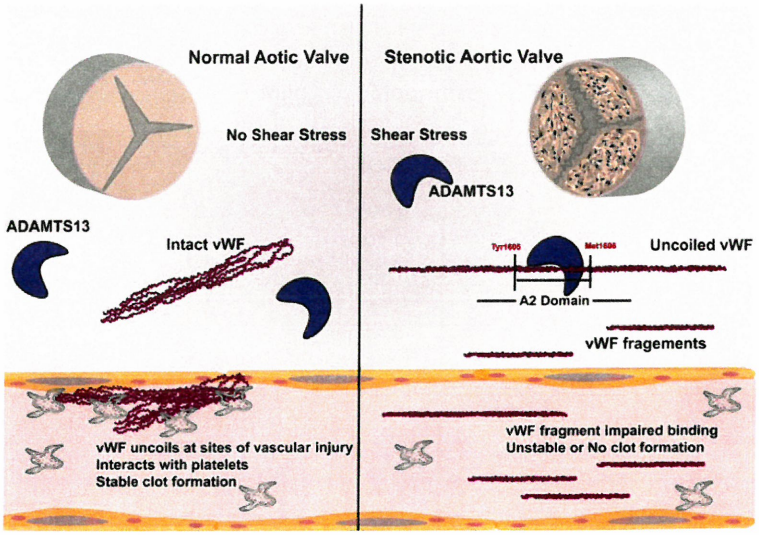
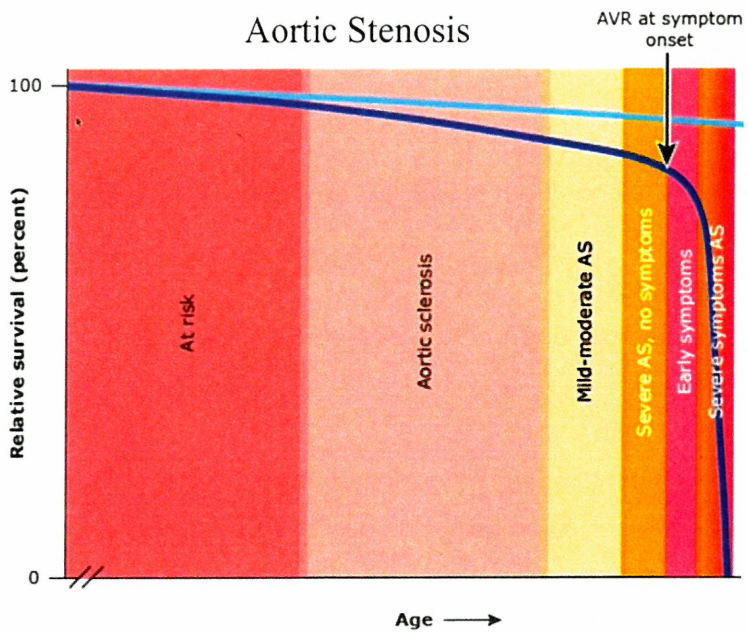


multimeric VWF → passes → uncoiled → bleeding

- Correlate with severity of AS & correctable by AVR.



AS have Long course of time until symptoms develop.
LV compensates → LV fails/decompensate → symptoms.

Signs

- Pulses parvus et tardus :
  - most specific.
  - Palpated over carotids.

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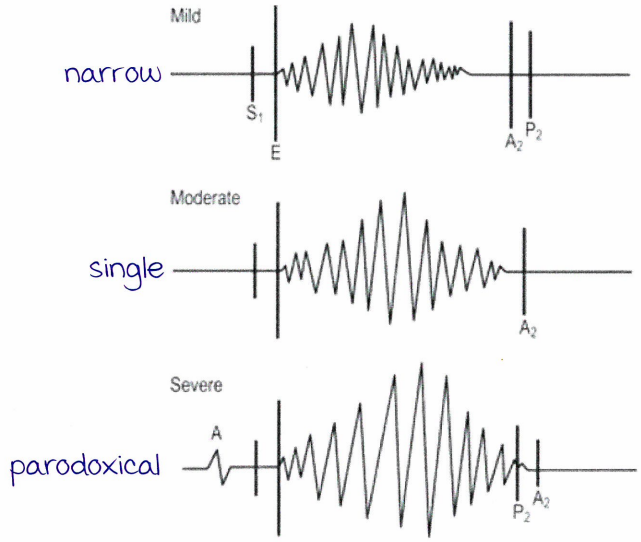
- Carotid shudder :
  - Thrill palpated over carotids.
- Heaving apex
  - Long,louder, late peaking murmur



Gallavardin phenomenon

- Split of S2 :
  - As per severity of AS .
  - Severe AS : Longer period for LV emptying, hence AV closure delayed.

