Spectrum of Aortic Stenosis:
Etiology and Echo Quantification

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Review Question #1

- What can lead to underestimation of the aortic valve gradient on echo as compared with invasive hemodynamics at cath:

A. Pressure Recovery

B. Equating peak instantaneous gradient to "peak-to-peak" gradient

C. A large incident angle to the aortic outflow

D. Failure to account for high subvalvular flow

E. Low stroke volume

Reflect upon the image below Transesophageal (TEE):

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LVOT diameter = 2.0 cm
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The aortic valve area is most likely:

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C. Moderately reduced
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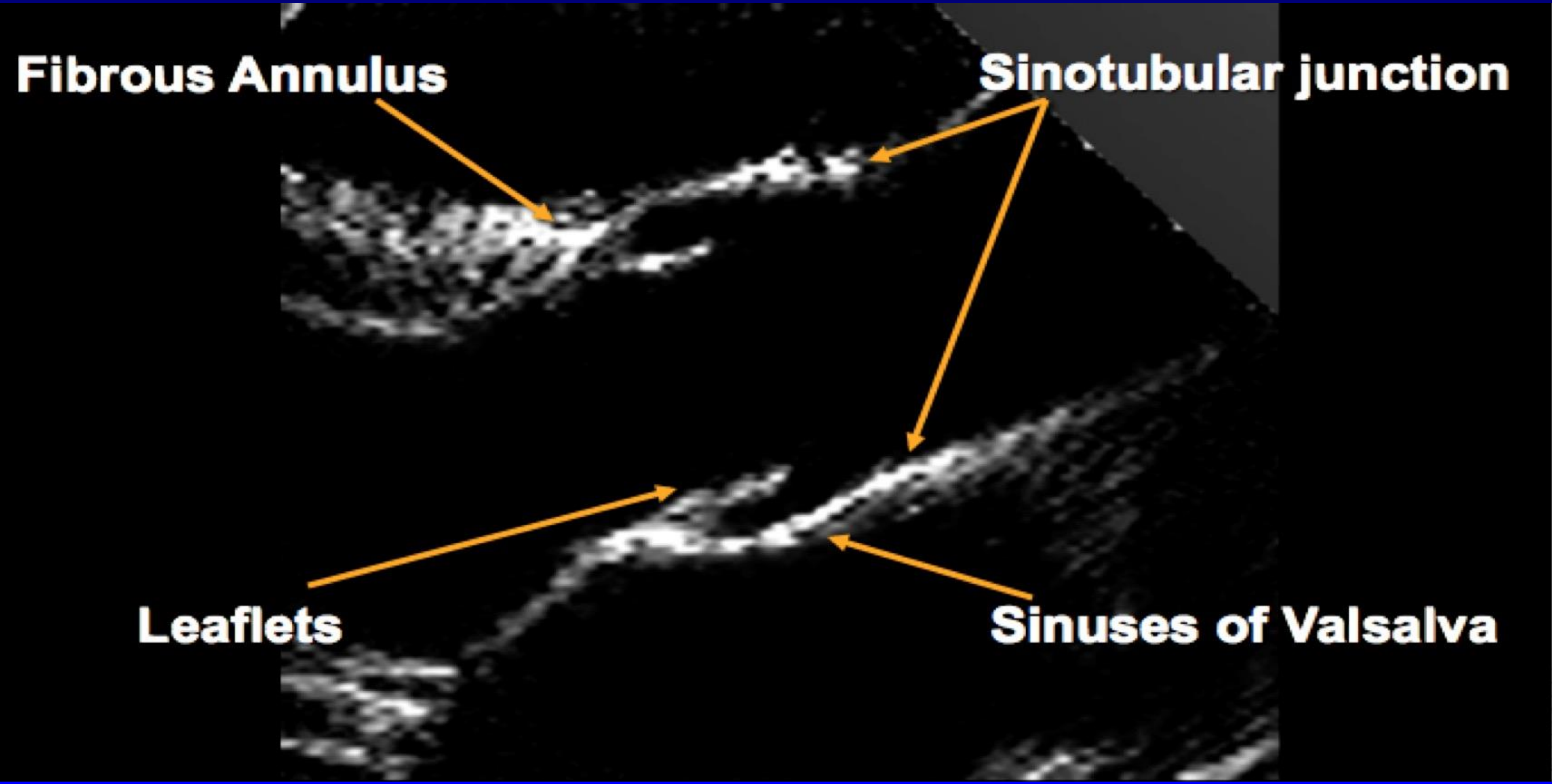
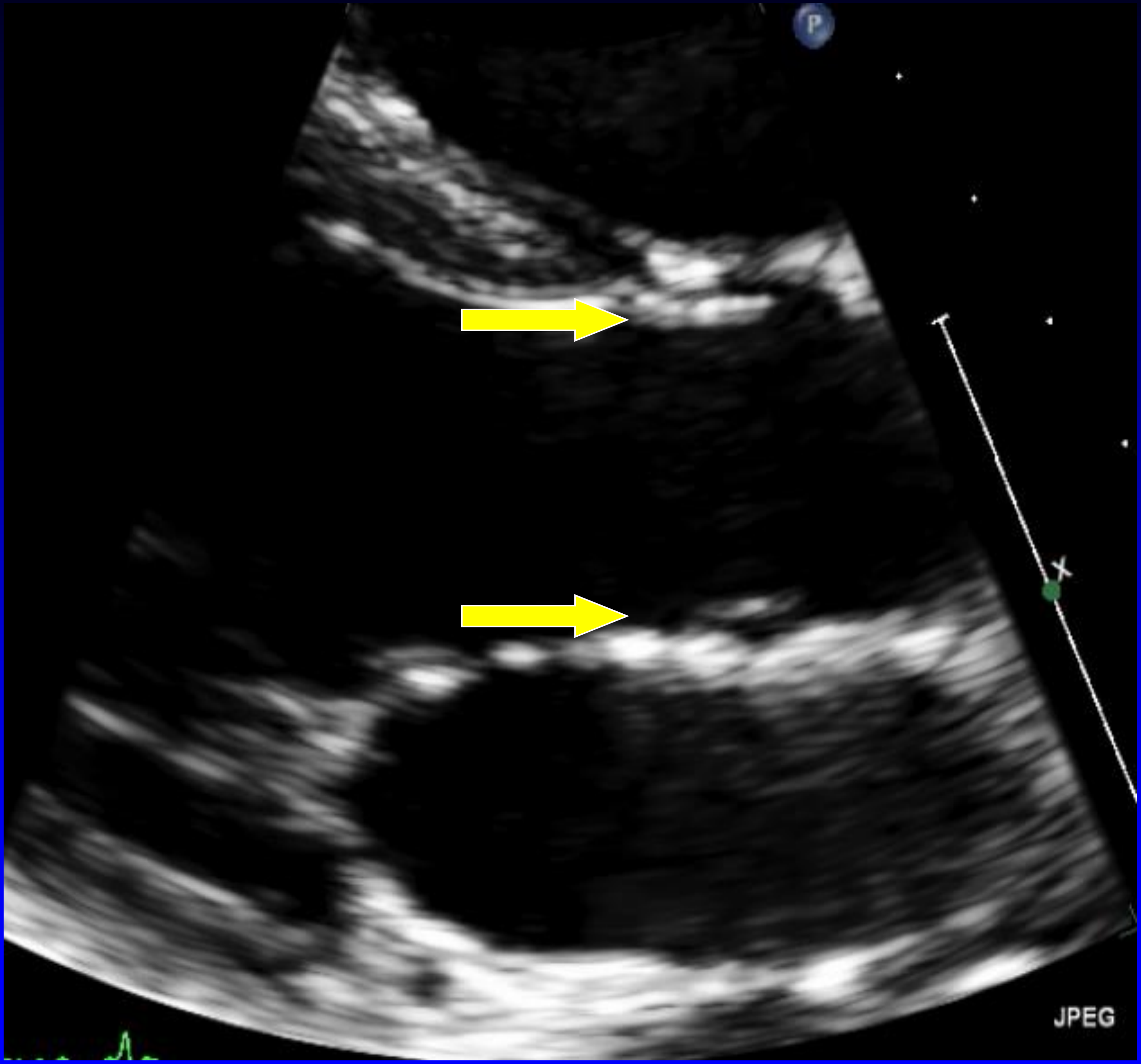
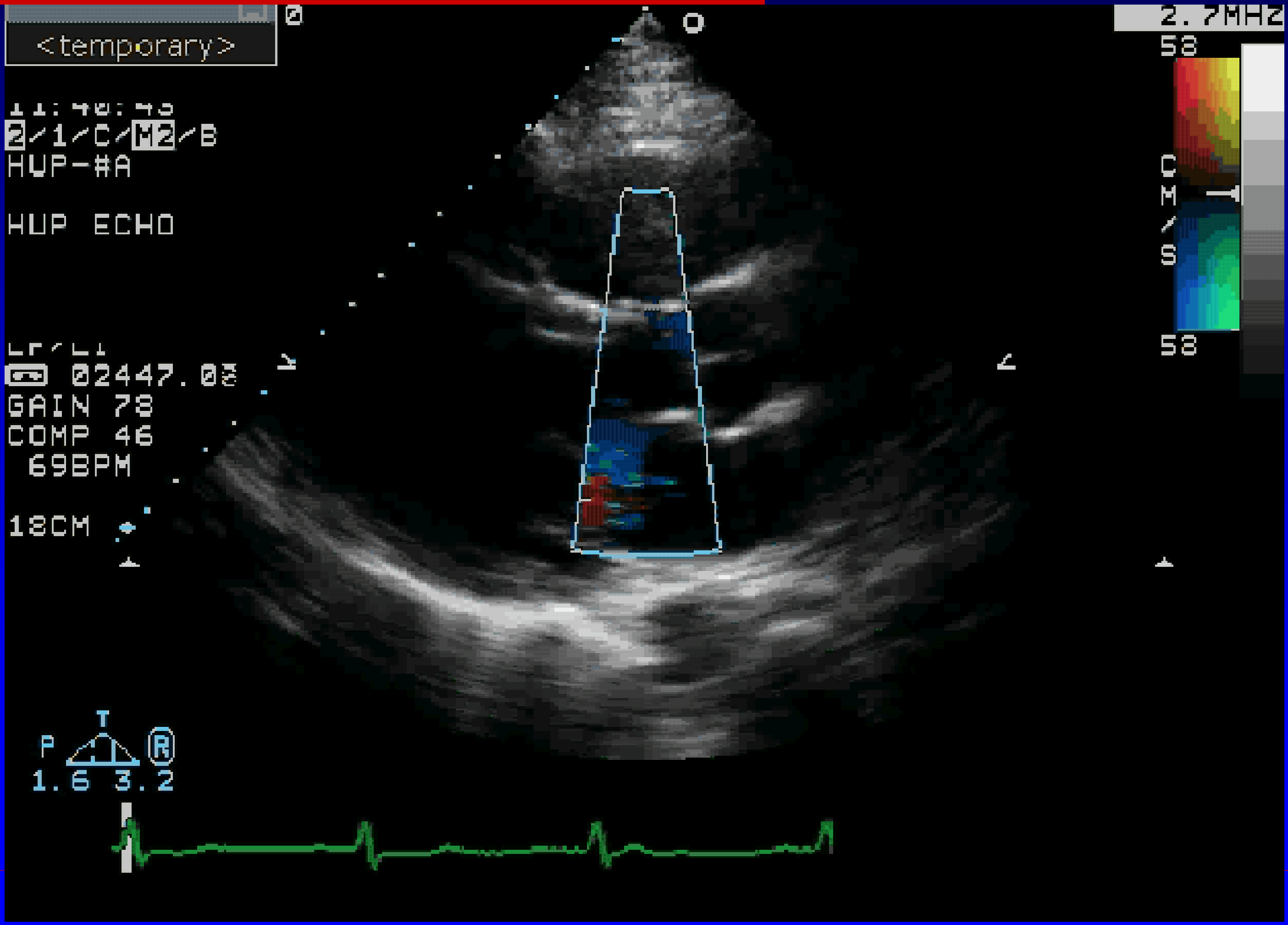
Basic root structure
Parasternal Long Axis View

