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Multimodality Imaging in Heart Valve Disease

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Disclosures

- Excision Medical – consulting
- Edwards Lifesciences – honoraria

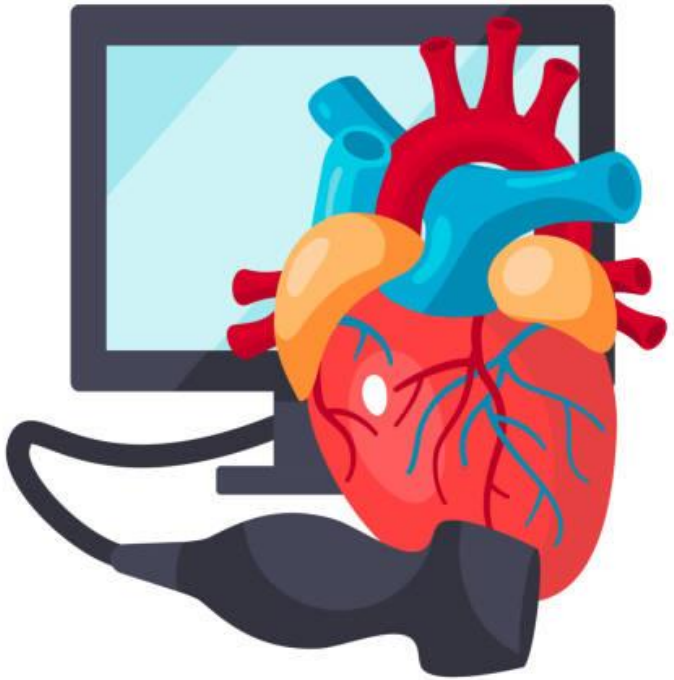
- I am not a cardiac imager. This is my simplified view of the space.

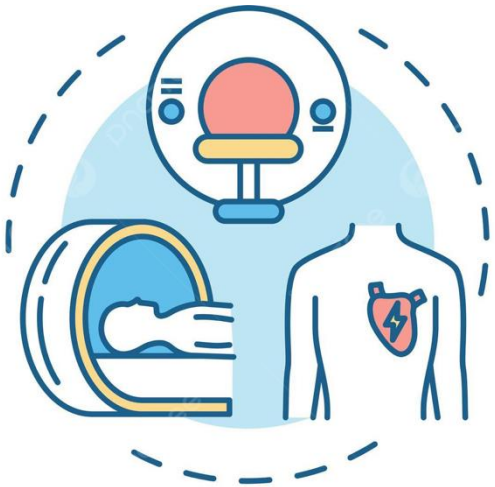
Outline

- Assessment of aortic stenosis
  - Particularly in non-high gradient phenotypes
- Quantification of aortic regurgitation
- Mitral and tricuspid valve imaging and procedural planning

Echocardiography remains gold standard first test

- Simple, widely available.
- Easy to perform, no risk.
- Most guideline documents based on TTE parameters.
- However, clearly falls short in certain situations and multimodality assessment is vital.