Initially narrowed split → single → parodoxical.

- Intensity of S2 : soft
- LVS4 : due to diastolic dysfunction and atrial contraction.

Gallavardin phenomenon :

- Typical AS murmur is mixed frequency with low & high frequency.
- In calcific AS : Selective propagation of high frequency component to apex.
  - Longer & musical quality +.
  - Mimics MR.

Which of the following increase in intensity following VPC/long cycle of AF :

- MR
- TR

  — Resistant to cycle length changes
- AS/PS : increases
- HOCM : decreases

ECG :

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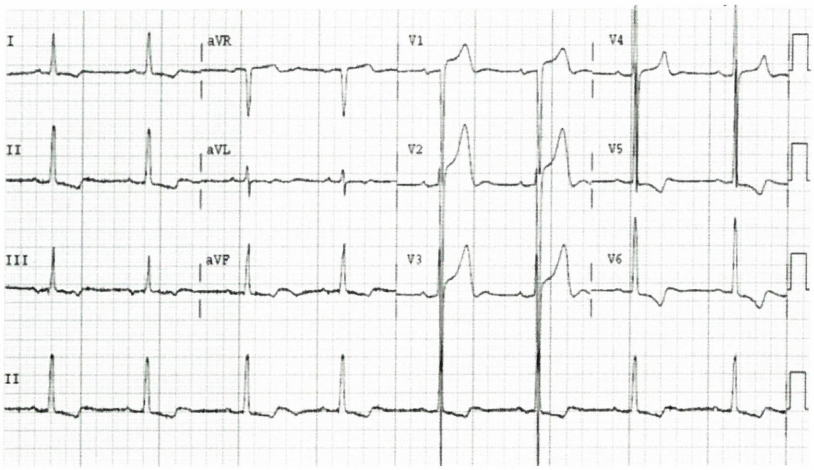
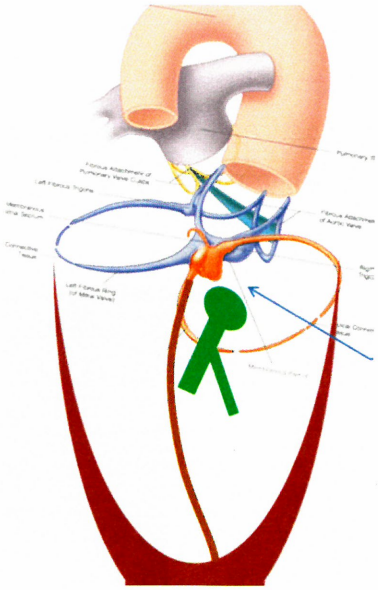


AV Blocks are common in AS as :

- AV ,AV node & AV bundle are close to each other.
- The contraction - relaxation → some degree of mechanical erosion.

Calcification can easily extend to conducting system.



ECHO :

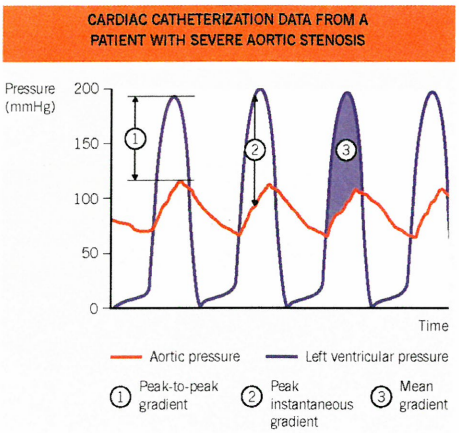
- Assess LV function
- Assess AV opening, morphology.
- Assess transaortic jet velocity.
- Assess Pressure gradient.
- Calculate valve area - Continuity equation.

Exercise ECG :

- Symptomatic severe AS : Absolute CI
- Indication for TMT in AS : differentiate between pseudoasymptomatic & asymptomatic.