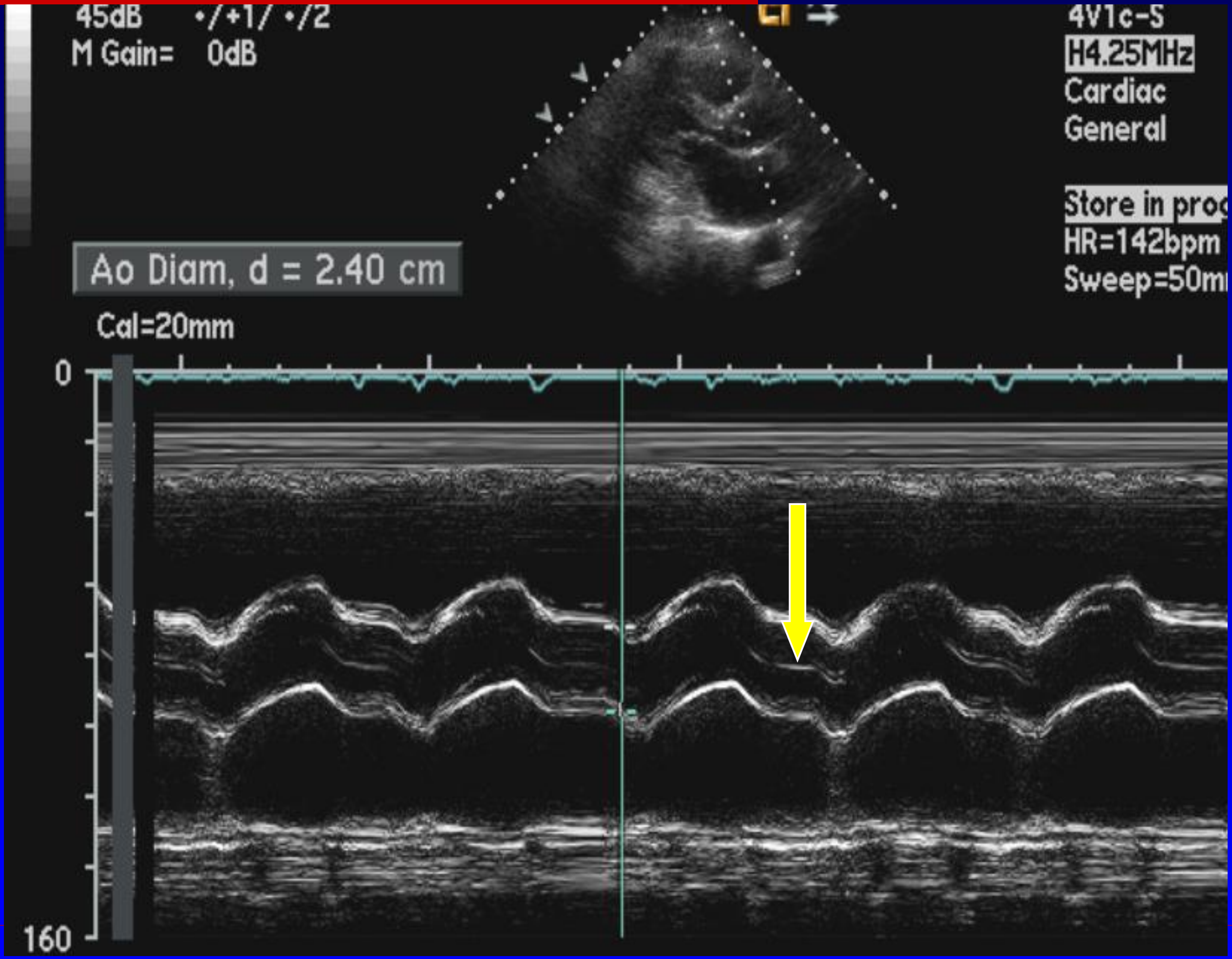



JPEG

Normal AV
parasternal color Doppler

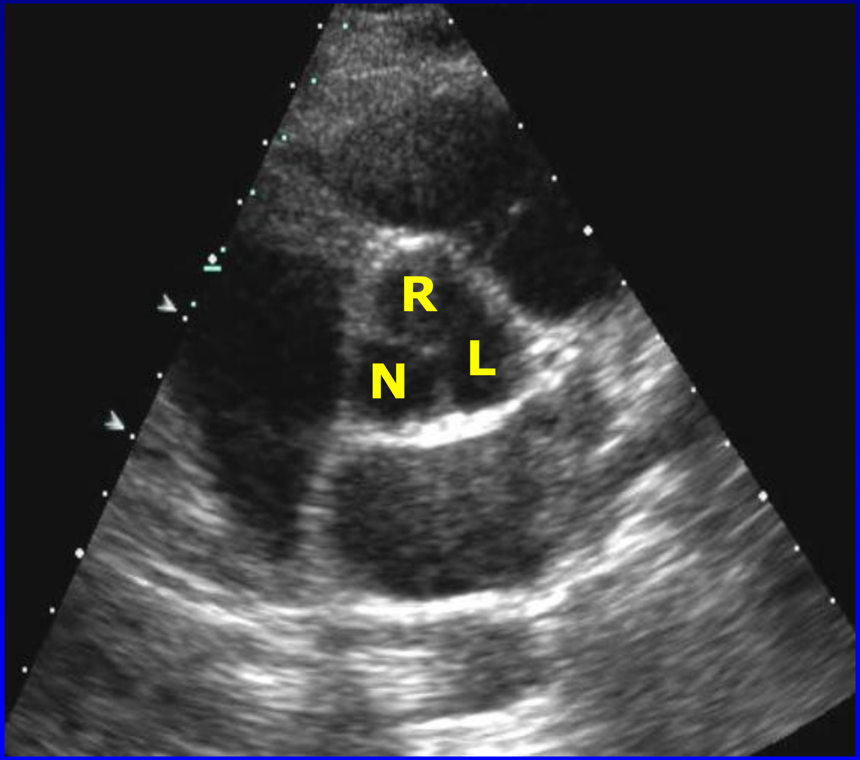


Normal AV M-Mode
coaptation in center of aortic root

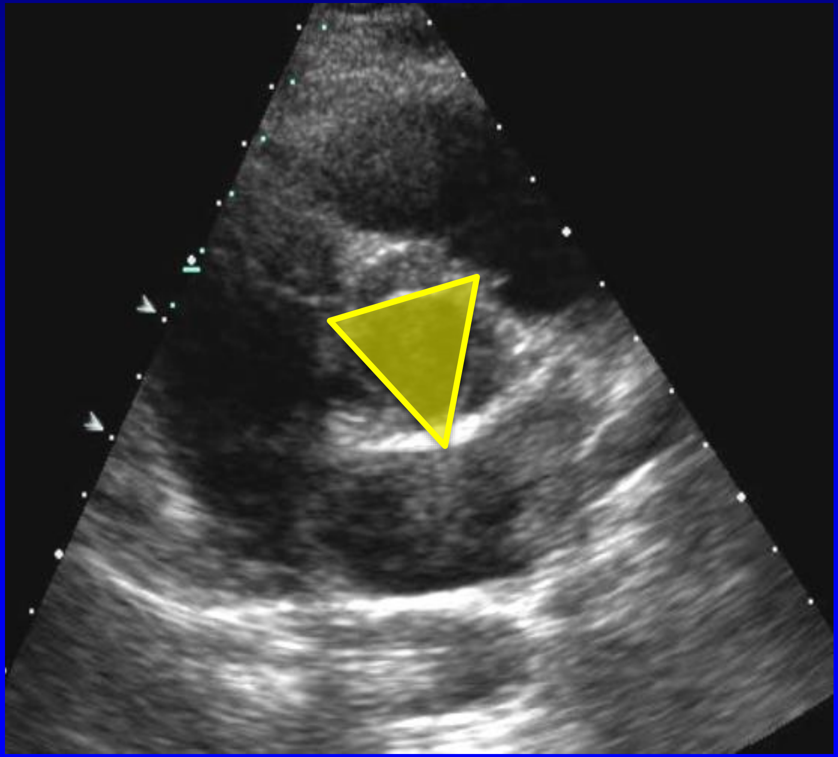


Normal AV
orientation and opening

Diastole

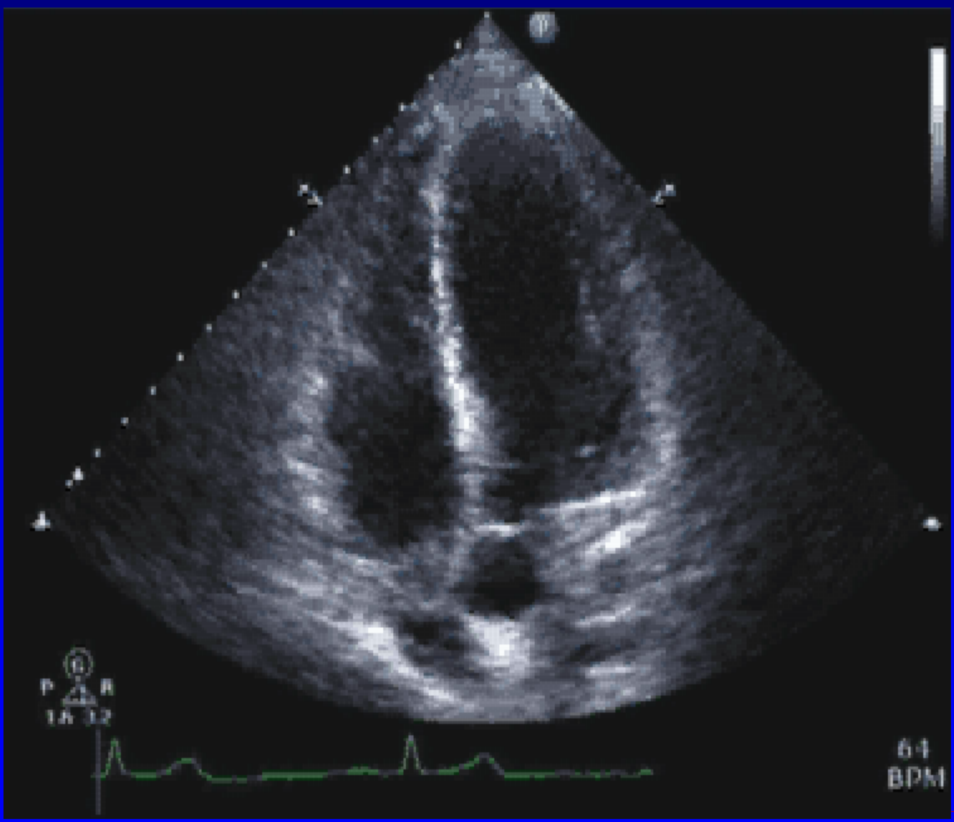


Systole

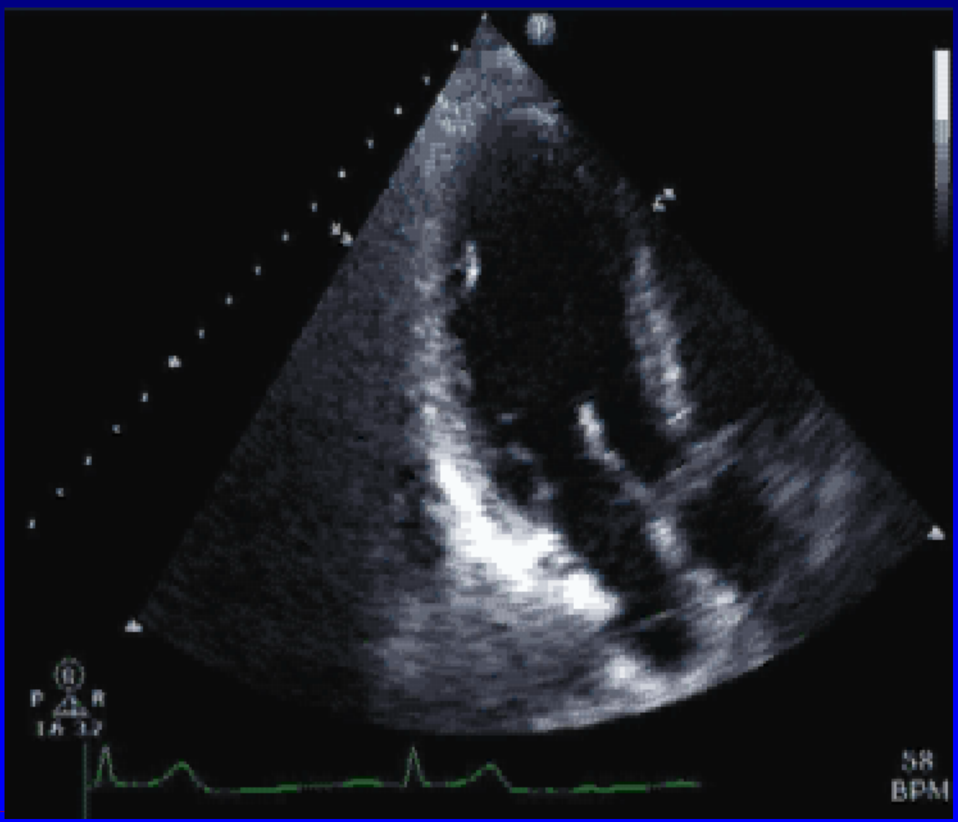


Normal AV
Apical views

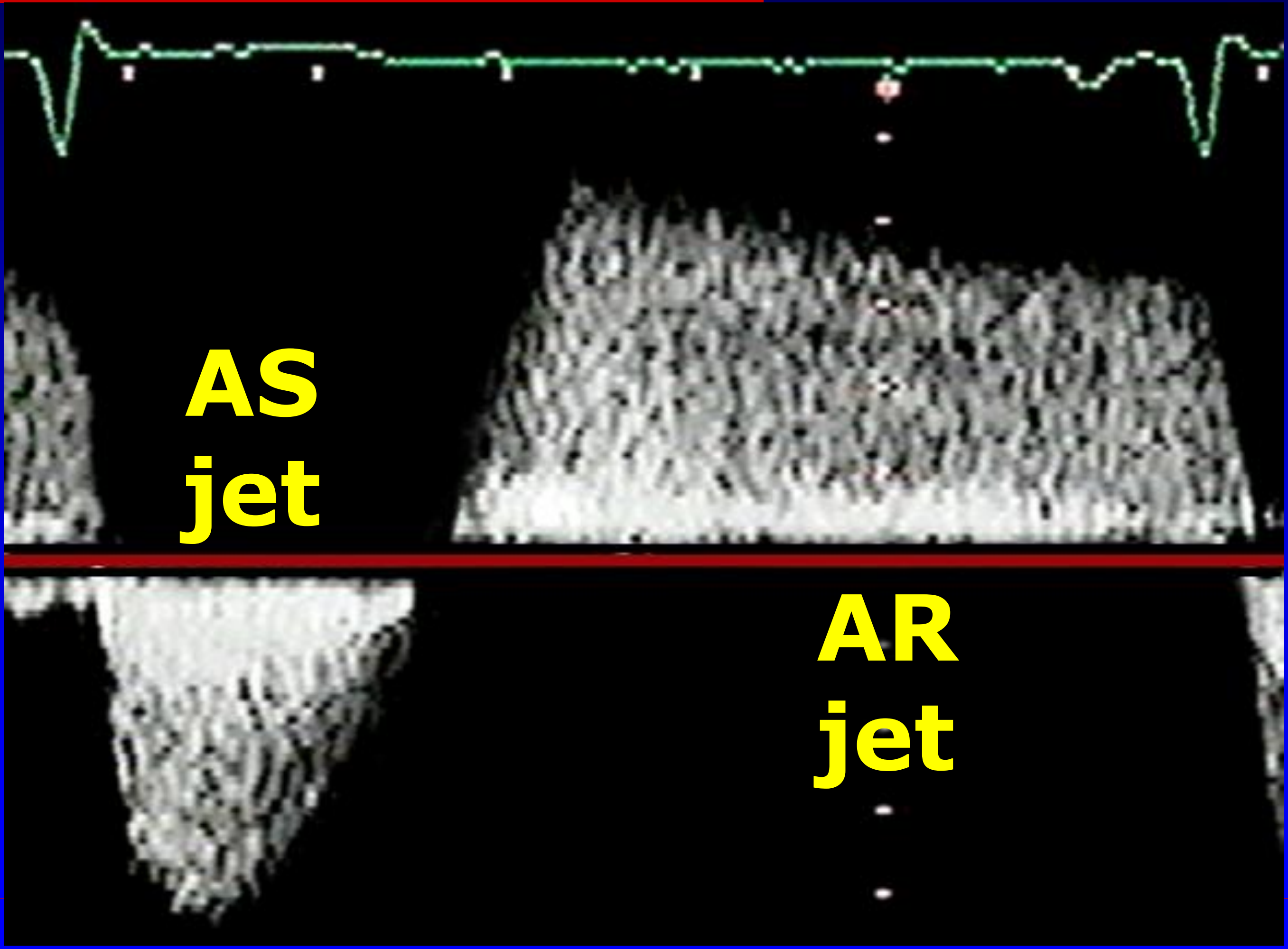
Apical 5-Chamber



Apical Long Axis



