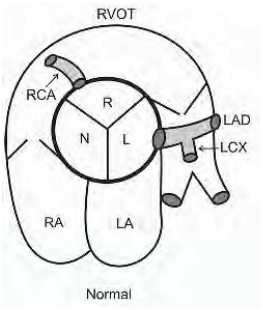
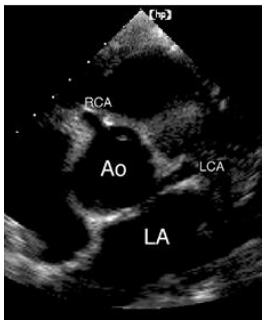
AORTIC VALVE DISEASE

ANATOMY OF THE AORTIC VALVE



- The aortic valve has three cusps of equal size, which open widely during systole and close in a Y-shaped appearance in diastole.
- Each cusp is surrounded by (and named after) an outpouching in the aortic root called "sinus". The sinuses support the cusps during systole and provide a reservoir of blood to augment coronary flow during diastole.
- The anatomy of aortic valve and root is best studied from the parasternal view
- The right and left sinuses give rise to the right and left main coronary arteries respectively (can be seen in the PSAX at 4 O'clock & 11 O'clock respectively) and the third sinus is conveniently termed the non-coronary aortic sinus.

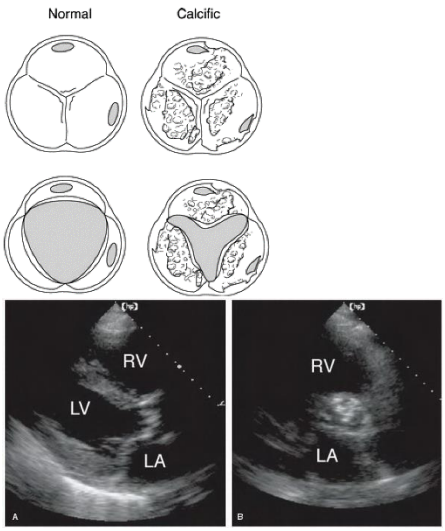


- In PSAX, the right coronary cusp is anterior, closest to the RV and the left coronary cusp is posterior and leftward, closest to the PA. The third, or noncoronary cusp, is posterior and rightward, just above the base of the IAS.
- In PLAX, the upper (anterior) aortic cusp is the RCC and the lower (posterior) one is the non-coronary cusp
- The AV supports its own structure and does not have papillary muscles.
- It is most commonly tricuspid but it may be bicuspid (1-2%), unicuspid (0.02%) and quadricuspid (0.0003%).

AORTIC STENOSIS

COMMON CAUSES OF AORTIC STENOSIS- calcific degeneration of aortic leaflets is the most common cause of aortic stenosis in patients >70 years old in developed countries. The leading cause of aortic stenosis in younger patients is bicuspid aortic valve.

(Senile) calcific degeneration of the AV is one of the commonest causes of AS. The AV commonly calcifies in a process similar to atherosclerosis. This is characterized by thickening and calcification, beginning at the base of the cusps and most prominent in the central and basal parts of the cusp (no commissural fusion) resulting in a stellate-shaped systolic orifice. The early stage of this process is often referred to as 'aortic sclerosis'.

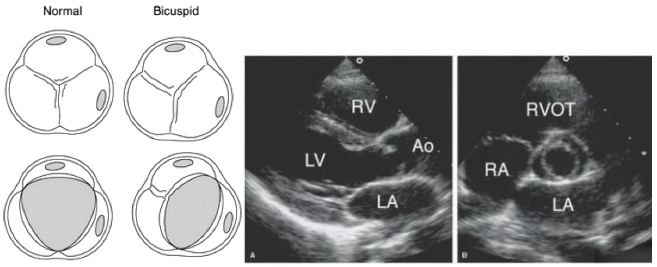


A: The long-axis view reveals an echogenic and very immobile aortic valve.
B: The corresponding short-axis view suggests a high degree of calcification of the valve and minimal mobility during systole.

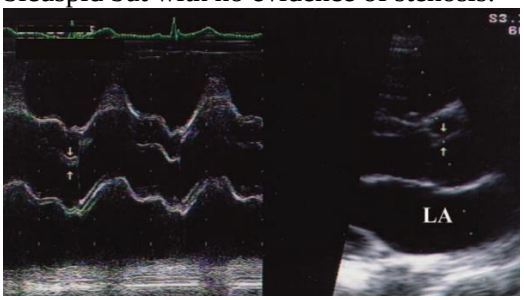
Bicuspid AV has two cusps, usually of unequal size. It often results from fusion of the right and left coronary cusps resulting in a larger anterior and smaller posterior cusp with both coronary arteries arising from the anterior cusp (~80% of cases). Fusion of the right and non-coronary cusps resulting in larger right than left cusp, with one coronary artery arising from each cusp is less common (~20% of cases). Fusion of the left and non-coronary cusps and valves with two equally sized cusps ("true" bicuspid valve) are rare.
Fibrosis typically starts in a patient's teens, with gradual calcification from their 30s onwards. Stenosis typically results from superimposed calcific changes, which often obscures the number of cusps. Patients who require surgery for bicuspid AS do so on average 5 years earlier than those with calcific tricuspid AV.
Diagnosis: is most reliable in the **PSAX** view when the two cusps are seen in systole with only two commissures framing an elliptical systolic orifice. Diastolic images may mimic three cusps when a raphe is present.
PLAX (2D & M-mode) may show (less specific):

- Asymmetric (eccentric) closure line.
- Systolic doming (similar to diastolic doming of mitral leaflets in mitral stenosis).
- Diastolic prolapse of one or both of the cusps.