Cardiac CT :

- Aortopathy in BAV : Before surgery always assess for morphology of the valve as in BAV aortopathy is possible.
- Pre TAVR evaluation.
- Assess valve calcification : Predicts progression, low flow low gradient AS



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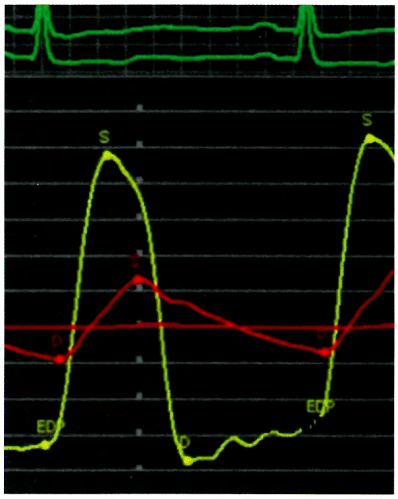
- Peak to peak gradient :
  - Aortic peak & LV peak.
  - 75 mm.
- Peak instantaneous gradient :
  - Maximum possible peak.
  - Maximum possible difference - between aortic & LV pressures.
  - 105 mm.
- Mean gradient : Area under curve
  - 55 mm.
  - Peak instantaneous gradient > Peak to peak gradient.

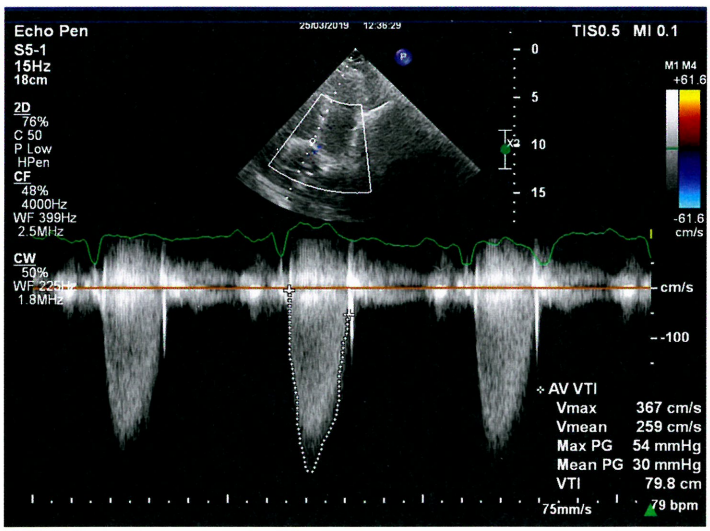


Cath

Measure P-P gradient, Mean gradient.

ECHO : measures Peak instantaneous gradient, Mean gradient.

Physiological marker : Peak instantaneous gradient.

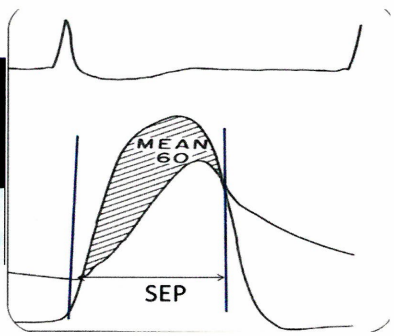


Calculation of AV area in cath lab :

- Gorlin formula :

$$AVA = \frac{CO / (SEP \times HR)}{44.3 \times \sqrt{\text{Mean Gradient}}}$$

$$\text{Aortic Valve Area} = \frac{\text{Cardiac Output}}{SEP \times HR \times 44.3 \sqrt{G}}$$



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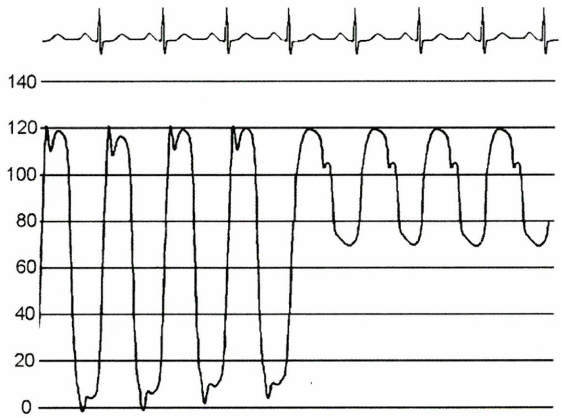
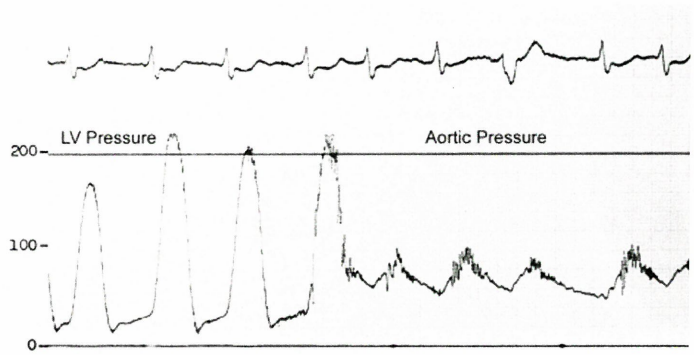
- Hakki formula :

$$\text{Aortic Valve area (in cm}^2\text{)} \approx \frac{\text{Cardiac Output}(\frac{\text{litre}}{\text{min}})}{\sqrt{\text{Peak to Peak Gradient (mmHg)}}}$$