Cardiac CT

MRI

classroomclipart.com
http://classroomclipart.com

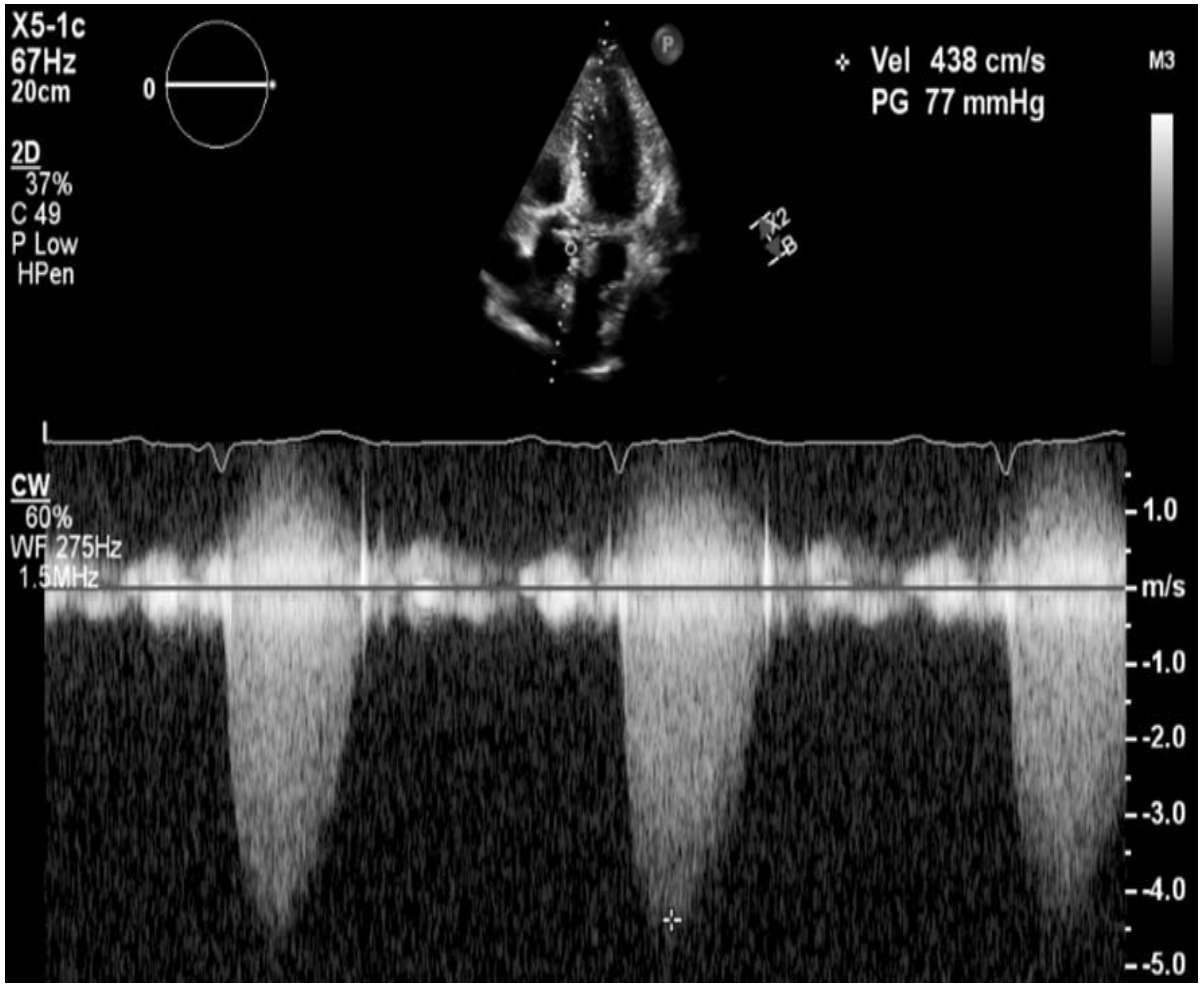
Aortic stenosis