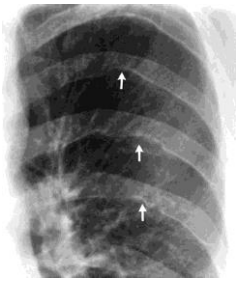
– **Pseudo-bicuspid ('functionally' bicuspid)** valves have three cusps, but with fusion of two of the cusps.
– **The prevalence** of bicuspid aortic valve is **1–2%** of the population. It is the single most common congenital cardiovascular anomaly and is often familial.
– Bicuspid aortic valves are associated with an increased risk of stenosis, regurgitation, aortic aneurysms and coarctation
– **50% of cases of severe AS in adults** are thought to be due to Bicuspid AV.
– **50% of cases of aortic coarctation** are associated with bicuspid AV.
– Patients are also at risk of **aortic root dilatation** and **infective endocarditis**.
– Although rare, **bicuspid PVs** are recognised associations of bicuspid AVs



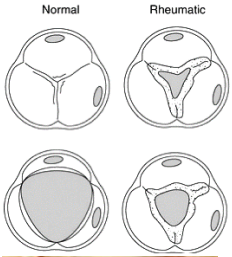
A functionally normal bicuspid aortic valve from a young patient.
A: The long-axis view demonstrates doming of the valve in systole.
B: The basal short-axis view confirms that the valve is bicuspid but with no evidence of stenosis.

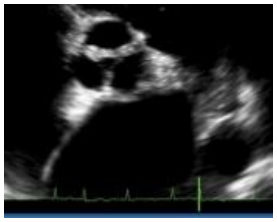
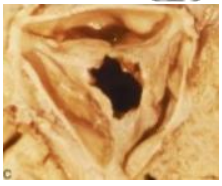


M-mode and two-dimensional (2D) diastolic frame in the parasternal long-axis view of a bicuspid aortic valve with eccentric closure (arrows). Although not diagnostic, an eccentric line of closure should prompt one to evaluate the aortic valve closely for BAV


Marked rib notching (arrows) characteristic of coarctation of aorta in a 30-year-old farmer who was referred for evaluation of a bicuspid aortic valve.

Rheumatic AS is less common than rheumatic MS, and the two often coexist in the same patient. There is commissural fusion , resulting in a triangular systolic orifice, with thickening and calcification most prominent along the edges of the cusps. Sometimes the valve, though being trileaflet, appears functionally bicuspid because of fusion along the commissures.



SUB- AND SUPRAVALVULAR OBSTRUCTION- cause a form of aortic stenosis in which the valve itself is unaffected but the obstruction lies below or above the valve. Subvalvular aortic stenosis results from a fixed obstruction in the LVOT, usually a fibromuscular ridge or membrane, and may be associated with other congenital heart defects in up to half of cases. In supravalvular aortic stenosis there is a fixed obstruction in the ascending aorta, just above the sinuses of Valsalva, due to a diffuse narrowing or a discrete membrane. Subvalvular or supravalvular stenosis is distinguished from valvular stenosis based on:

- Subvalvular membrane is better visualized in apical 5c and 3c rather than PLAX as the U/S beam is perpendicular to the membrane in apical views while parallel to the membrane in PLAX.
- Commonly, patients with subaortic stenosis demonstrate AR as well. This is due to the chronic turbulent flow of the ejected blood in the LVOT that impacts with the aortic cusps
- Colour Doppler is useful to differentiate valvular/subvalvular/supravalvular stenosis. If turbulence is seen proximal to the AV, look carefully for any evidence of LVOT obstruction.
- **PW Doppler** helps differentiate valvular/subvalvular/supravalvular stenosis by localizing the site of increase in velocity as it allows velocities to be measured at a specific point in the LVOT and aorta. Colour flow Doppler also helps differentiate valvular/subvalvular/supravalvular stenosis
- **TOE** will allow accurate visualisation of underlying cause of supravalvular/subvalvular obstruction, particularly in the current era of 3D imaging.

ASSESSMENT OF SEVERITY OF AORTIC STENOSIS

	Normal	Mild	**Moderate**	Severe
Peak velocity (m/s)	**≤1.6**	**1.7-2.9**	**3-4**	
Peak pressure gradient or drop (mmHg)			**36-64**	
Mean pressure gradient or drop (mmHg)			**25-40**	**>40** (>50 for subaortic membrane)
Valve area (cm2)		**1.5-2**	**1.4**-1	**≤ 1**
AVA indexed to BSA (cm^2/m^2)				**≤0.6**
VTI ratio or velocity ratio (dimensionless index) (m/s)		**≥0.5**		**≤0.25**
Systolic separation of the leaflets (M-mode)				**< 12-15** (significant AS)
Aortic valve resistance ($dyne.sec.cm^{-5}$)				**≥ 280**
CW velocity curve	Triangular- peak in early systole			Rounded- peak in mid systole *(HCM → late peaking)*

1. Peak pressure gradient- The CW Doppler trace will give peak transaortic velocity (V_{max}), which relates to peak transaortic pressure gradient (ΔP_{max}) via the simplified Bernoulli equation: ($\Delta P\ max = 4 \times V_{max}^2$).
If peak velocity in the LVOT is >1 m/s, the full Bernoulli equation should be used for greater accuracy: $\Delta P\ max = 4 \times (V_2^2 - V_1^2)$.