Spectral Doppler of the AV
Apical Five Chamber

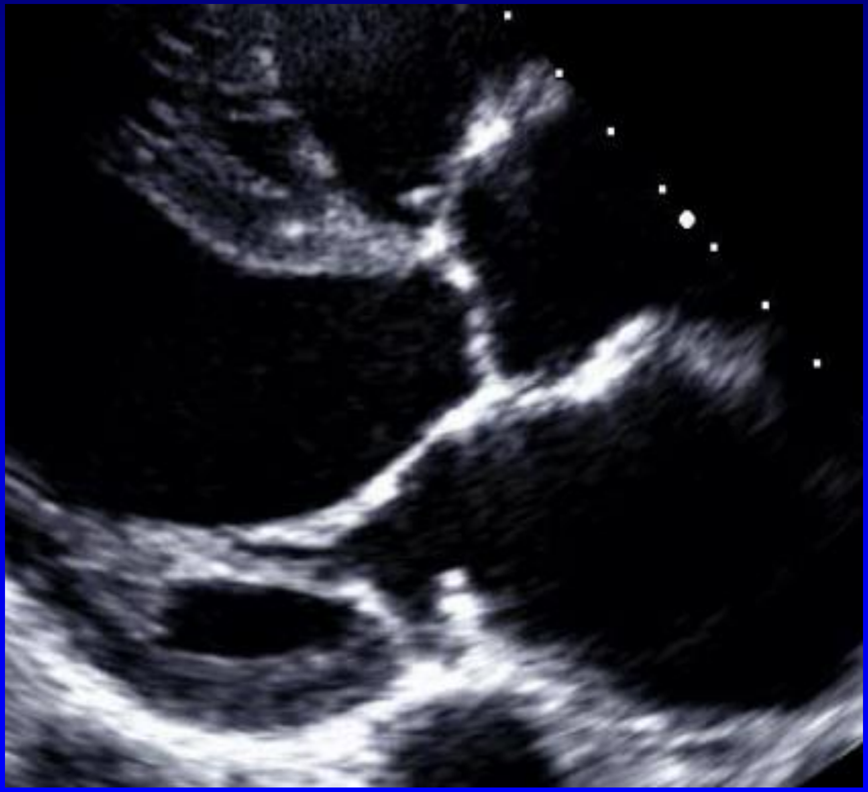


Bicuspid Aortic Valve

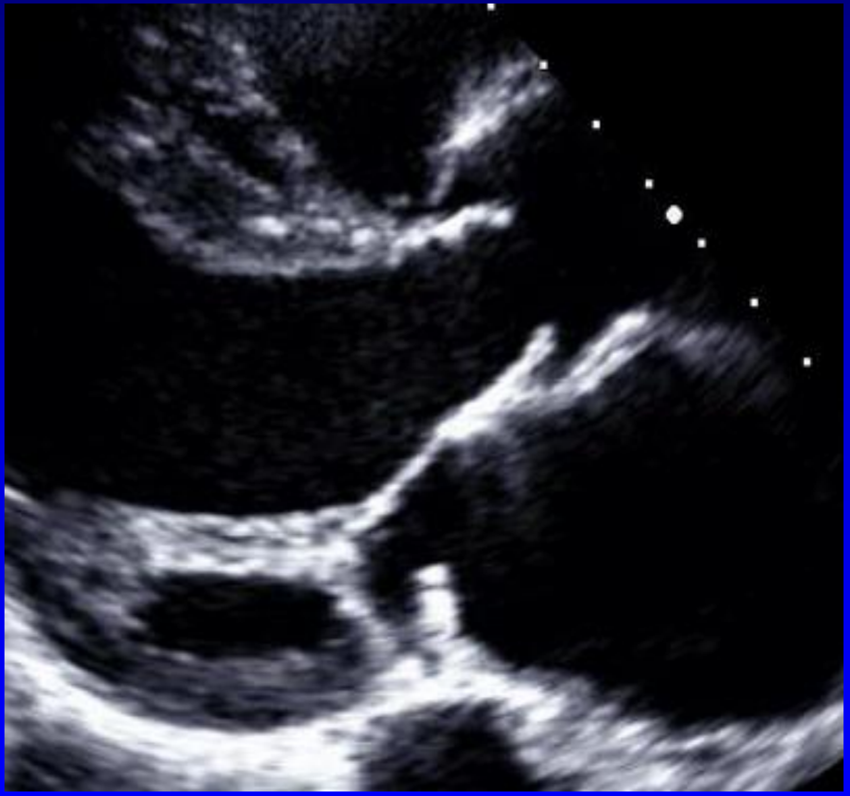
- Most common congenital anomaly (1-2%)
- Commissure may be horizontal or vertical
  - Horizontal: Anterior and Posterior leaflets
  - Vertical: Right and Left (coronary) leaflets
- Accel. calcification & premature stenosis
- Proximal aortopathy (even in normals)
- Associated aortic abnormalities