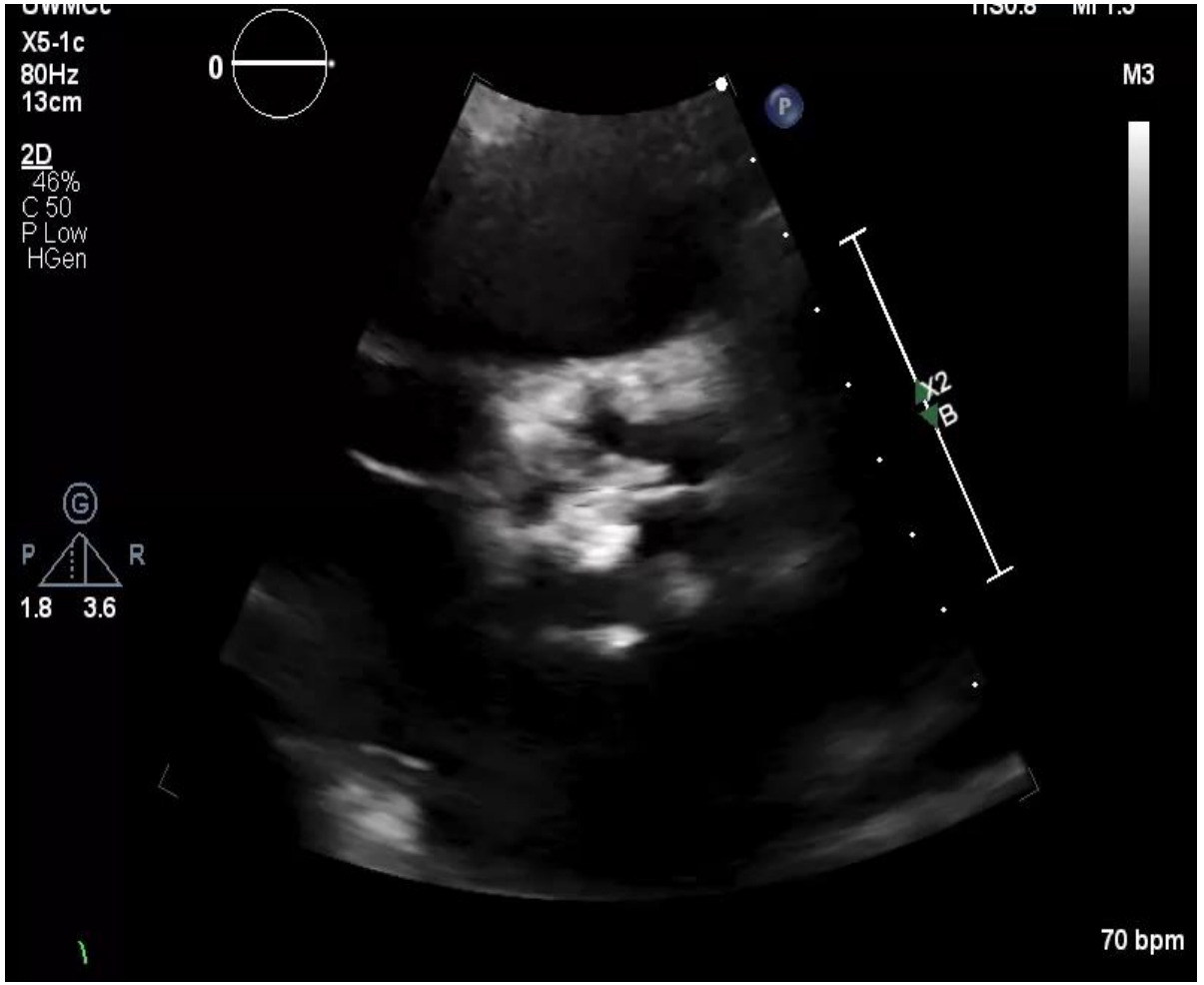
Straightforward aortic stenosis cases

Aortic Stenosis

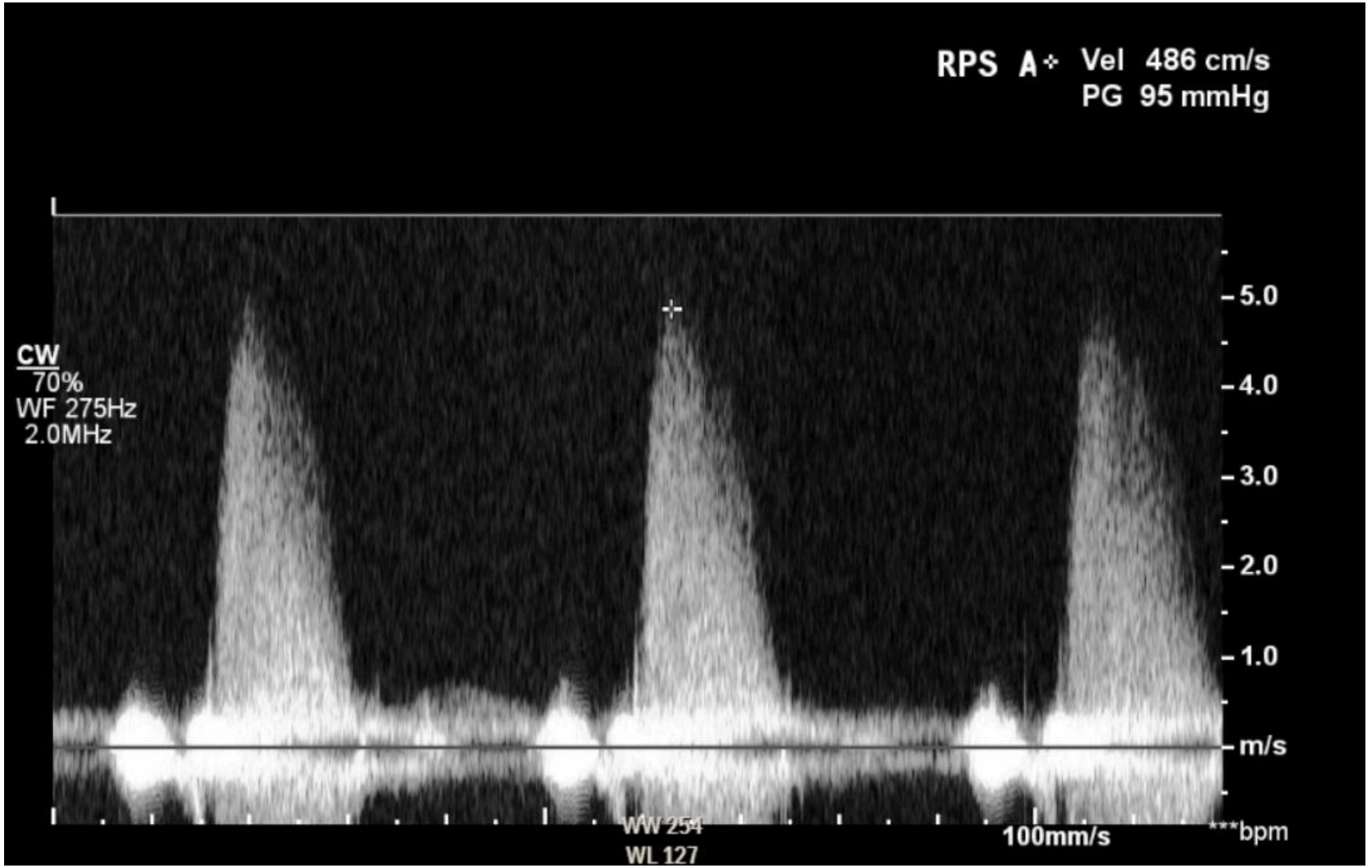
	Mild	Moderate	Severe
AV Peak Velocity (m/s)	2.6 - 2.9	3.0 - 4.0	≥ 4.0
AV Mean PG (mmHg)	< 20	20 - 40	≥ 40
AVA (cm^2)	> 1.5	1.0 - 1.5	< 1.0