- As blood accelerates through the valve, peak velocity coincides temporally with the maximal pressure gradient.
- In general, pressure gradients are affected by flow rate (i.e. affected by SV), such that conditions that increase the flow rate (AR, pregnancy) increase pressure gradient and overestimate the severity of AS, whereas conditions that decrease flow rate (LV dysfunction and MS) decrease the pressure gradient and underestimate the severity of AS.
- The stand-alone CW Doppler is more accurate than the imaging probe for peak aortic velocity measurement as it has a smaller area and allows better alignment with the direction of flow. It can be used in the apical, suprasternal and right parasternal windows. In general, using apical windows only to measure the maximum velocity across the aortic valve will lead to underestimation of velocity in around 30% of cases

Instantaneous vs. peak to peak gradient- Catheter pullback measures peak-to-peak pressure gradient between the LV and the aorta. These pressures do not occur at the same point in time. CW Doppler measures peak instantaneous pressure gradient that is greater than the peak-to-peak gradient. This explains in part why transaortic pressure gradients calculated at catheterisation are lower than those calculated from Doppler.

2. The mean pressure gradient (ΔP mean)- can be obtained by tracing the CW Doppler envelope, from which the echo machine can calculate a mean value by averaging the instantaneous gradients throughout the trace. A simplified approach to calculation of the mean gradient is derived from the empiric observation that there is a close linear correlation between maximum and mean gradients for native aortic valve stenosis.
ΔP mean can be estimated from the (ΔP max) using the equation: **(ΔP mean) = [(ΔP max) ÷ 1.45] + 2 mmHg**
ΔP mean can be estimated directly from the peak velocity as: **ΔP mean = 2.4 (V^2)**

3. Calculating Aortic valve area (AVA) (EOA_{AV}) using the continuity equation ($LVOT_{SV} = AV_{SV}$):
Since (SV = Area x VTI)… then ($Area_{LVOT}$ x VTI_{LVOT} = $Area_{AV}$ x VTI_{AV})
Then **$Area_{AV}$ = $Area_{LVOT}$ x VTI_{LVOT} ÷ VTI_{AV}**
$Area_{AV}$ = [3.14 (½ LVOT diameter)^2] x VTI_{LVOT} ÷ VTI_{AV}

AV area can be approximated using the velocities instead of VTIs in the continuity equation:
$Area_{AV}$ = $Area_{LVOT}$ x V_{LVOT} ÷ V_{AV}. *(the V_{AV} can be estimated from the MG, by the equation: ΔP mean = 2.4 (V^2)*

Valve areas calculated using the continuity equation are not affected by increased flow rate. N.B. low flow rate may inhibit the valve opening, leading to an underestimation of AVA → pseudo severe AS.
LVOT diameter can be measured in the PLAX at the base of the aortic cusps at peak of systole (aortic valve maximally open). It may also be measured in 5CV or 3CV at similar level as LVOT PW Doppler velocity trace obtained, typically 0.5-1.0 cm below AV annulus in calcific AS, and just at AV annulus level in bicuspid AS. The importance of performing this measurement accurately cannot be overemphasized. Because the radius is squared to determine area, small errors in measuring this linear dimension will be compounded in the final formula. Therefore, LVOT diameter measurement should be made in optimised windows i.e. zoomed, for a minimum of 3 beats (5 in AF). AVA calculated using the continuity equation is not affected by increased flow rate, but continuity equation cannot be used if the LVOT is not circular, as in HOCM or subvalvular stenosis, or if there are serial stenoses, i.e. sub/supravalvular stenoses.

Indexed AVA- current guidelines recommend indexing AVA to body surface area (BSA); a value of 0.6 cm^2/m^2 has been used as a cut-off for defining A.S as severe. It is noteworthy that indexing on BSA overestimates the severity of valve stenosis in obese patients, because valve area does not increase with excess body weight.

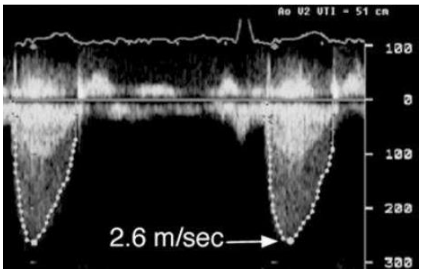
Direct planimetry of the AV- is difficult to reproduce accurately because of the complex nature of its tricuspid appearance, thus Doppler provides the best functional assessment of valve area. However, planimetry is an acceptable alternative when Doppler estimation is unreliable (e.g. coexisting LVOT obstruction)

4. VTI ratio or velocity ratio (dimensionless index)- is a simple and useful alternative for evaluating stenosis. It is calculated as the ratio between the outflow tract velocity and the transvalvular velocity. In the absence of any gradient, the two velocities will be the same, yielding a ratio of one. A ratio < 0.5 and > 0.25 suggests moderate AS in native aortic valves. Because all prostheses are somewhat stenotic, the expected range for normally functioning aortic prostheses is 0.35 to 0.5. Although a useful additional measure, by removing the potential inaccuracies of LVOT measurement, remember that it ignores inaccuracies due to abnormal LVOT anatomy e.g. isolated basal hypertrophy. Hence, its particular use is in the setting of serial measurements within the same individual or when assessing prosthetic valves, especially where the size of the valve is unknown.

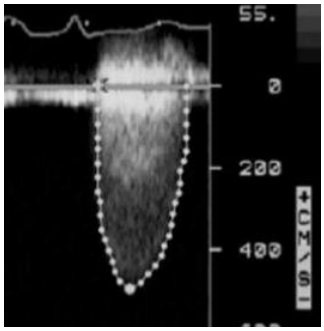
5. Systolic separation of the leaflets (M-mode)- Leaflet separation of <12-15 mm is suggestive of significant AS. Although this method is not very accurate in determining the severity of the stenosis, and leaflet opening of <12-15 mm does not distinguish between mild, moderate or severe stenosis, opening of >15 mm reliably excludes severe stenosis.