Bicuspid Aortic Valve
PLAX View – Doming

Diastole

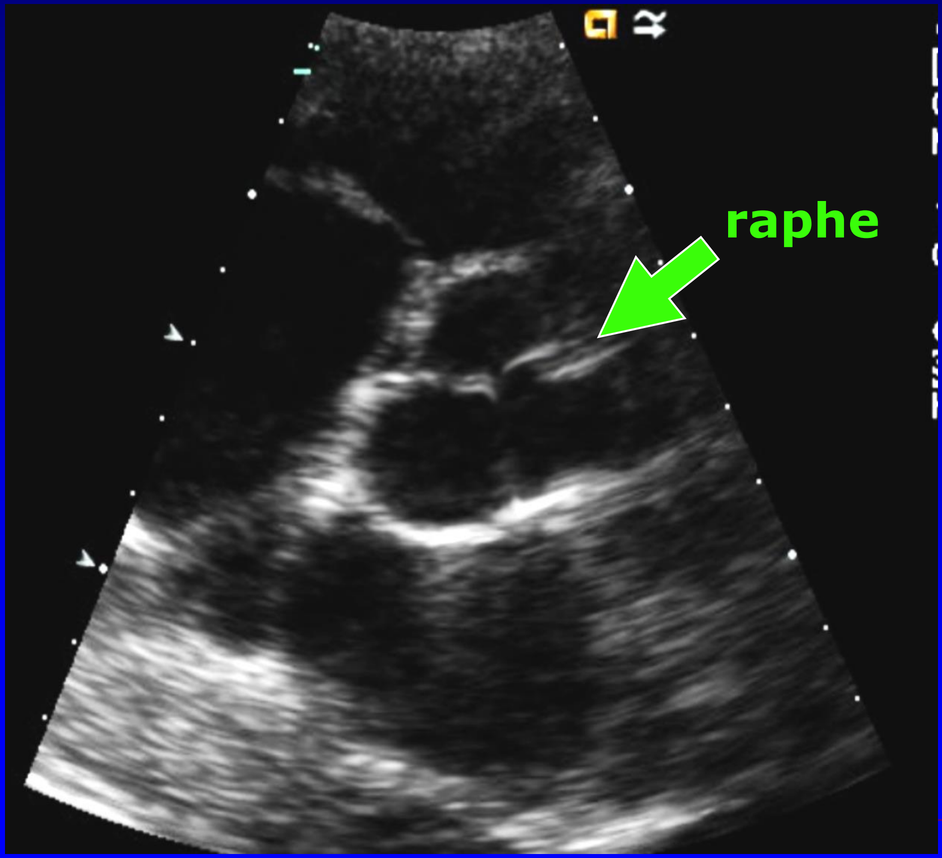


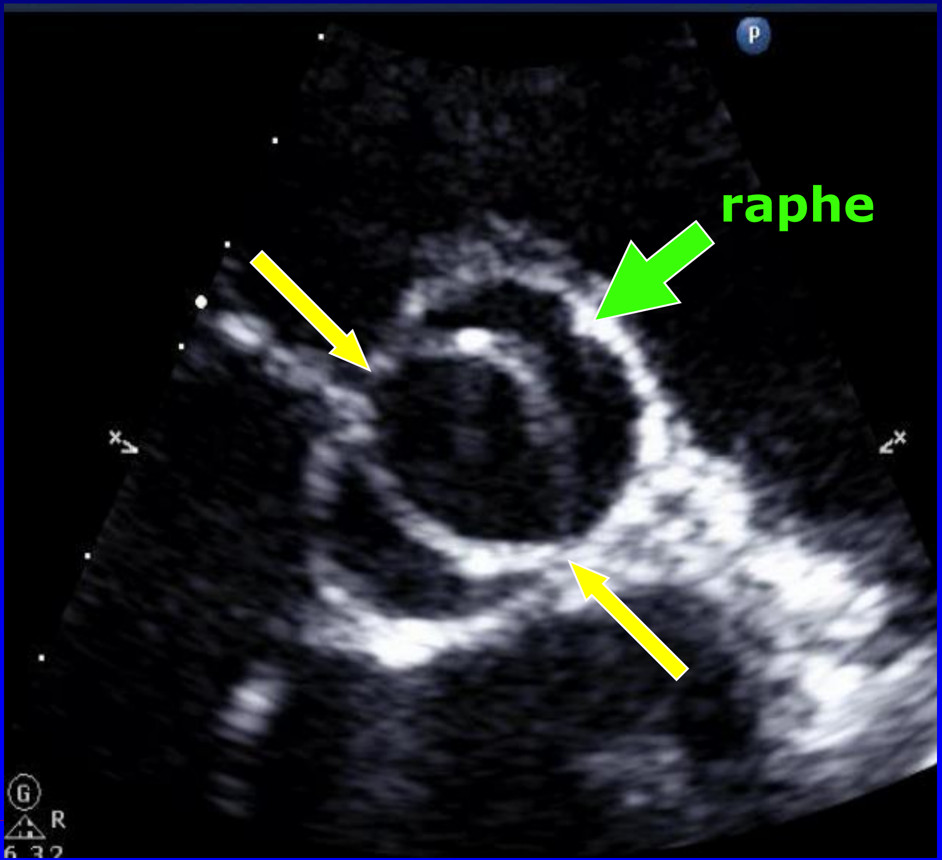
Systole



Bicuspid Aortic Valve
PSAX view morphology

Diastole **Systole**

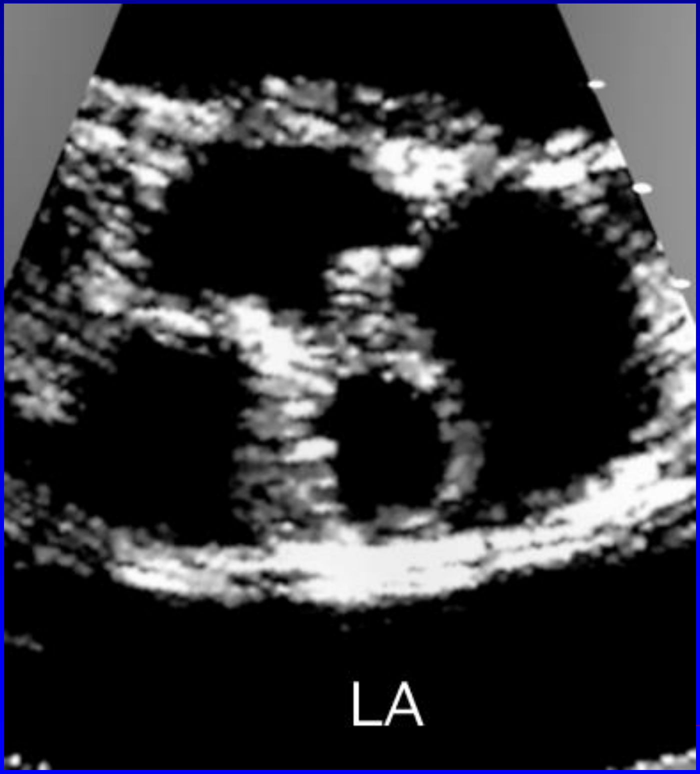




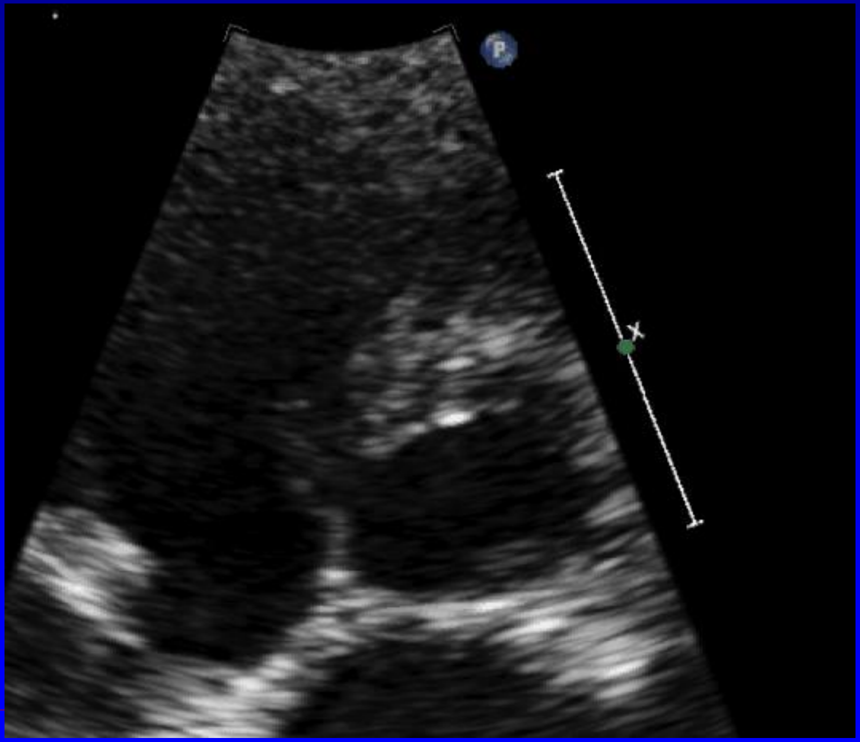
Systolic ellipsoid orifice identifies as