Indexed valve area to BSA is preferred for specificity: <0.6 cm^2/m^2

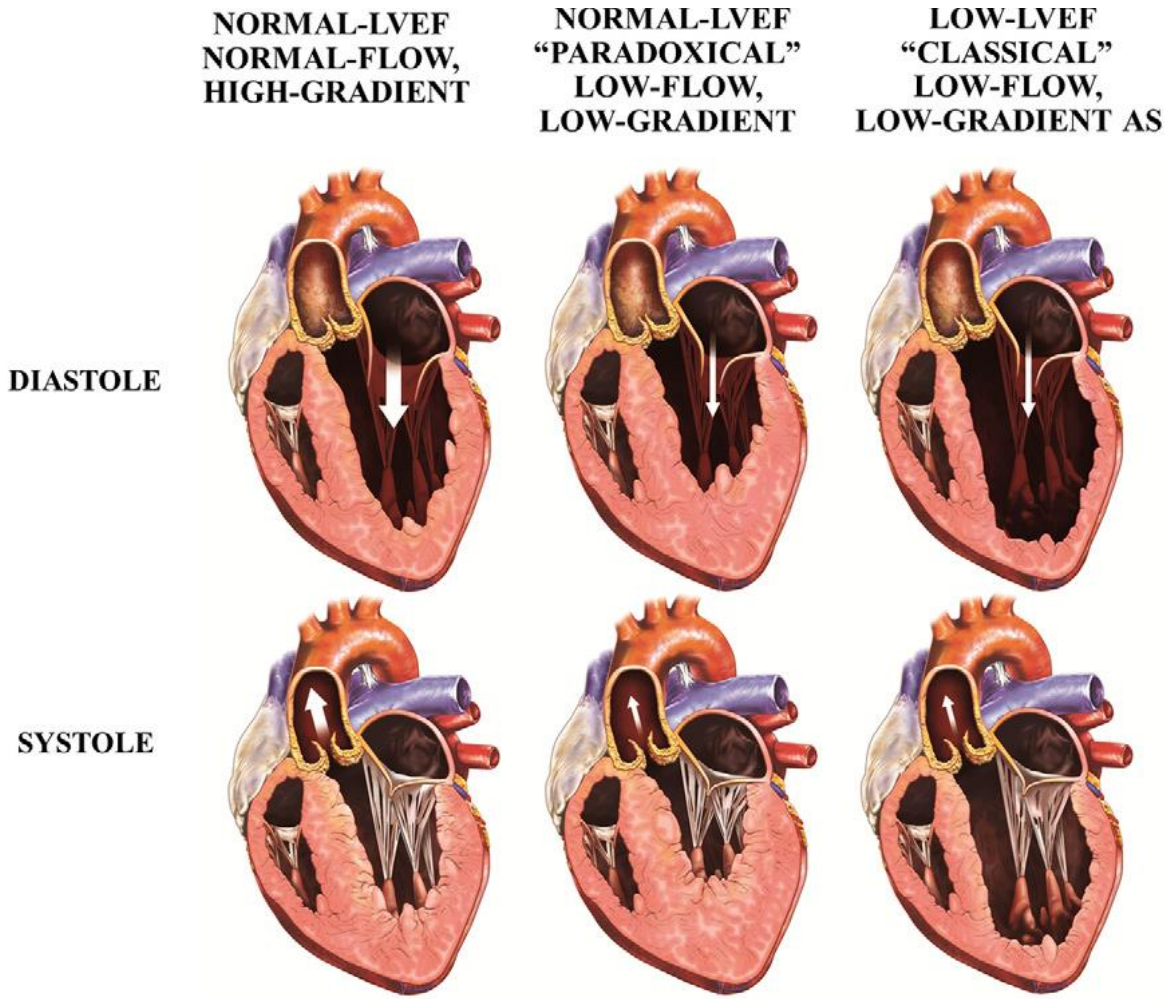

X5-1c
67Hz
20cm
2D
37%
C 49
P Low
HPen
Vel 438 cm/s
PG 77 mmHg
M3
CW
60%
WF 275Hz
1.5MHz
1.0
m/s
-1.0
-2.0
-3.0
-4.0
-5.0