6. Aortic valve resistance = (mean pressure gradient) ÷ (flow per ejection period) x 1.33 (where 1.33 is conversion factor from mmHg to dyne.sec.cm^{-5})
Aortic valve resistance = 1.33 × [MG ÷ (SV ÷ ET)]
Aortic valve resistance = 1.33 × (MG x ET) ÷ SV
The cut-off for severe AS is ≥ 280 dyne.sec.cm^{-5}

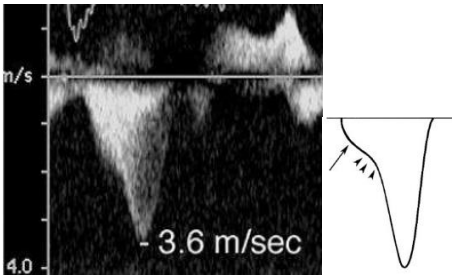
7. CW velocity curve in assessment of severity of aortic stenosis and differentiation from HCM



Mild AS: triangular curve with the peak in early systole



Severe AS: rounded curve with the peak moving towards mid-systole



Dynamic subaortic obstruction (HOCM): late peaking curve, usually with upward concavity in early systole

8. LVH- is commonly seen as the LV adapts to overcome the obstructive valve. Aortic stenosis increases the afterload (the "load" that the heart must eject blood against). The afterload of the LV is closely related to the aortic pressure and is related to ventricular wall stress by a modification of the LaPlace Law and is, therefore, directly proportionate to the pressure and radius, and inversely proportionate to the wall thickness. To avoid ↑ wall stress, the LV undergoes compensatory concentric hypertrophy (increased thickness), which allows the wall stress to normalize. But when the LV failure occurs eventually, the radius increases leading to increased wall stress.

9. 3D ECHO

- 3D echo provides useful information regarding the AV and annulus morphology
- 3D echocardiography is highly recommended over the 2D echo for direct AV planimetry and for determining the LVOT area that can be used for calculation of the AVA by the continuity equation: AVA = $Area_{LVOT} \times VTI_{LVOT} \div VTI_{AV}$
- 3D LV volumes estimation (using semi-automated LV border detection) can be used to calculate stroke volume that can be used at the numerator of the continuity equation for calculation of the AVA = $SV_{LVOT} \div VTI_{AV}$. This may be more accurate than stroke volume derived from measurements of the (LVOT diameter) and (VTI by PW Doppler). 3D colour Doppler can also overcome inaccuracies of spectral Doppler for stroke-volume calculation.

STRESS TESTING IN AORTIC STENOSIS

1. **Stress testing for asymptomatic severe AS**- In patients with aortic stenosis (AS), the onset of symptoms and/or LV systolic dysfunction represents a clear indication for AVR. Exercise testing is contraindicated in patients with severe AS with definite or probable cardiac symptoms. However, exercise testing is recommended to unmask symptoms or abnormal blood pressure responses in AS patients without apparent symptoms. Exercise testing, with appropriate physician supervision and close monitoring of the ECG and blood pressure, is safe in AS patients without apparent symptoms. Approximately one-third of patients exhibit exercise-limiting symptoms; these patients have worse outcomes. In patients with asymptomatic severe AS exercise stress echo has been shown to provide incremental prognostic value beyond exercise testing alone. (1) An increase in mean aortic pressure gradient by ≥18–20 mmHg, (2) the absence or limitation of LV contractile reserve (decrease or no change in LVEF suggesting subclinical LV dysfunction) and (3) induced PH (SPAP . 60 mmHg) during exercise are markers of poor prognosis.
2. **Stress testing for low gradient AS**
   - **Mean gradient is flow-dependent** (a squared function of flow) such that low flow rate may underestimate the MG → underestimate severity of AS. In LV dysfunction, there is a low-flow rate, resulting in a lower gradient and underestimation of severity of AS. Infusion of dobutamine augments cardiac output and if the valve is truly severely stenosed, the MG will increase due to increased flow rate.
   - **Valve opening is also flow dependent** such that low flow rate may inhibit the valve opening, leading to a lower AVA → overestimation of severity of AS (pseudo severe AS).
   - **Low gradient AS** is defined as MG <40 mmHg & AVA ≤1 cm^2
   - **Low flow** is defined as a **SVi** <35 mL/m^2 and is present in up to 35% of patients with AS *(SVi represents the flow)*
   - **LF-LG** is devided into (1) classical LF-LG (EF<50%) and (2) paradoxical LF-LG (EF≥50, but SVi <35).
   - If SVi >35 → normal flow, low gradient AS.
   - **Classical LF-LG** is divided into (1) true severe AS (MG↑ in response to low dose DSE) and (2) pseudo severe AS (AVA↑ in response to low dose DSE)

Stepwise management of low gradient AS (MG <40 mmHg & AVA ≤1 cm^2):

Step 1: Echo derived LVEF
A. EF < 50% → Classical LF-LG → low dose DSE (→ step 3 to differentiate true from pseudo severe AS)
B. EF ≥ 50% → Calculate SVi (→ step 2 to differentiate paradoxical LF-LG from normal flow-LG)