Aortic Valve:
Other Anomalies associated with AS

Unicuspid AoV



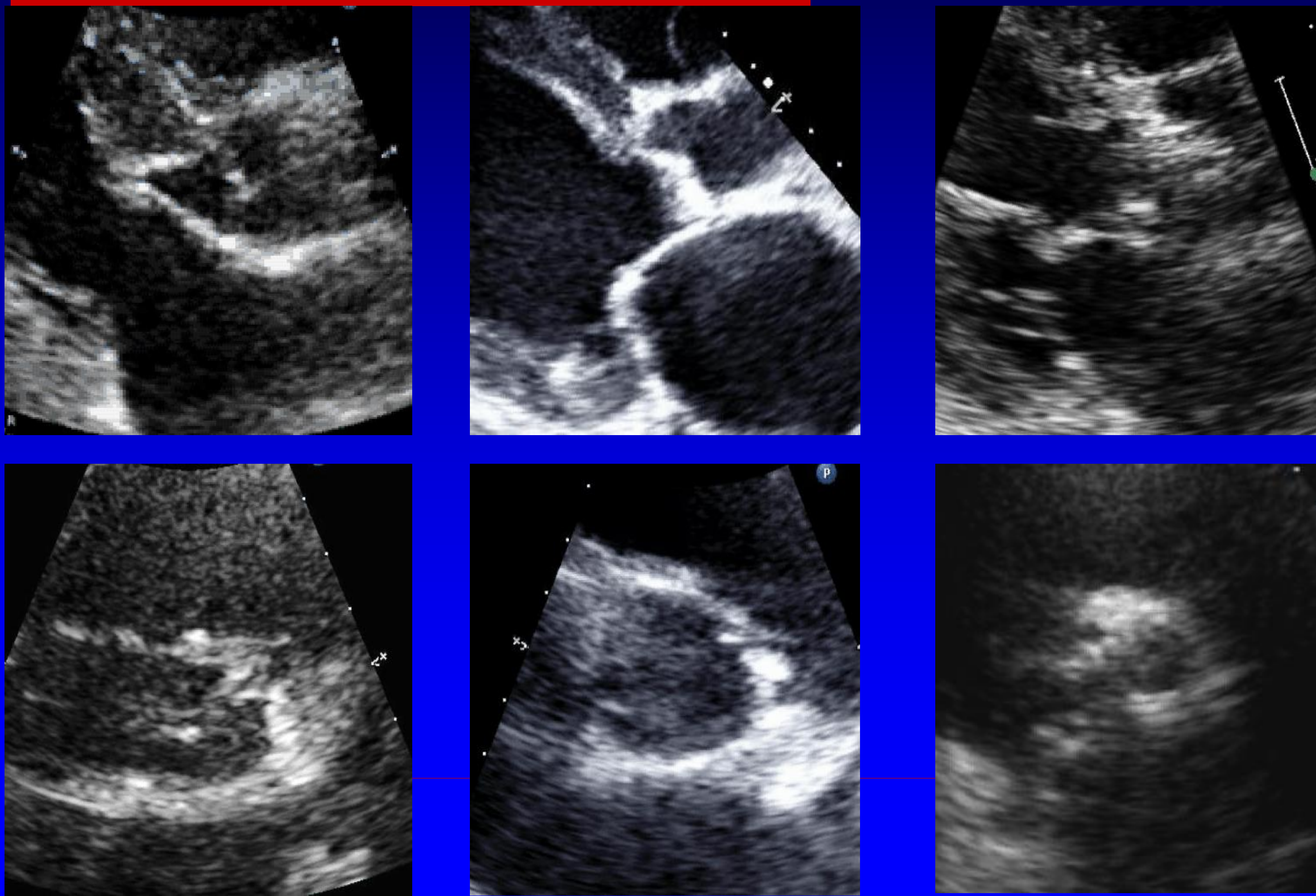
Quadracuspid AoV



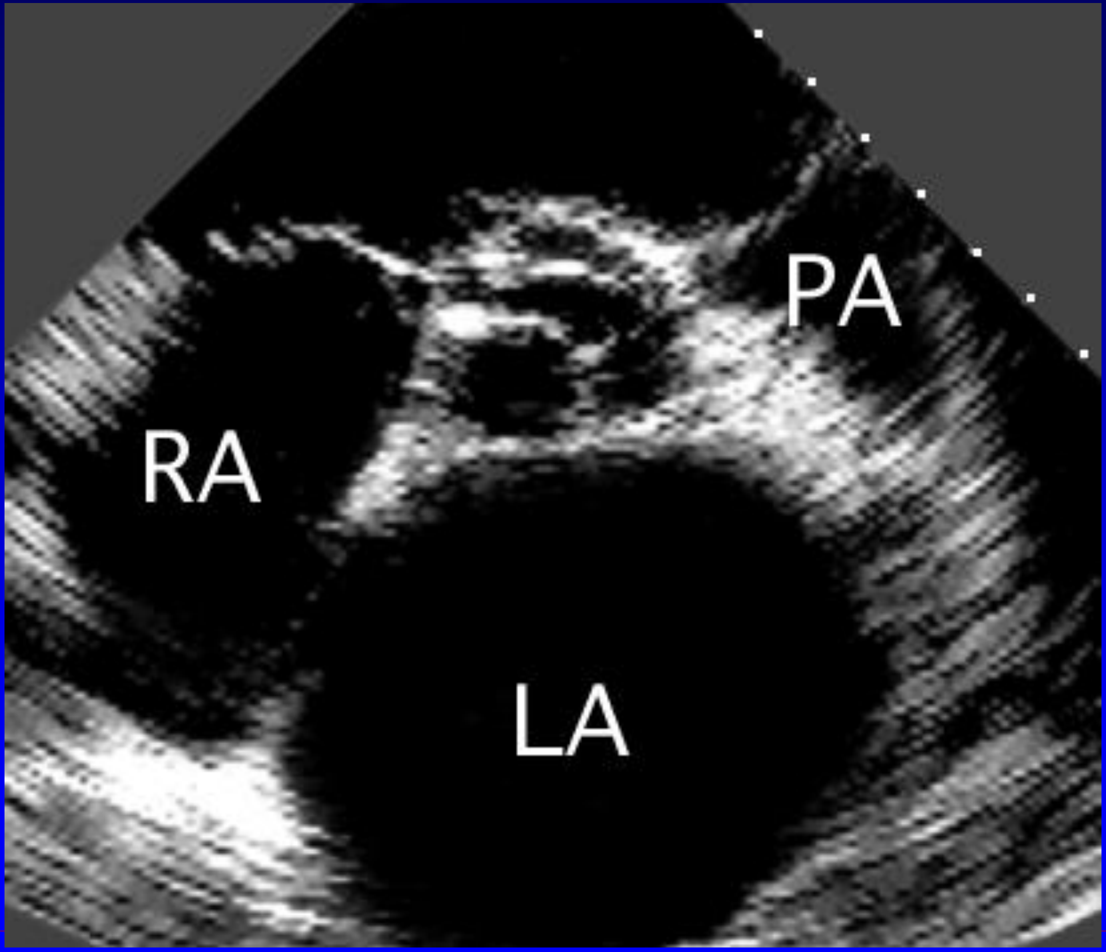
Aortic Stenosis
Etiology

- Senile/Degenerative Calcific
  - Calcification resembles ectopic bone
  - Risk factors similar to those for atherosclerosis
  - Renal dysfunction may accelerate
- Premature Calcific Bicuspid / Congenital
- Rheumatic
  - Less common in the United States
- Less common
  - Type 2 Hyperlipidemia, SLE, Irradiation, Paget's Dz

Calcific Aortic Stenosis:
Reduction in leaflet motion



Rheumatic Aortic Stenosis: *Commissural fusion*



Aortic Stenosis:
Physiologic Sequelae

- Chronic LV pressure overload
  - Myocardial Hypertrophy - Progressive
  - LA dilatation
- END STAGE: Limited Cardiac Output
  - Systolic Dysfunction
  - Diastolic Dysfunction
- SYMPTOMS:
  - Dyspnea and Fatigue (often subtle)
  - Typical and Atypical Chest Pressure
  - Syncope
  - Congestive Heart Failure

Evaluation of AS:
Echo Essentials

- Valve Anatomy - establish etiology
  - Exclude other forms of LVOT obstruction
- Severity of stenosis
- Physiologic sequelae
  - LV hypertrophy, diastolic fxn, systolic fxn
  - LA dilatation, Pulmonary hypertension
- Evaluate concurrent disease
  - Proximal aorta and arch
  - Aortic Valve Regurgitation, Mitral Disease

Aortic Stenosis:
Assessing Severity

- Peak AV Jet Velocity
- Mean AV Gradient
- Valve Area by continuity equation

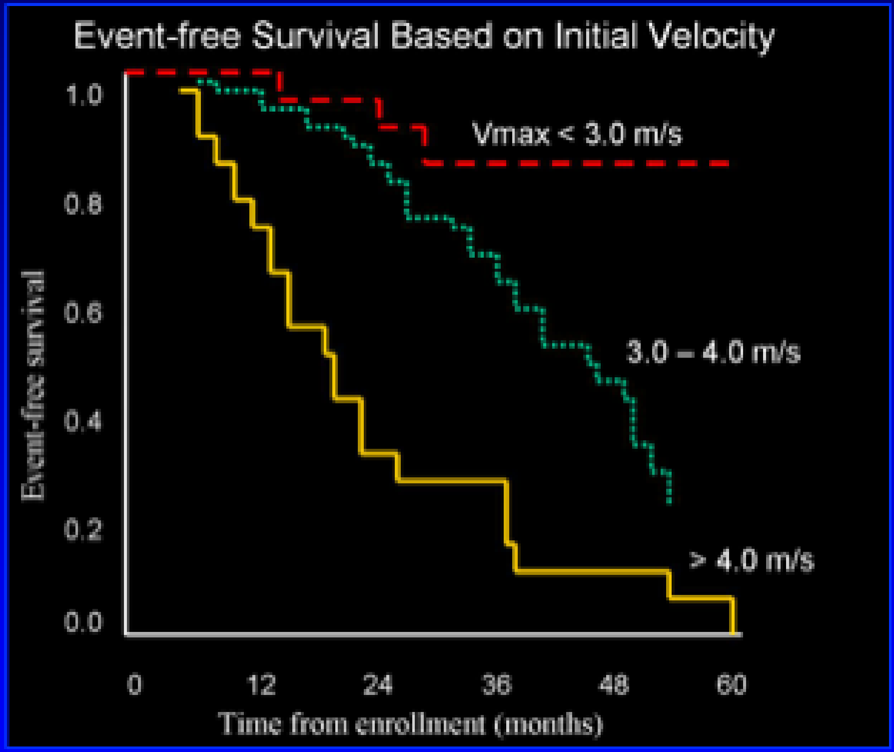
} ASE / EAE Recommend

- Velocity Ratio ("Dynamic Index")
- Planimetry

Baumgartner H, et al. JASE (2009) 22:1-23

Aortic Stenosis:
Prognosis of Velocity

- Variable Rate of Progression
  - Avg ~0.3 m/sec/year
- High rate of events, even for "asymptomatic" AS
- Baseline AoV Peak Jet Velocity, rate of change of velocity and functional status predict clinical outcome



Otto C, et al. Circulation (1997) 95:2262

Aortic Stenosis:
Peak Velocity

- Continuous Wave (CW) Doppler in Apical 5 Chamber View
- Must be parallel to the ejection jet
- Confirm – Right Parasternal
  - Suprasternal also possible
- Use highest velocity
  - Avoid feathery signals at tip
  - Piedoff – "non-imaging" probe
  - Decrease gains & adjust baseline

Aortic stenosis
Assessment by Peak Velocity

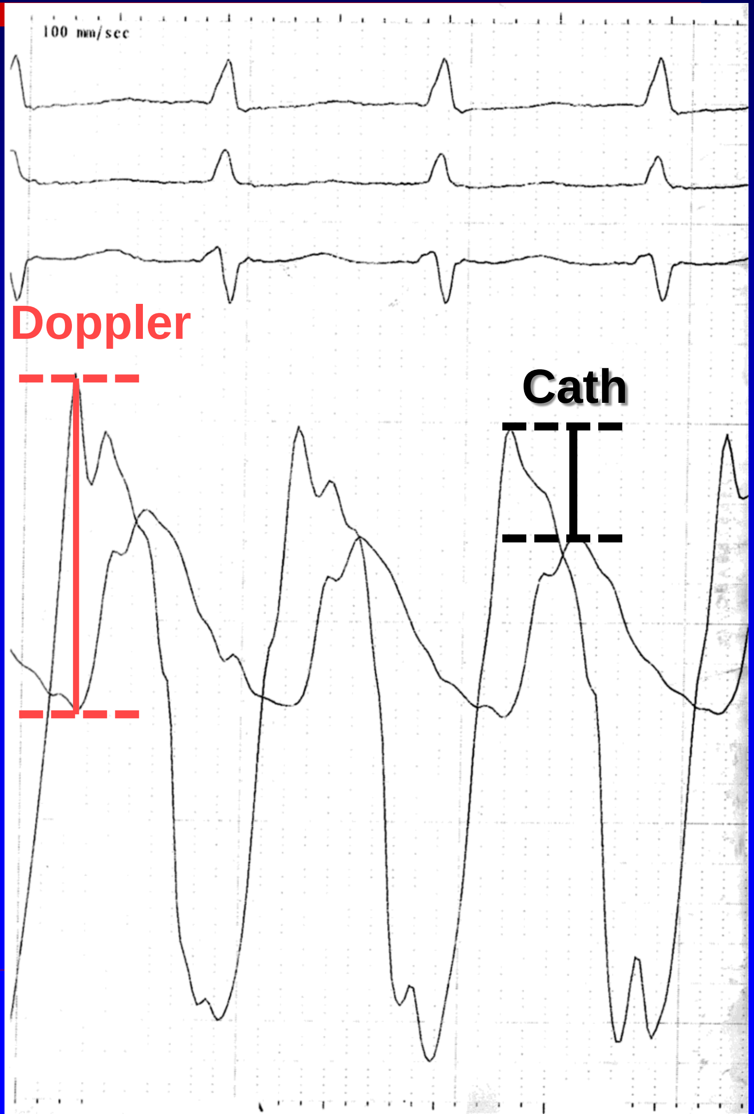
- Mild stenosis: 2.0 – 2.9 m/s
- Moderate stenosis: 3.0 – 3.9 m/s
- Severe stenosis: > 4.0 m/s
- “Very Severe” or “Critical” stenosis: > 5.0 m/s

Aortic Stenosis:
Peak Gradient

- **Peak Gradient = 4 $(V_{AV})^2$**
  - Peak Instantaneous Gradient

Instantaneous Gradient vs. Peak–to–Peak

- Echo a more "physiologic" measurement
- Doppler peak gradient always higher
- Mean gradient and AVA should correlate
- Gradients are flow dependent



Aortic Stenosis:
Mean Gradient

- **Mean Gradient**
  - Integration of velocity over time
  - Estimate – 0.7 * Peak Grad.
  - Correlates with cath Peak-to-Peak gradient

Aortic stenosis
Assessment by Mean Gradient

- Mild stenosis: < 20 mmHg
- Moderate stenosis: 20 – 39 mmHg
- Severe stenosis: ≥ 40 mmHg

Velocity and Gradient pitfall: *Influence of Cardiac Output*

- **High CO = High gradient**
  - Aortic regurgitation
  - Hyperdynamic function
- **Low CO = Low gradient**
  - Reduced ejection fraction
  - Small ventricular cavity/LVH
  - High systemic vascular resistance/impedance
  - Significant mitral regurgitation

Aortic stenosis
Assessment of Valve Area

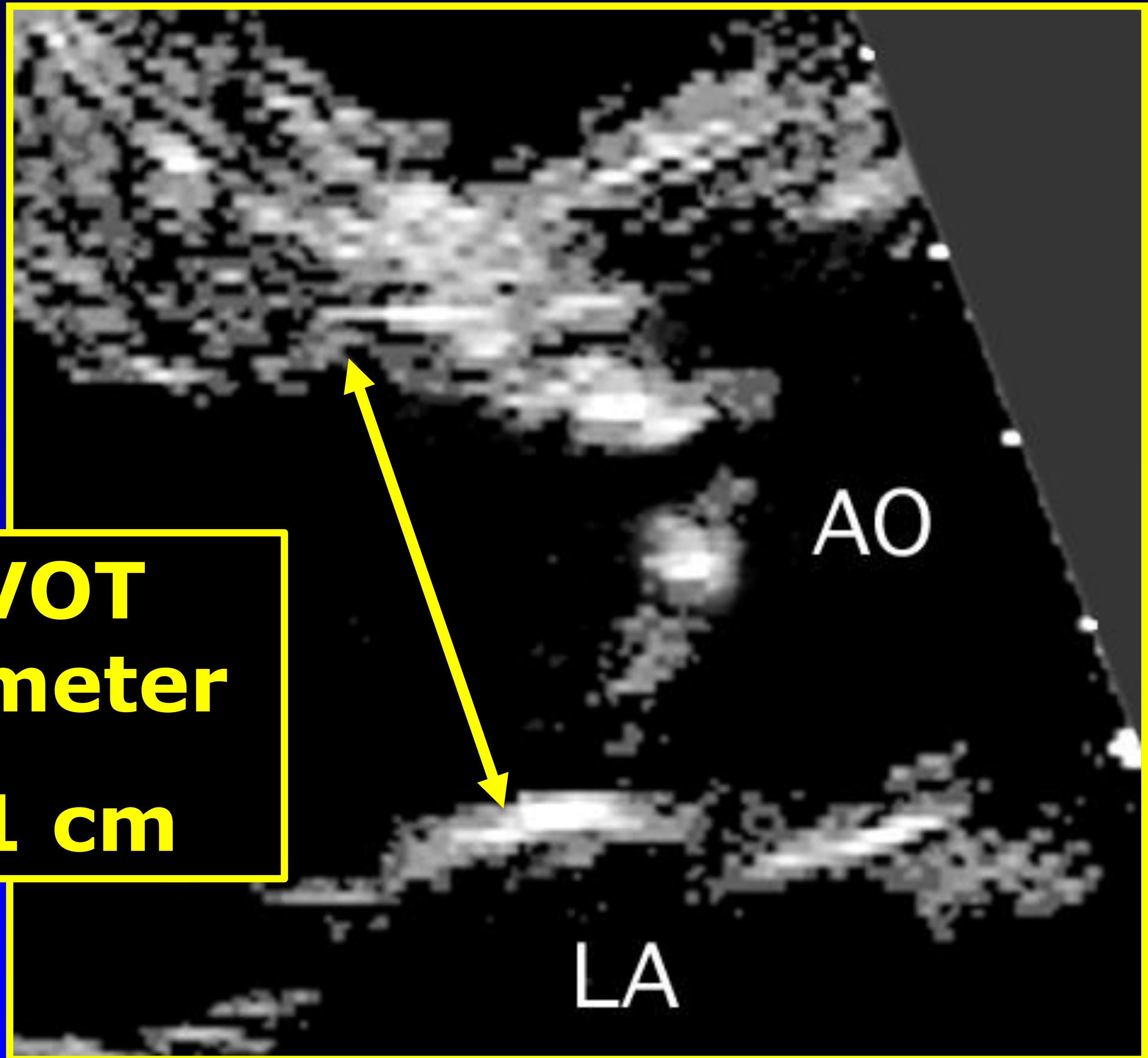
- Normal valve area: = 3 - 4 cm^2
- Mild stenosis: > 1.5 cm^2
- Moderate stenosis: 1.0 – 1.5 cm^2
- Severe stenosis: < 1.0 cm^2
- "Critical" stenosis: < 0.7 cm^2