- Normal LV – Ao pull back :

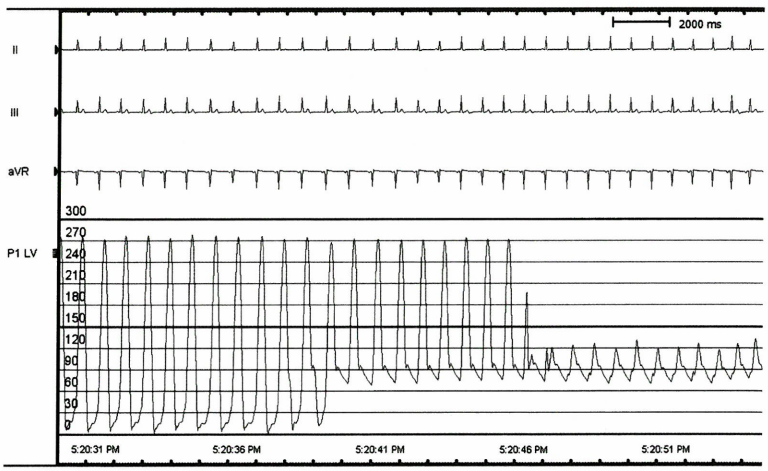


- Pull back- LV-Ao :



Continuous Withdrawal Tracing from LV to Aorta

- Pull back pressure tracing – LV Ao :



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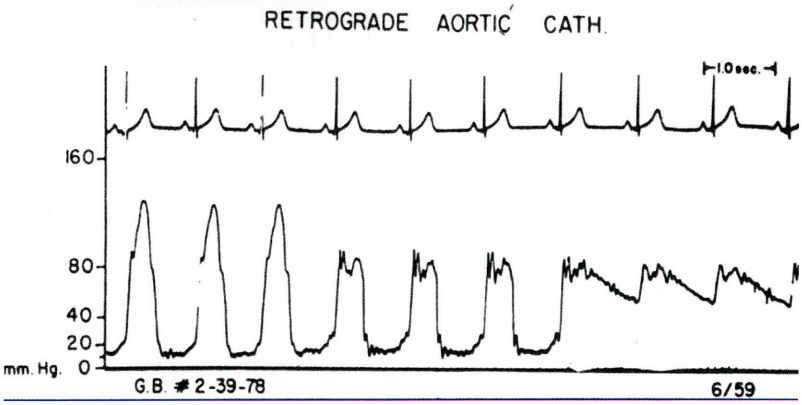
- This tracing associated with AS.
- HOCM.
- Rib notching.
- Elfin facies : Supraventricular AS.



Intraventricular pressure curve

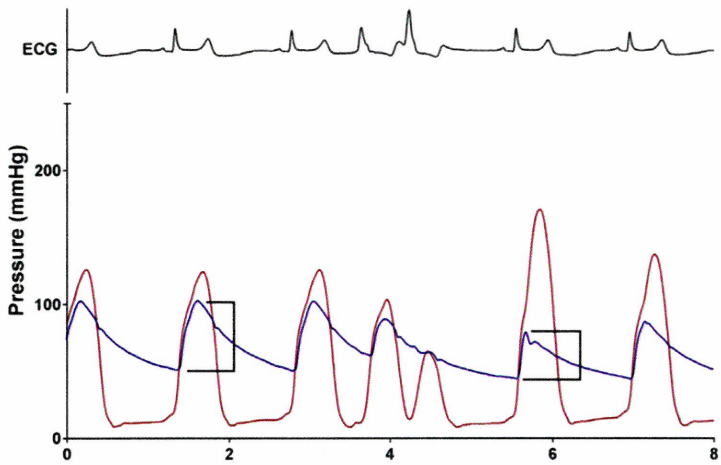
HOCM : spike & dome pattern.

Dynamic obstruction.

If no spike & dome pattern : subvalvular AS.

Simultaneous pressure tracing :

- 2 pressure curves : 1 cather in LV & other in aorta.
- Both catheter simultaneoulsly measures pressure.
- LV : Low diastolic pressure.
- Aorta : High diastolic pressure.