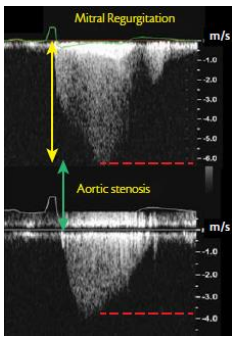
Step 2: Echo derived SVi ($Area_{LVOT}$ x VTI_{LVOT}) /BSA
A. SVi ≤ 35 ml/m^2 *(low SV paradoxical to normal EF)* → Paradoxical LF-LG
B. SVi > 35 ml/m^2 *(normal SVi = normal flow)* → Normal flow-LG → (1) rule-out measurement errors, (2) assess symptomatic status, (3) check for presence of hypertenison (may lead to a substantial decrease in gradient) and then (4) confirm stenosis severity by MDCT AV calcium scoring and/or DSE (step 3)

Step 3: low dose DSE (5-20 mcg/kg/min)
A. MG↑ ≥ 40 mmHg → True severe AS
B. AVA↑ > 1 cm^2→ Pseudo severe AS
C. No change → Check projected AVA & AV Ca score → If AVA_P < 1 cm^2 and/or AV_{Ca} > 2000 in men/1200 in women → True severe AS. If doubt remains about the diagnosis a TOE could be considered

Paradoxical LF-LG AS is defined as AVA < 1 cm2 (< 0.6 cm2/m2) + LV ejection fraction (EF > 50%)
+ Mean Ao pressure gradient < 40 mm Hg
+ SV index < 35 mL/m2
+ Severely thickened/calcified valve

Additional echo features in favour of paradoxical AS
- End-diastolic diameter < 47 mm
- End-diastolic volume index < 55 mL/m2
- Relative wall thickness (RWT) ratio > 0.50
- Valvulo-arterial impedance (Zva) > 4.5 mmHg/ml/m2
- Impaired LV filling
- Global longitudinal strain (GLS) < 16%

PITFALLS IN THE ECHO ASSESSMENT OF AS



- **Preferred method: continuity equation is preferred method** for AVA calculation. Continuity equation is not affected by increased flow rate (valid in presence of associated AR or MR- whereas cannot be used to assess MVA in presence of AR or MR), but continuity equation cannot be used if the LVOT is not circular, as in HOCM or subvalvular stenosis, or if there are serial stenoses, i.e. sub/supravalvular stenoses.
- The AV Doppler flow should be differentiated from associated MR Doppler flow in the apical 5C view. Associated MR Doppler flow starts within the QRS whereas the AV Doppler flow starts after the QRS. Also, an associated MR Doppler flow usually has a greater maximum velocity than the AV Doppler flow (AS Doppler has later onset an lower velocity). The terminology "pre-ejection time" fits better for the time duration between the onset of MR and the onset of ejection through the AV rather than the term IVCT as, in cases with MR, the use of terms such as IVCT and IVRT is inappropriate, since during these time periods blood is constantly ejected back to the left atrium lowering the left ventricular blood volume.
- In general, pressure gradients are affected by flow rate/SV, such that conditions that increase the flow rate/SV (associated AR, pregnancy) increase pressure gradient and overestimate the severity of AS, whereas conditions that decrease flow rate (LV dysfunction and associated MS) decrease the pressure gradient and underestimate the severity of AS.
- Low flow low gradient AS is not infrequent finding in presence of associated MR or MS.
- Simplified Bernoulli equation and Gorlin formula using thermodilution may be invalid for AS in case of associated AR. In cases of AS with associated severe AR, proximal velocity is frequently > 1 m/s and cannot be ignored for transaortic pressure gradient determination. The following formula should be used: pressure gradient = $(V_2^2 - V_1^2)$, where V2 = transvalvular velocities obtained with CW Doppler and V1 = LVOT velocities obtained with PW Doppler
- Maximal anterograde transaortic velocity reflects both AS and AR severity in patients with moderate or severe AS and moderate or severe AR and preserved LV function
- Indexing on BSA overestimates the severity of valve stenosis in obese patients, because valve area does not increase with excess body weight.

ECHO SURVEILLANCE

- Patients with aortic stenosis should be advised to report symptoms immediately.
- Asymptomatic patients with an aortic V max of >4 m/s should be reassessed every 6 months, and if V max increases by >0.3 m/s per year, surgery should be considered.
- Annual reassessment is advised for those with lesser degrees of stenosis.
- **ACC**: annual echo for moderate-severe valvular stenosis/regurgitation and every three years for mild valvular stenosis/regurgitation

AORTIC REGURGITATION

CAUSES OF AORTIC REGURGITATION

1) **Valvular causes include:**
   - Bicuspid aortic valve, causing incomplete closure of the valve, common cause in Western Countries
   - Calcific degeneration of the aortic valve
   - Rheumatic aortic valve disease (calcified valves suggest primary calcific disease or previous rheumatic disease), uncommon cause in Western Countries
   - Infective endocarditis (→ leaflet perforation and malcoaptation due to vegetations)
   - Connective tissue diseases (e.g. rheumatoid arthritis, SLE).
   - Myxomatous (→ thickened, redundant leaflets, which may sag in diastole, distorting the normal crown shape such that a leaflet is seen fully face on, giving the erroneous appearance of an ill-defined mass in the PSAX)
2) **Aortic root causes-** result from dilatation and/or distortion of the aortic root, not rare in Western countries. These include:
   - Hypertension (aortic root dilatation is commonly caused by hypertension; Marfan's syndrome and rheumatoid arthritis are less likely causes)
   - Marfan's syndrome (characterized by effacement of the sinotubular junction)
   - Ehlers–Danlos syndrome: the conformation of the aortic root sinuses can be lost in Ehlers–Danlos syndrome
   - Osteogenesis imperfecta
   - Aortic dissection (type A)
   - Sinus of Valsalva aneurysm
   - Cystic medial necrosis
   - Syphilitic aortitis
   - Behçet disease.
   - Some conditions, such as ankylosing spondylitis, can affect both the aortic valve and the aortic root.
3) **Subpulmonary VSD** is commonly associated with AR due to prolapse of the right coronary cusp of the AV