X5-1c
80Hz
13cm
2D
46%
C 50
P Low
HGen
M3
1.8
3.6
70 bpm

Remember to assess from suprasternal notch



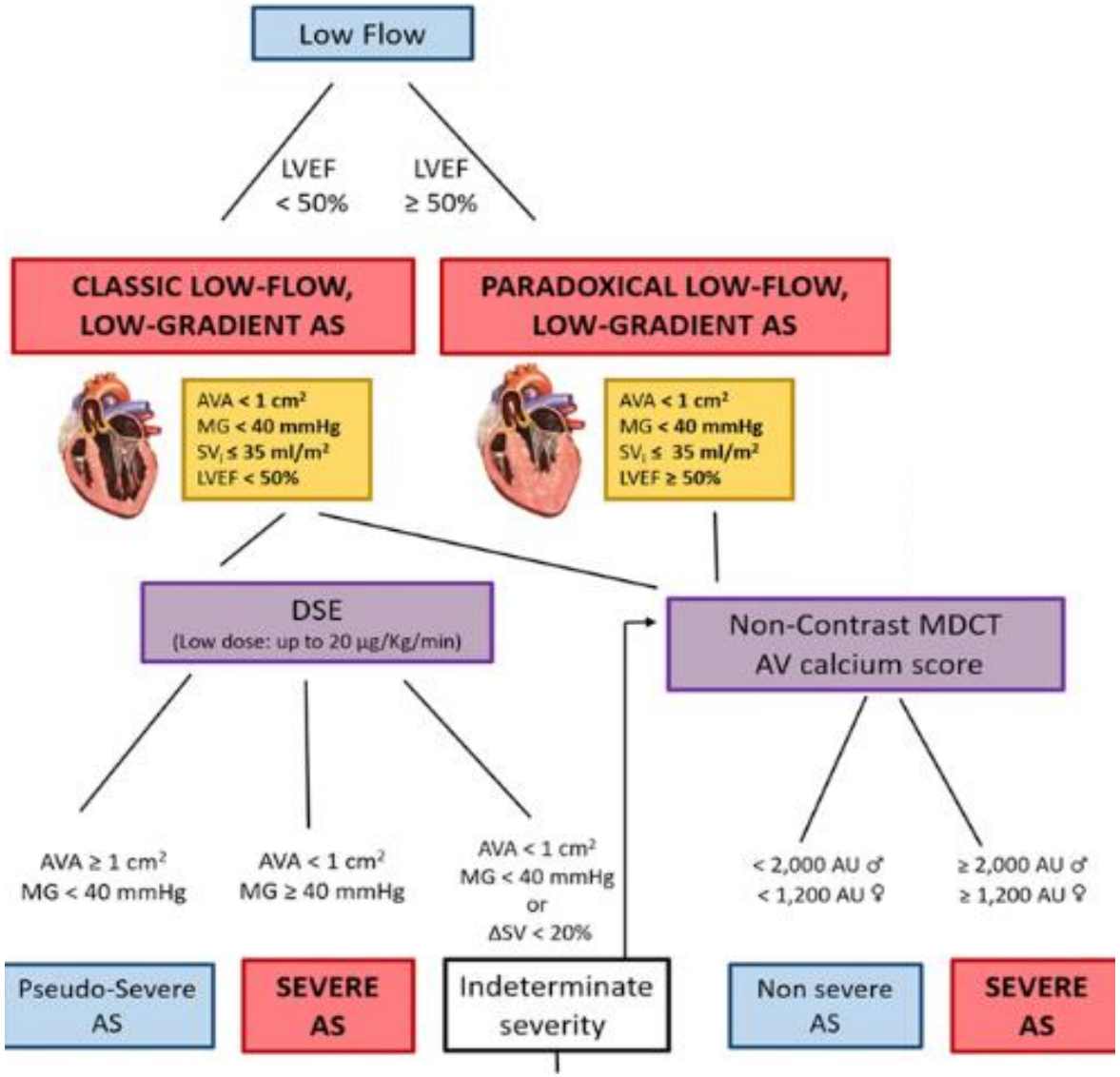
Quantification and assessment in low flow states