Calculation of AV Area: *Continuity Equation*

- Based on conservation of mass

Flow within LVOT = Flow across AV

- $\text{LVOT area} * VTI_{LVOT} = AVA * VTI_{AV}$
- $[\pi * (LVOT_{rad})^2] * VTI_{LVOT} = AVA * VTI_{AV}$
- $\dfrac{[\pi * (LVOT_{radius})^2] * VTI_{LVOT}}{VTI_{AV}} = AVA$

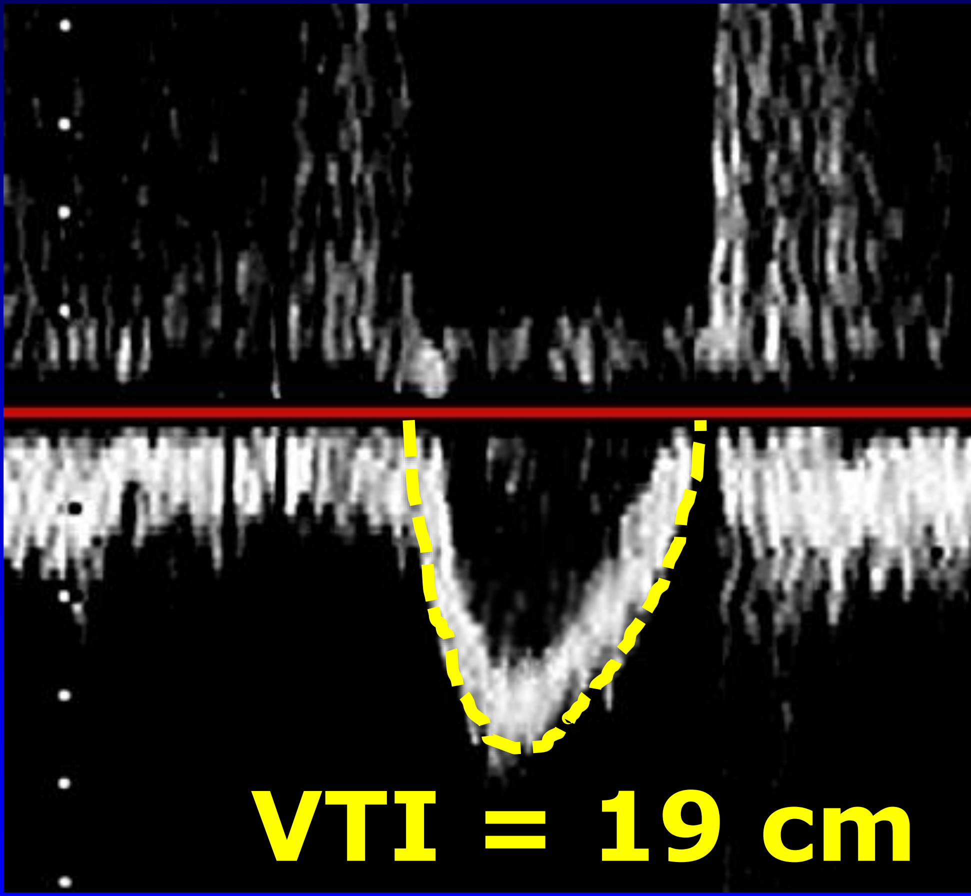


LVOT diameter

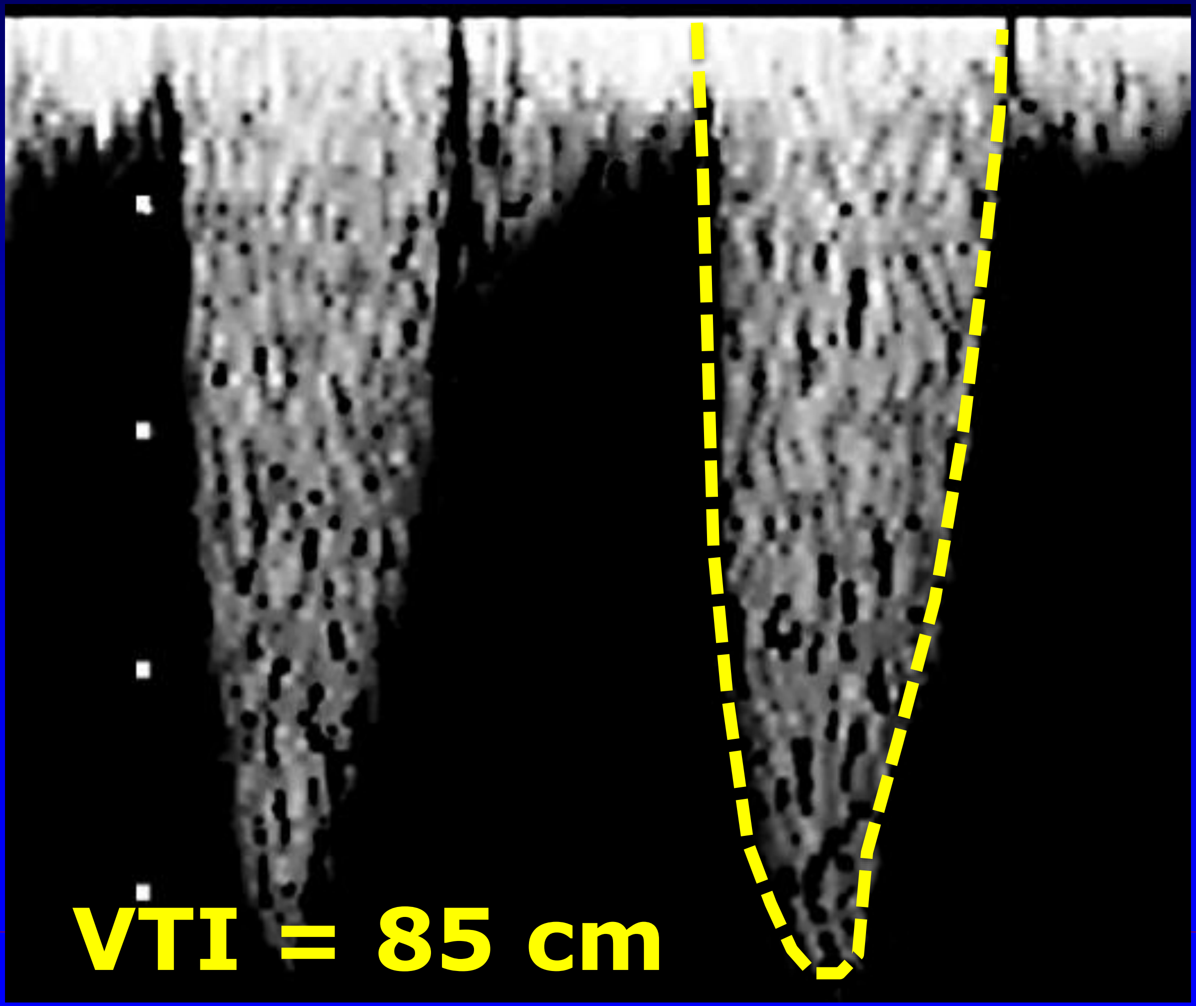
2.1 cm

Flow through LVOT
Pulse Wave Doppler

- Spectral Envelope
  - With sample volume in LVOT
- **Velocity Time Integral (VTI)**
  - flow through a single point



Flow Across the Aortic Valve: *Continuous Wave Doppler*



Calculating Aortic Valve Area

- $$AVA = \frac{(Diameter_{LVOT} / 2)^2 \times \pi \times VTI_{LVOT}}{VTI_{AV}}$$
- $$AVA = \frac{(2.1\ cm / 2)^2 \times 3.14 \times 19\ cm}{85\ cm}$$
- **AVA = 0.7 cm^2**

Pitfalls of the Continuity Equation

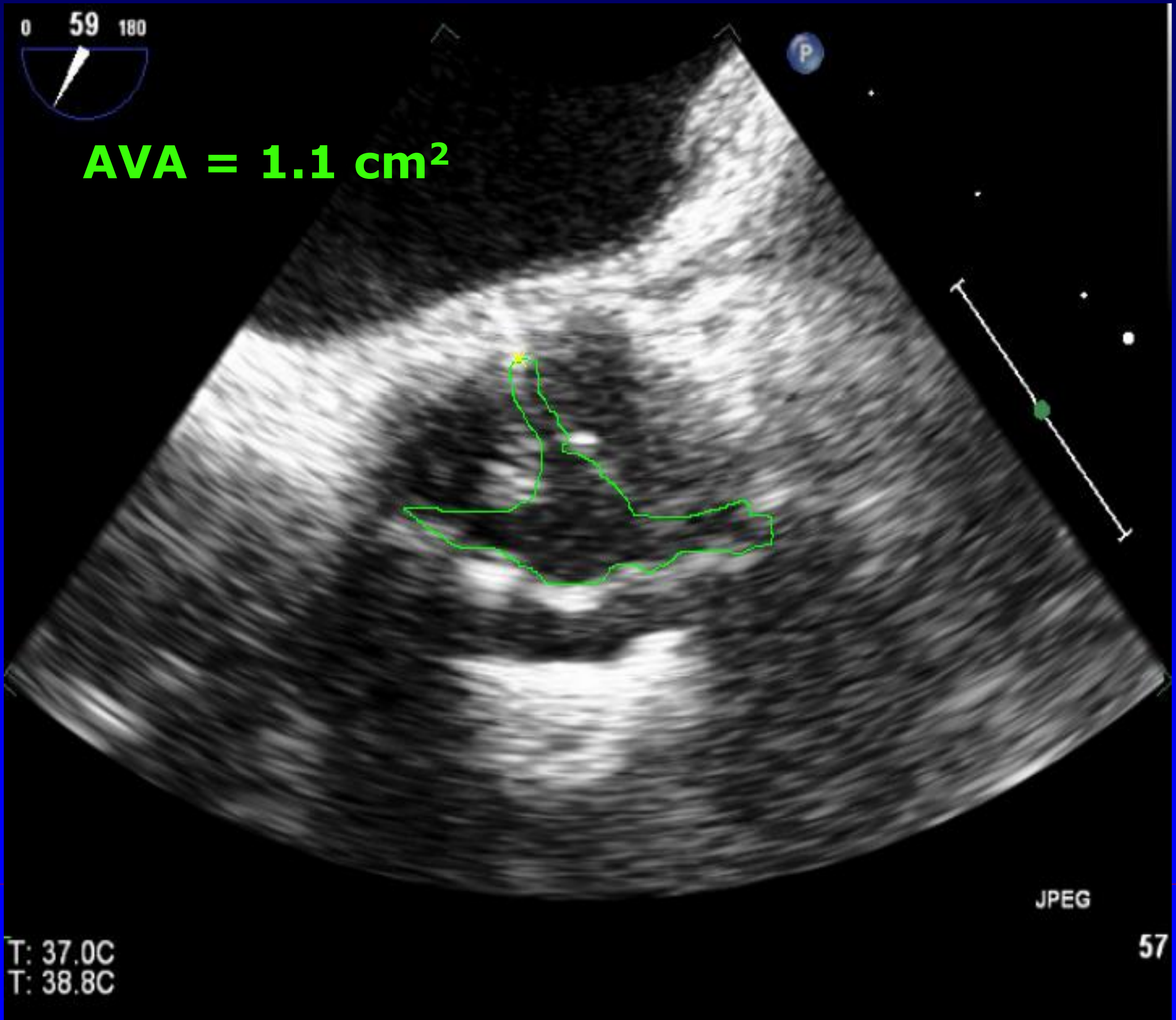
- LVOT measurement
  - Diameter is squared - can propagate large error
- LVOT velocity
- AV velocity
  - Missing the Peak: use multiple sites / Piedoff
  - Use highest velocity obtained
  - Beware MR