PATHOPHYSIOLOGY AND HAEMODYNAMICS

Acute AR is a medical emergency. Acute severe AR → no time for LV compliance to adapt (normal LV dimensions, often with vigorous function) → LVEDP ↑ rapidly, occasionally as high as aortic end-diastolic pressures (leading to rapid equalisation of trans-aortic pressures and short pressure half time proportionate to severity of AR) → pulmonary oedema, cardiogenic shock and death usually develop quickly. Other relevant pointers of acute onset include evidence of dissection, endocarditis and trauma.

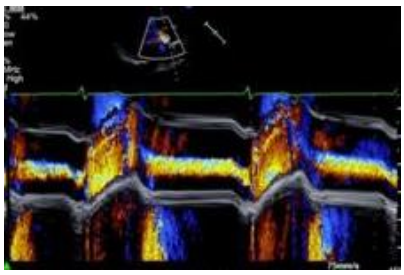
Chronic AR allows the LV to adapt and remodel (dilated LV with eccentric hypertrophy and systolic function may become impaired) → unimpeded regurgitation back into the LV → aortic pressures ↓ very low in diastole (leading to delayed equalisation of trans-aortic pressures and misleadingly longer pressure half time) → Pulse pressure widening due to ↓DBP (and ↑SBP due to increased stroke volume). During initial stages, the regurgitant volume increases LVEDV without an increase in LVEDP, but eventually LVEDP↑ as systolic dysfunction supervenes and heralds the onset of symptoms. **Remember:** Concentric hypertrophy is usually founded in patients with arterial hypertension and AS (pressure loaded conditions). Eccentric hypertrophy is usually founded in patients with aortic or mitral regurgitation (volume loaded conditions). Asymmetric and apical hypertrophy are 2 typical types of hypertrophy in patients with hypertrophic cardiomyopathy.

AR peak gradient is not an indicator of severity of AR, but can be used to estimate the LVEDP: LVEDP = DBP – AR gradient

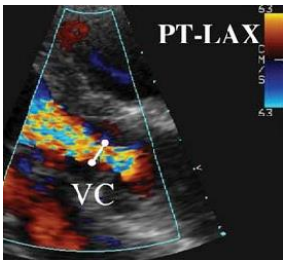
ECHO ASSESSMENT OF AR

	Mild	Moderate	Severe
Vena contracta width (cm)	<0.3	**0.3 - 0.6** *(MR **0.3** - 0.7)*	>0.6
Regurgitant orifice area (cm^2)		**0.1** - 0.3 *(MR **0.2** - 0.4)*	≥0.3
Regurgitant volume (ml/beat)		**30** - 60 *(AR/MR)*	>60
Regurgitant fraction (%)		**30** - 50 *(AR/MR)*	>50
Jet width/LVOT diam. ratio (%)		**25** - 65 *(MR **20 – 40**/LA)*	≥ 65
PHT (ms)	>500	**500 – 200** *(MS **140**-220)*	<200 *(MS>220)*
VTI of diastolic flow reversal (upper descending aorta) (cm)			>15
CW Doppler trace density	Faint	Dense	Dense

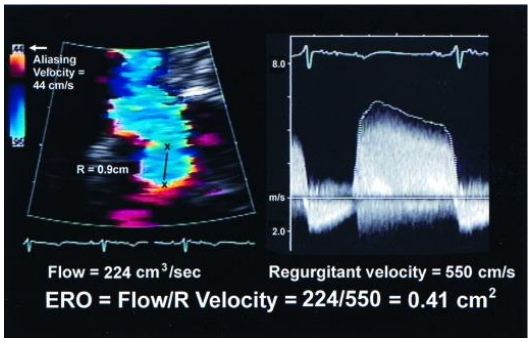
1. **Jet width / LVOT height-** The regurgitant jet width to LVOT diameter ratio refers to the maximal proximal jet width measured in the LVOT. Similar to VC width measurements, use PLAX with zoom & colour M-mode to maximise axial and temporal resolution. Colour M-mode imaging in the PLAX, with the cursor placed just below the AV, can be a useful way to measure the width of the jet and of the LVOT, and can minimise measurement errors .



2. **Vena Contracta-** VC width is the narrowest portion of colour flow at or just below the level of the AV. Measure in parasternal windows (better axial resolution compared to apical windows). It's advisable to use zoom & colour M-mode to minimise errors in measurement. VC width is a more reliable measure of regurgitant severity than jet width/LVOT, particularly if the regurgitant jet is central. VC is, however, valid for eccentric jets, if measurements are made perpendicular to the direction of the jet rather than to the long axis of the LVOT. VC width is not reliable if there are multiple jets or the jet is irregularly shaped.