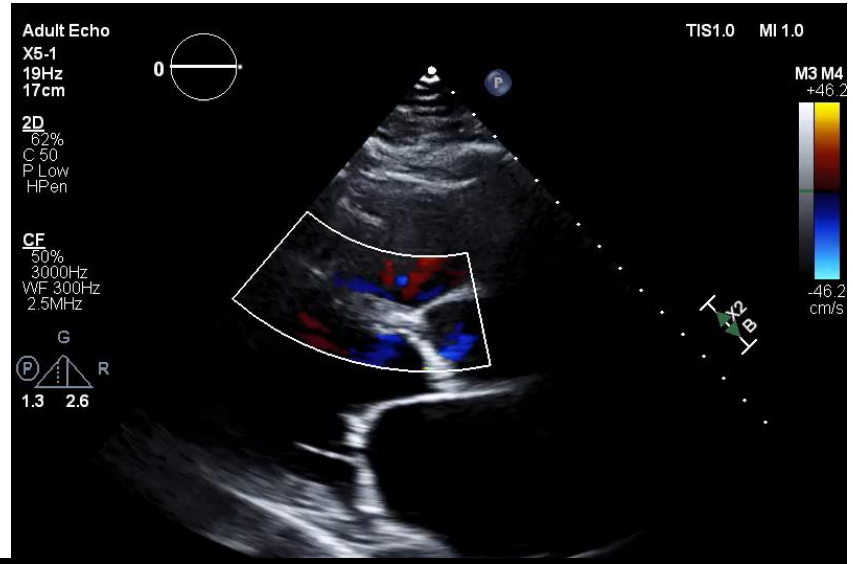


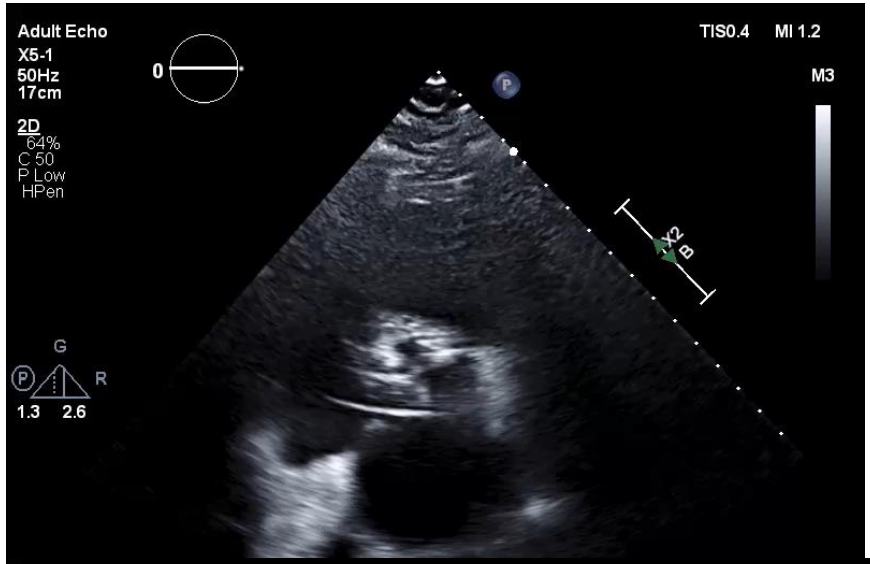
Critical to not assume normal valve function if peak velocity or gradients are not elevated.

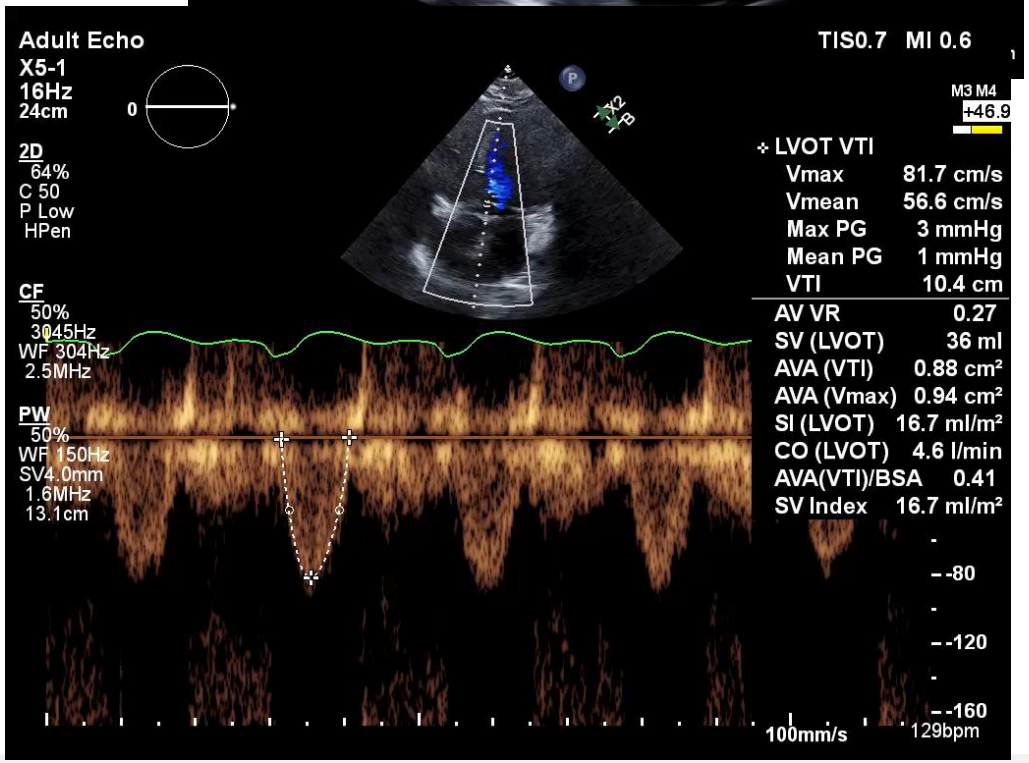
Particularly if valve appears visually calcified/abnormal.