Brockenbrough Braunwald Morrow sign :

- Post extra systole beat .
- Pulse pressure decreasing post VPC beat → HOCM.
- Pulse pressure remain same/increasing post VPC beat → AS.

Hemodynamic subsets of AS

----- Active space -----

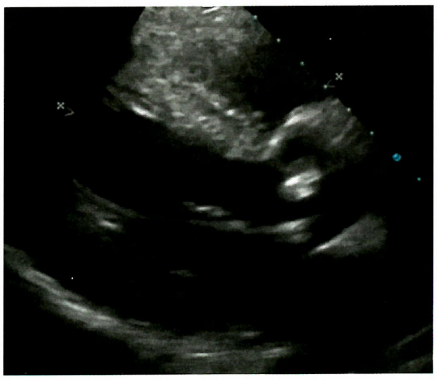
	Mild	Moderate	Severe
Peak velocity (m/s)	2–2.9	3–4	>4
Mean gradient (mmHg)	<20	20–40	>40
Valve area (cm^2)	>1.5	1–1.5	<1
Indexed valve area (cm^2/m^2)	>0.85	0.60–0.85	<0.60
Velocity ratio	>0.50	0.25–0.5	<0.25

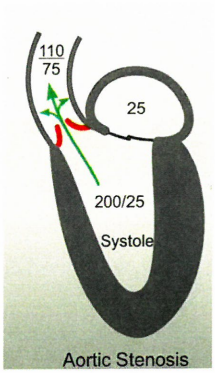
Adapted from Nishimura *et al.*[1]

In normal or high flow states (SV > 35mL/m^3) :

- Measurement error
- Indexed AVA to BSA.
- HTN.

Normal flow high gradient AS





Routinely seen condition.

AVA <1 cm^3.

Vmax >4m/sec.

PG → 40 mm Hg.

Low flow Gradient AS :

- Classical LFLG → AS+ HFrEF/ LV SD.
- Paradoxical LFLG → AS + HFpEF/ LV DD.

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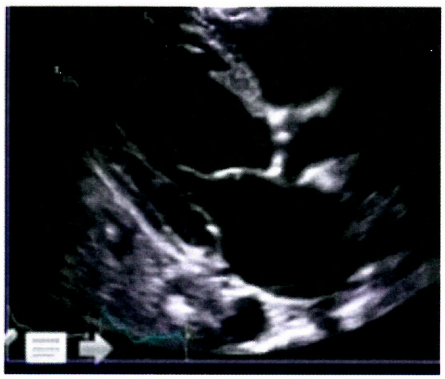
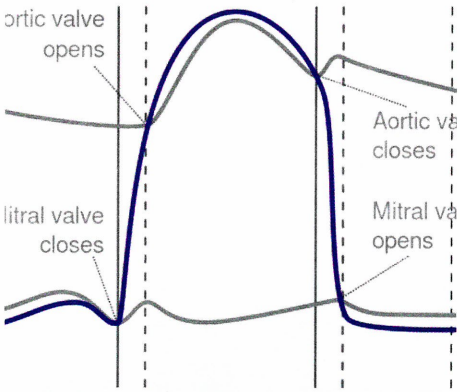
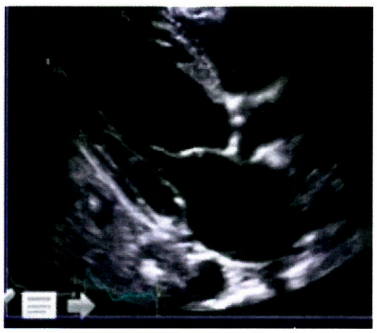
Classical low flow low gradient AS :

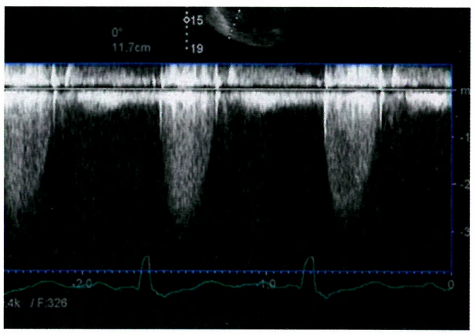


- AVA > 1 cm^2.
- Vmax < 4m/sec.
- PG < 40mm hg.

AV opens when LV pressure exceeds the aortic pressure.







Is it severe AS with LV dysfunction ?

OR

Is it DCM + severe LV dysfunction + moderate AS ?

Both the above conditions give the same values :

- AVA → 0.7 cm^2.
- Vmax → 2.7m/sec.
- PG → 30 mm Hg.

Giving ionotrope (Dobutamine) use dobutamine stress test.