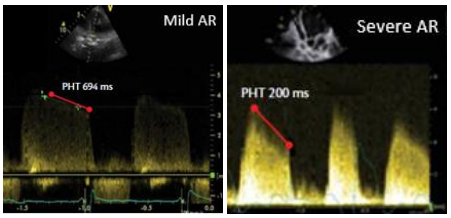


3. **PISA or flow convergence (& EROA)**
   1) Apical 5C for central jets (PLAX for eccentric jets)
   2) Optimize colour flow imaging of AR
   3) Zoom of the selected zone
   4) Increase the Nyquist limit in apical views (decrease or increase in PLAX) to obtain hemispheric PISA
   5) With the cine-mode select the best PISA
   6) Display the colour off and on to visualize the AR orifice
   7) Measure of the PISA radius at diastole using the first aliasing and along the direction of the ultrasound beam
   8) Measure AR peak velocity and VTI (CW)
   9) Calculate flow rate, EROA, regurgitant Vol and regurgitant fraction
      PISArea = 2 x 3.14 x $(PISA\ radius)^2$
      EROA = (PISArea X aliasing velocity) ÷ AR peak velocity
      Regurgitant volume = EROA x VTI (similar to SV equation)
      Regurgitant fraction = Regurgitant volume ÷ $LVOT_{SV}$ x100



PISA, in theory, can be applied to any regurgitant valve to measure regurgitant area and volume. However, because of the technical challenges of visualizing the isovelocity shells that converge on the aortic regurgitant orifice, this technique has limited application to the aortic valve. Limitations also include suboptimal images in the presence of AV calcification and underestimation in aortic aneurysms. Moreover, Validity is questionable for multiple/eccentric jets.

4. **Aortic Regurgitant Volume**
   - In AR, the regurgitant volume will be added to the stroke volume at the LVOT.
     Therefore the regurgitation volume = SV_{LVOT} - SV_{MV} (in the absence of significant MR or a VSD)
     = ($Area_{LVOT}$ x VTI_{LVOT}) – ($Area_{MV}$ x VTI_{MV})
     To obtain the $Area_{LVOT}$, Measure LVOT diameter in PLAX
     Measure VTI_{LVOT} using PW Doppler in Apical 5C
     To obtain the $Area_{MV}$, Measure MV annulus diameter in Apical 4C
     Measure VTI_{MV} using PW Doppler in Apical 4C at mitral annulus level
   - Regurgitant volumes in severe AR can be calculated using PW Doppler at a single site in the proximal descending aorta, where the forward flow and stroke volume can be calculated, as well as the retrograde regurgitant jet. VTI of the antegrade flow is multiplied by the systolic aortic area, whereas VTI of the diastolic flow reversal is multiplied by the diastolic aortic area. Either 2-D or M-mode imaging of the aortic arch can be used to determine both systolic and diastolic areas.
   - It is possible to calculate aortic regurgitant fraction from the ratio of reversed to forward flow in the aortic arch using M-mode measurements to account for the systolic and diastolic changes in aortic diameter.
   - Regurgitant Fraction (%) = (Regurgitant Volume ÷ $LVOT_{SV}$) x 100 = [(SV_{LVOT} - SV_{MV}) ÷ SV_{LVOT}] x 100
   - Regurgitant Orifice Area (cm^2) = Regurgitant Volume ÷ VTI of AR Doppler trace
     Measure VTI of the AR Doppler trace using CW Doppler in Apical 3- or 5-chamber views



5. **Pressure half-time of the diastolic deceleration slope**- record CW in 5C or 3C aided by colour flow mapping to align the cursor along the direction of the AR jet as it originates from the regurgitant orifice. Measure peak velocity (can be used to estimate LVDP = DBP - AR gradient) and the deceleration slope of the flat part of the spectral trace. The machine will automatically calculate the pressure half-time i.e. time taken for pressure across the aortic valve to fall by half. The slope of the AR jet is steeper and the PHT is shorter in severer AR, particularly in acute AR (see pathophysiology above). Sepsis and Calcium channel blockers may affect pressure half-time values.
6. **CW Doppler trace density-** is faint in mild AR, and denser in moderate or severe regurgitation. A very dense signal jet equal to density of forward flow signal through the valve is in keeping with a severe jet of AR, although cannot reliably distinguish from moderate jet. CW Doppler trace of AR jet can be recorded in the suprasternal window although the AV is not seen in the suprasternal view and, therefore, suprasternal widow is mainly used for PW interrogation of the upper descending aorta to look for diastolic flow reversal (see below).
7. **Diastolic flow reversal in the upper descending aorta**, using PW Doppler in the suprasternal view and placing the sample volume in the descending aorta at the level of aortic isthmus (just beyond the origin of the left subclavian artery). It is normal to have a brief reversal of aortic flow in diastole, but flow reversal throughout the whole of diastole (pan-diastolic) indicates severe AR, although can occasionally be seen in moderate AR. End-diastolic velocity of > 20 cm/sec is a common finding in patients with severe AR. Obtain a VTI of the diastolic flow reversal – severe regurgitation is indicated by a VTI >15 cm. Pan-diastolic flow reversal may also be seen in the abdominal aorta, where it is a specific indicator of severe AR.
8. **PW Doppler can be used to map the extent of the regurgitant jet in the LV**, by positioning the sample volume at various points in the LV (in the apical 5-C) and checking for regurgitant flow, although this is not a good indicator of severity.