Doppler Velocity Index

- Eliminates errors of LVOT measurement
  - **$DVI = VTI_{LVOT} / VTI_{AV}$**
- Criteria for Severe AS:
  - **$DVI < 0.25$**

Relatively flow-independent measure of stenosis

Planimetry of the Aortic Valve



Planimetry

- Correlates with invasively obtained areas
- Flow dependent
  - Difficult to distinguish decreased opening due to LV failure
- TEE superior - use of color flow area
- Dense calcification reduces accuracy

Summary

able 3 Recommendations for classification of AS severity

	Aortic sclerosis	Mild	Moderate	Severe
ortic jet velocity (m/s)	≤2.5 m/s	2.6–2.9	3.0–4.0	>4.0
lean gradient (mmHg)	–	<20 (<30[a])	20–40[b] (30–50[a])	>40[b] (>50[a])
VA (cm^2)	–	>1.5	1.0–1.5	<1.0
ndexed AVA (cm^2/m^2)		>0.85	0.60–0.85	<0.6
elocity ratio		>0.50	0.25–0.50	<0.25

ESC Guidelines.
AHA/ACC Guidelines.

Baumgartner H, et al. JASE (2009) 22:1-23

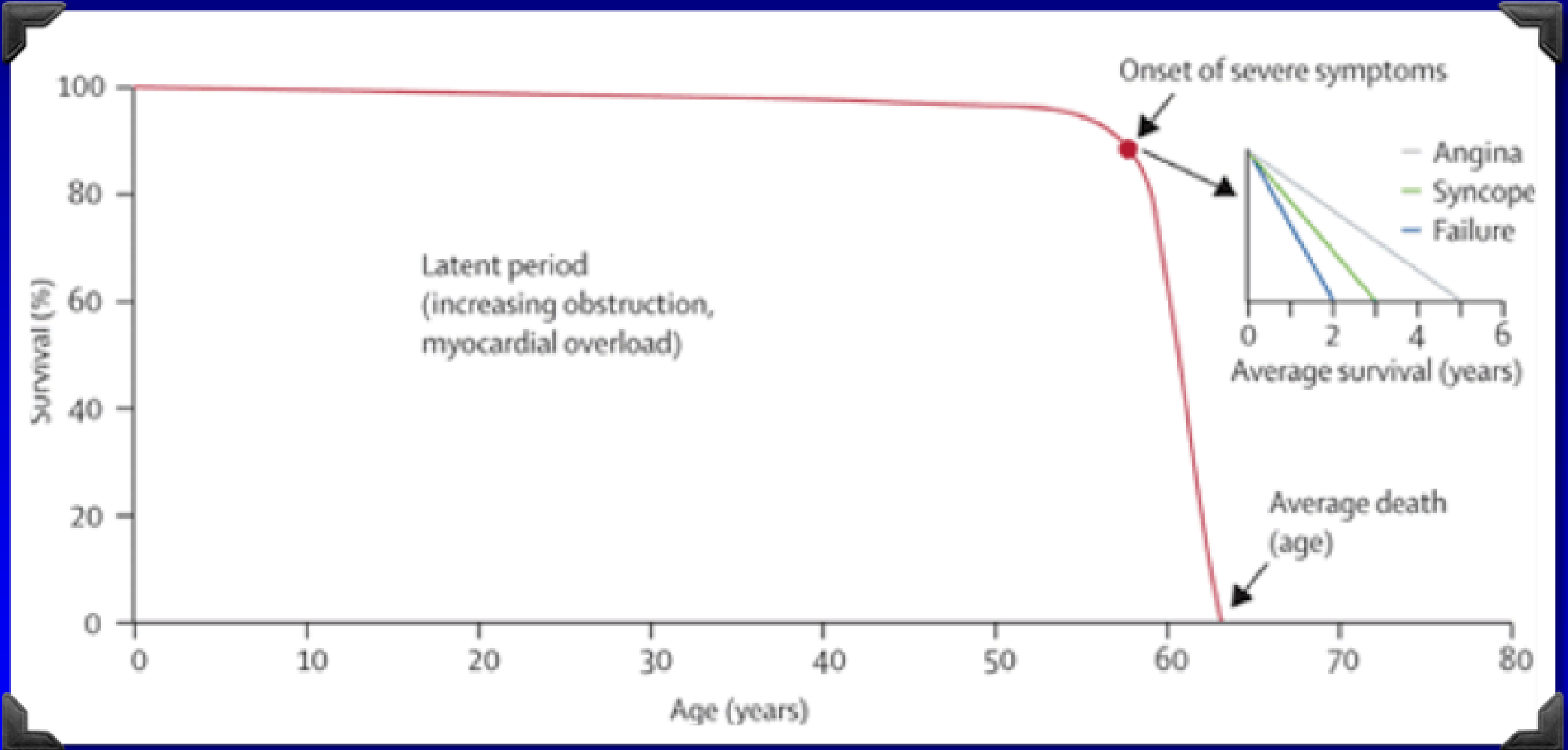
So…

My patient has severe aortic stenosis!

What do I do? Who do I talk to?

WHEN SHOULD I OPERATE?

The Good Old Days:
The Symptomatic "Cliff"



Braunwald E, et al. *Circulation* **(1968)** 38:61-67

Time for an Update:
The Asymptomatic "Slide"

Why??

- Progression of Stenosis
- Worsening LV Hypertrophy
  - Subclinical Diastolic Dysfunction
  - Subclinical Systolic Dysfunction
- LA Pressure Overload and Congestion
  - Pulmonary Hypertension
- Patients in denial
- Doctors in denial

←don't recognize/accommodate sx

“Stages” of Disease

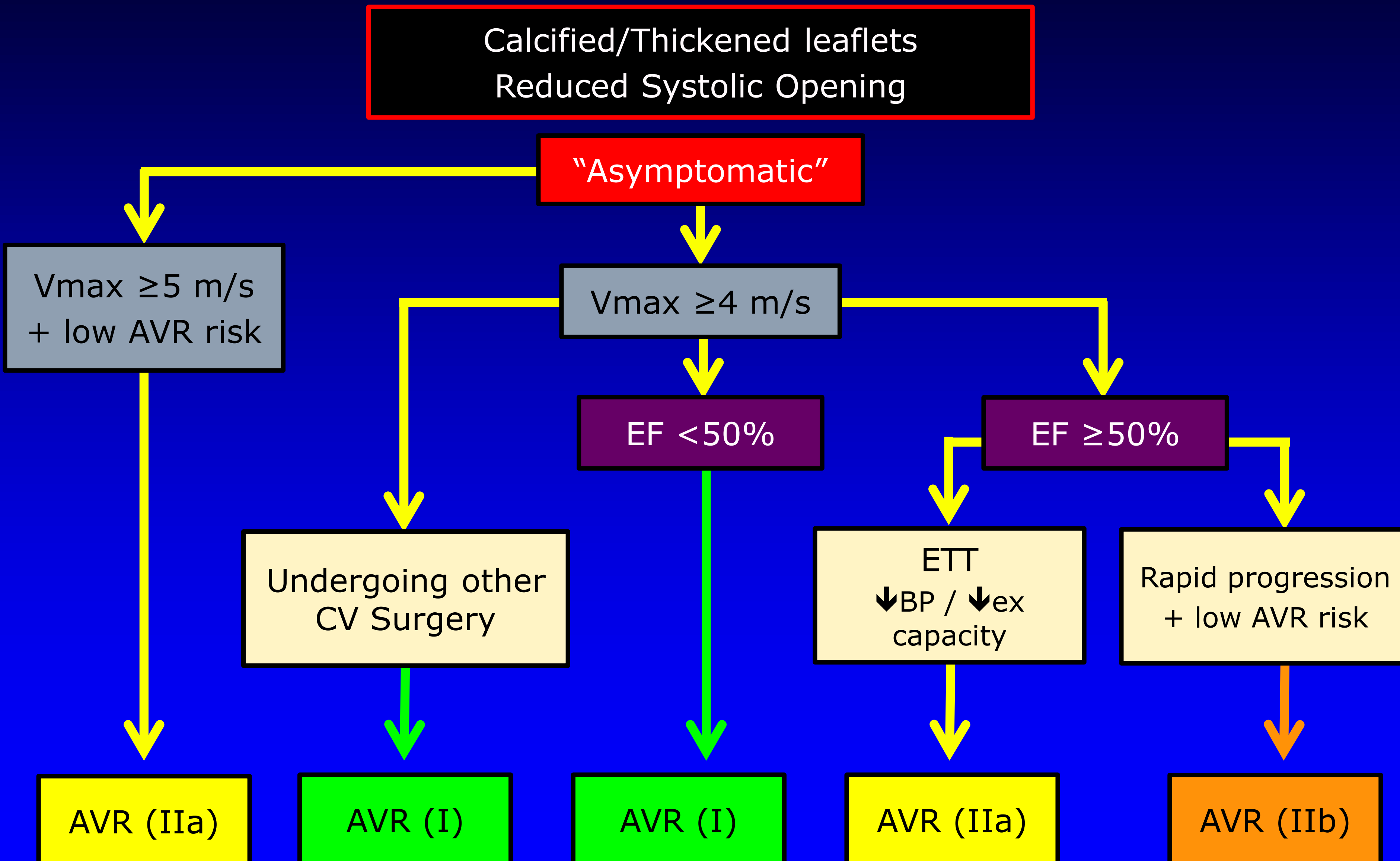
- **Stage A:**
  - At risk for disease
- **Stage B:**
  - Progressive disease
- **Stage C:**
  - Severe disease (asymptomatic)
- **Stage D:**
  - Severe disease (symptomatic)

- More accurate definition of severity
- More precise decisions on when to intervene