9. M-mode & 2-D findings in AR

a. LV dilatation and a characteristic change to a more spherical shape (in response to volume overload over an extended period, in chronic AR). **LV mass increases**, although the increase in wall thickness is modest and LV systolic function is preserved. The enlarging left ventricle remains compliant and is able to accept the simultaneous filling through the mitral and aortic valves throughout diastole without a significant increase in pressure. Eventually, LV function begins to deteriorate, although this generally does not occur until a significant increase in end-systolic volume is present and should be viewed as a late and sometimes irreversible change in the natural history of the disease. LVESD > 50 mm or LVEDD >70 mm is an indication for surgery in severe AR. A normal LV size virtually excludes significant chronic regurgitation. **Remember:** LVESD > 45 mm, LV dysfunction (<60%), AF or pulmonary hypertension (>50mmHg) are indications of surgery in severe, asymptomatic

b. Abnormal MV motion *(when the AV leaks, the MV flutters and closes early)***:**

- **Diastolic fluttering of the AML-** As the aortic jet cascades across the AML, it creates a high-frequency fluttering that requires the rapid sampling rate of M-mode echocardiography for detection.
- **Premature closure of the MV**- due to rapidly increasing left ventricular diastolic pressure, particularly in acute regurgitation. Premature closure of the MV → functional MS (Austin Flint murmur). (*While HCM → SAM → functional MR)*
- **Diastolic "reverse doming" of the AML** (concavity toward the IVS) can be seen in AR if the regurgitant jet impinges on the AML

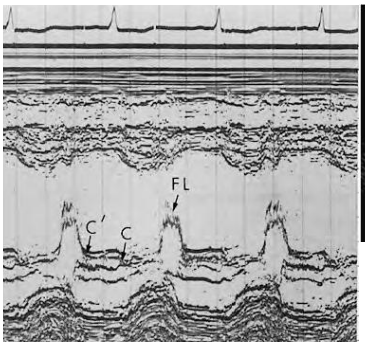


Fig. An M-mode recording from a patient with acute and severe AR demonstrates both fluttering (FL) of the AML and premature closure (C′) of the MV, the result of rapidly increasing diastolic LV pressure.

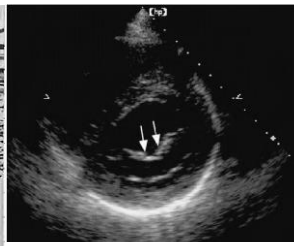


Fig. abnormal mitral valve motion due to impingement on the anterior leaflet by a posteriorly directed aortic regurgitation jet. Note how the mid-portion of the leaflet is deformed during diastole (arrows).

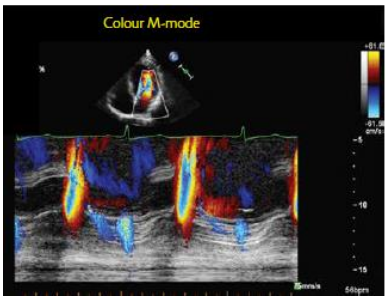
c. Hyperdynamic IVS motion (compared with posterior wall) occurs as a result of left ventricular volume overload due to unequal filling and stroke volume of the ventricles. This is best appreciated with M-mode imaging, which often reveals an exaggeration of the normal early diastolic septal dip and an overall increase in the amplitude of septal motion compared with the posterior left ventricular wall.

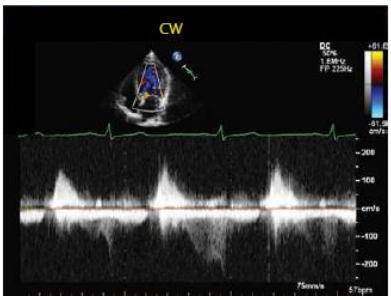
d. Increased E-point to septal separation (EPSS)- the normal EPSS is 6 mm, with progressively larger EPPS representing a lower ejection fraction. **But**, in patients with AR, increased EPSS is seen because of the restriction in opening of the anterior MV leaflet due to the AR jet, and has no consistent relation to the contractile function.

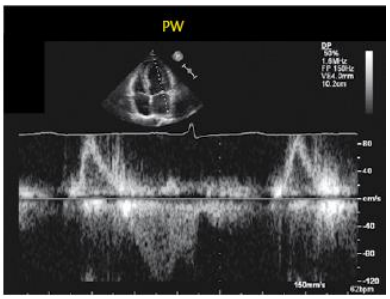
e. Measurement of aortic root diameter is essential as aortic root dilatation is not uncommon with AR

PITFALLS IN THE ECHO ASSESSMENT OF AR

- **Preferred method:** for AR assessment, consider multi-parametric evaluation including PISA method if feasible, vena contracta, demonstration of holodiastolic flow reversal in the descending aorta and of a dense CW retrograde Doppler signal across the AV.
- **Pressure half-time** method unreliable in presence of:
  - Associated AS (prolonged in the presence of LVH with impaired relaxation, or shortened if there is AS-induced elevation in LVDP)
  - Associated MR
- **Doppler volumetric method** (using Doppler mitral inflow and LVOT stroke volume) is inapplicable in presence of associated MR
- Associated MS may blunt the hyperdynamic clinical picture
- In acute AR, the presence of diastolic MR (a marker of premature mitral valve closure) should be assessed







ECHO SURVEILLANCE

- Patients with mild–moderate AR or moderate MR should be seen annually and have an echo every 2 years.
- Asymptomatic patients with severe AR and normal LV function should be reviewed every 6 months, or annually if stable and not close to needing surgery (Asymptomatic patients with severe MR and normal LV function) should be seen every 6 months and have an echo every year)
- **ACC**: annual echo for moderate-severe valvular stenosis/regurgitation and every three years for mild valvular stenosis/regurgitation

HINTS:

- **Severe AR** is a contraindication to an intra-aortic balloon pump and is likely to overload the LV further if inserted.
- Acute symptomatic AR warrants prompt surgical intervention
- Beta blockers may play a role in management of AR
- Systolic hypertension is often seen in AR, and diastolic BP is typically low.
- A flail aortic valve denotes severe aortic regurgitation