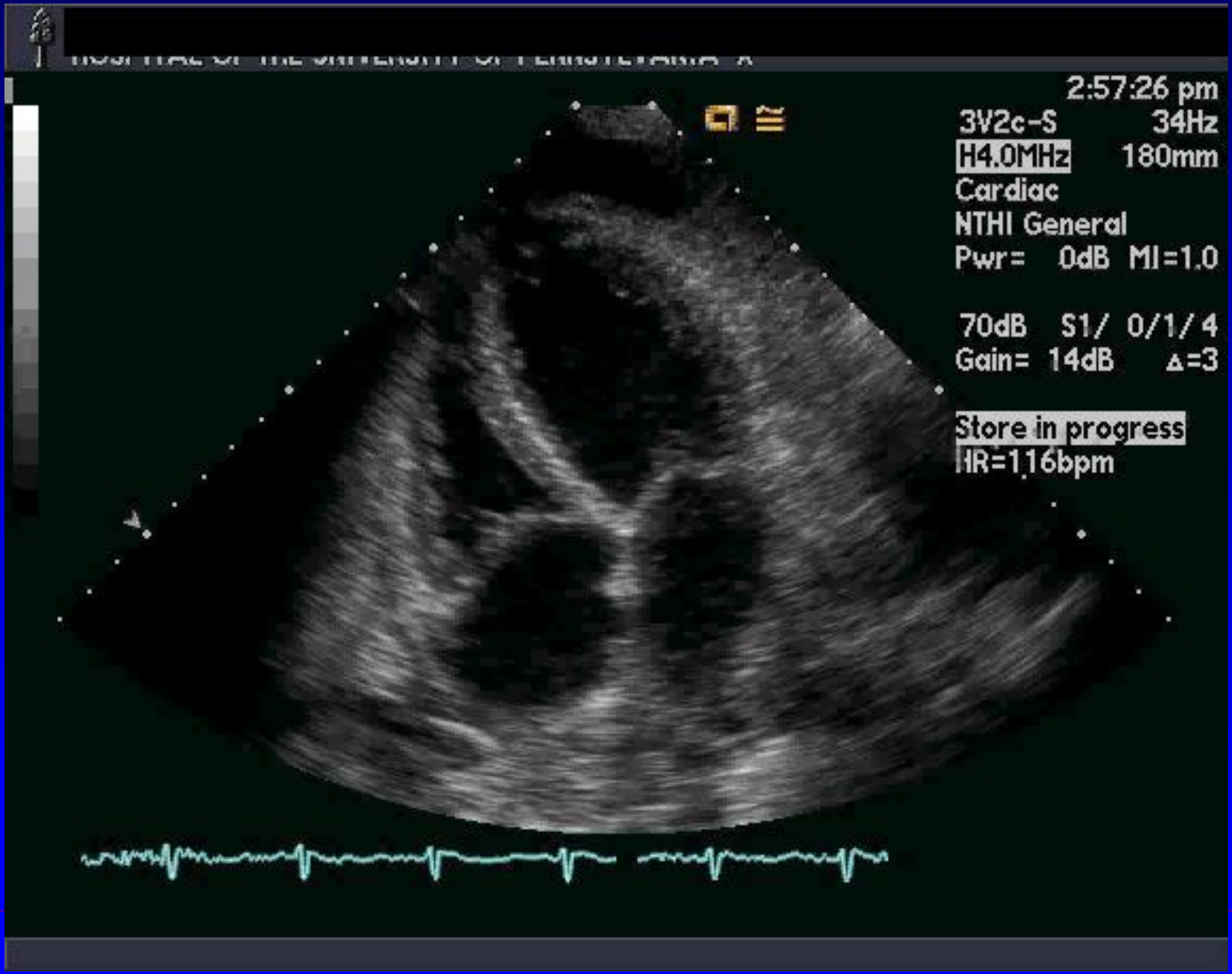
"Stage C" can be subdivided:

- **Stage A:** — **Observe**
  - At risk for disease
- **Stage B:** — **Observe**
  - Progressive disease
- **Stage C1:** — **???**
  - Severe (asymptomatic) – Compensated LV
- **Stage C2:** — **Intervene**
  - Severe (asymptomatic) – Decompensated LV
- **Stage D:** — **Intervene**
  - Severe disease (symptomatic)

Guidelines Assist in Decision-Making



Decreased LV Function:
"Low Gradient" Aortic Stenosis



Low Output – Low Gradient AS

- Why does a low EF pt have an AVA of 0.5 cm^2, but a mean gradient of 15mmHg?
  - Because low SV (low flow) leads to low gradients
- **"Real AS"**
  - 1^{o} Prob: Severe obstruction to flow
  - 2^{o} Prob: Depressed LVEF
- **"Pseudo-AS"**
  - 1^{o} Prob: Depressed LVEF
  - 2^{o} Prob: Mild/Mod obstruction is made to look severe by ↓SV

Low Output – Low Gradient AS

- Dobutamine Stress Testing
  - Increase LV contractility -> Increase Stroke Volume

- Increase Stroke Volume by 20% ->
  - **Real AS** Peak vel/mean gradient significantly ↑↑
AVA stays unchanged or ↓slightly
  - **Pseudo AS** Peak vel/mean gradient minimal ↑
AVA↑

- What if LV contractility / SV don't increase?

Low Gradient - Normal EF

- EF ≥50%, AVA <1 cm^2, mean grad <40mmHg
  - Whah???...
- **Still a stroke volume problem!!**
  - **SV_{index} ≤35 ml/m^2 despite EF**
- "Typical" patient:
  - Older, h/o hypertension, women
  - Concentric LVH, small cavity, impaired filling
  - Markedly increased vascular impedance

Pibarot P, Dumesnil JG. **Heart** (2010) 96:1431-33

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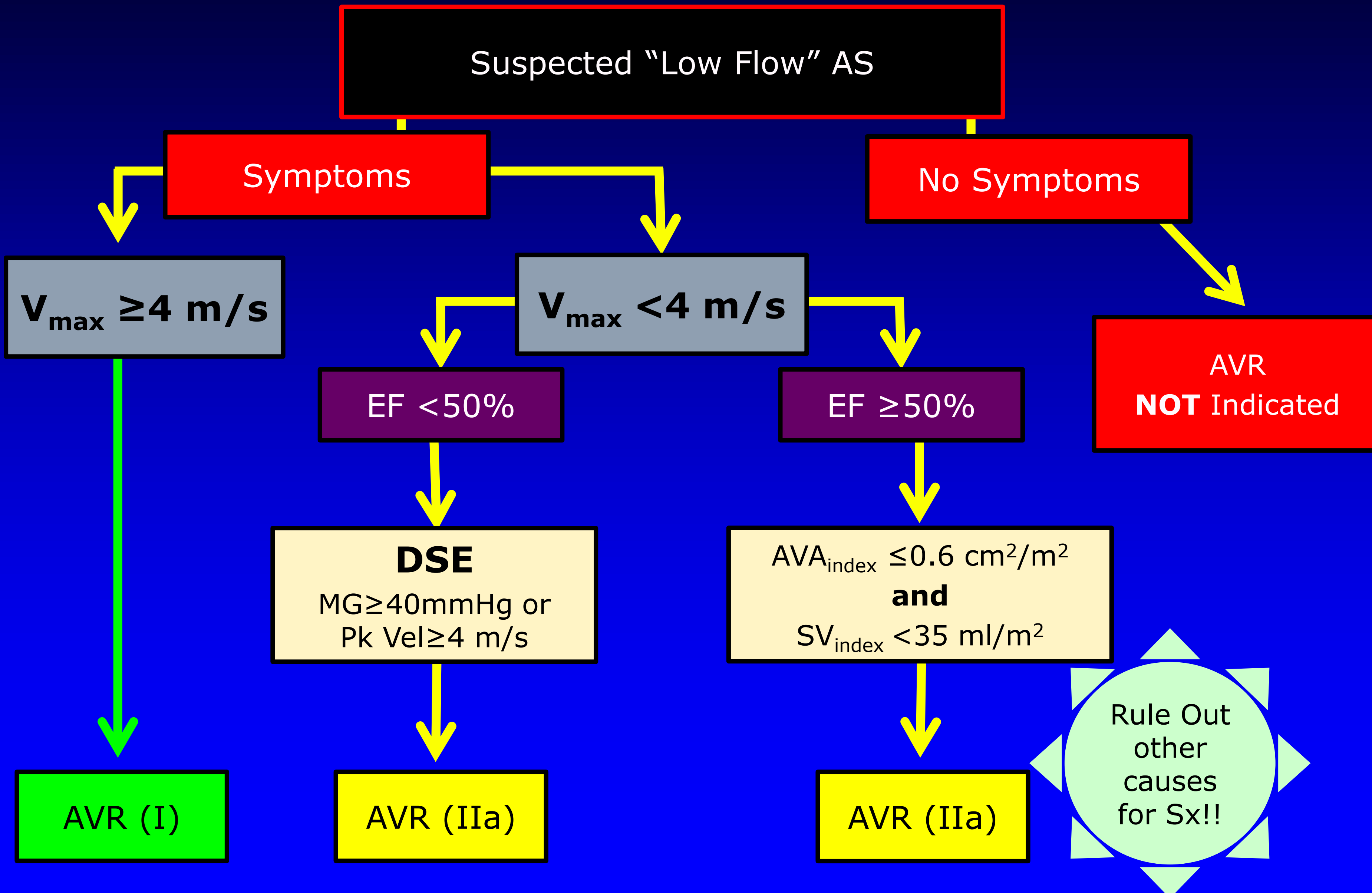

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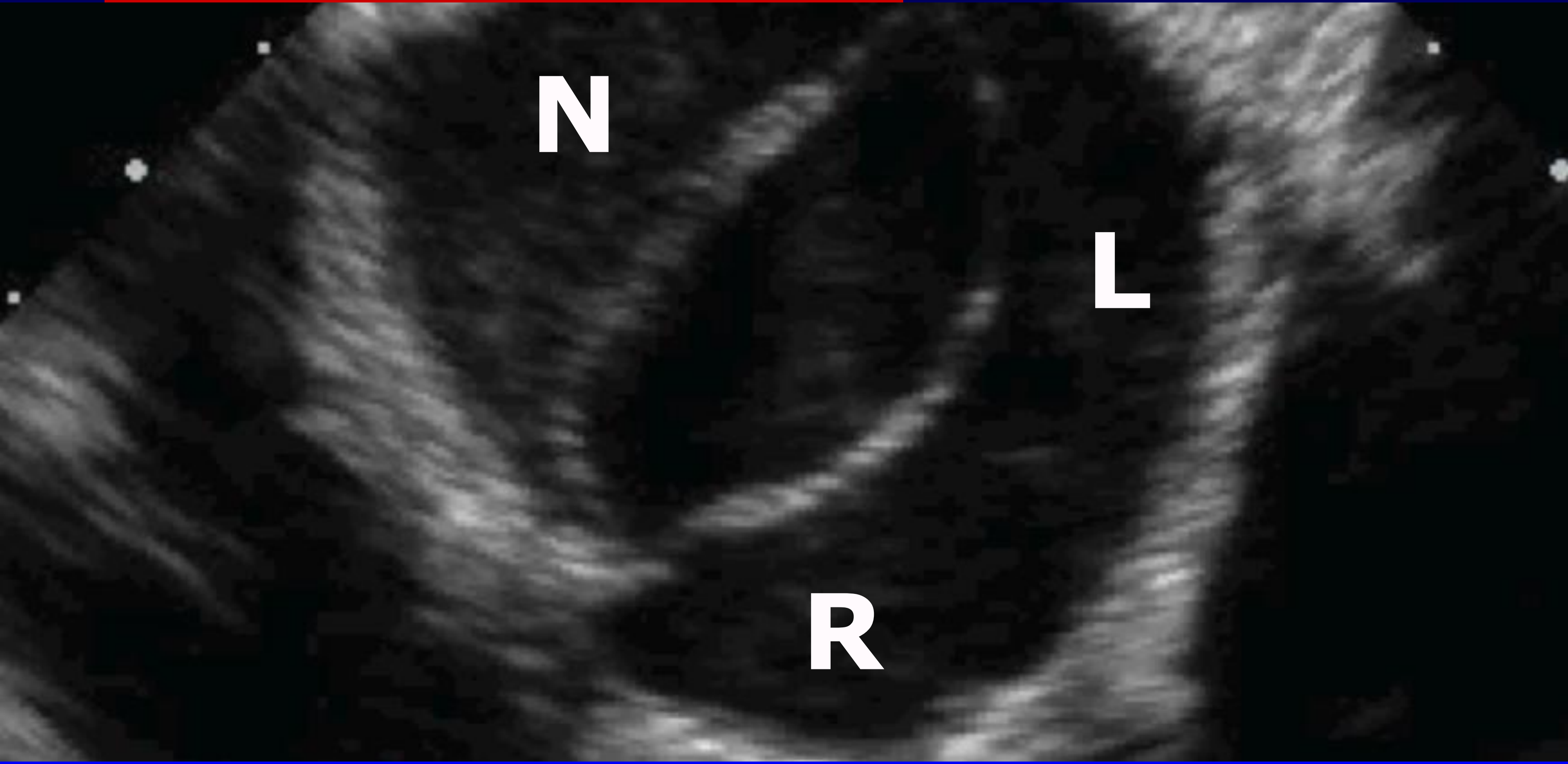
Short Axis TEE view - AoV



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DI = 130/410

DI = 0.32

Thank You!