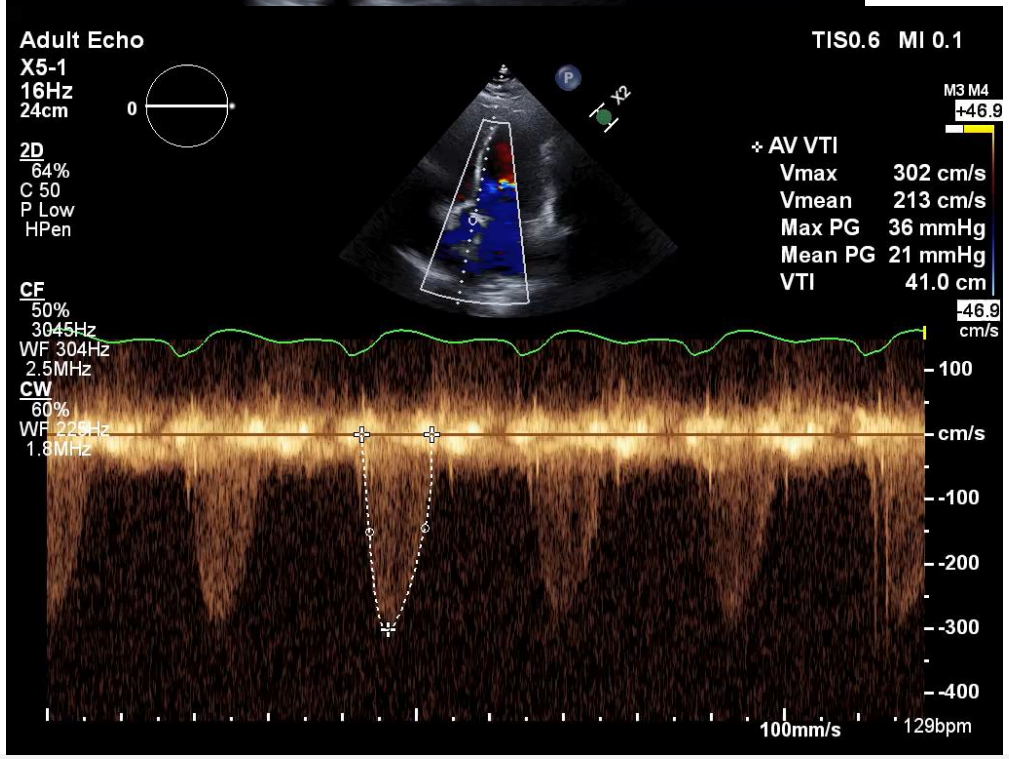
Evaluation of aortic valve anatomy and contractile reserve




Adult Echo
X5-1
19Hz
17cm
2D
62%
C 50
P Low
HPen
CF
50%
3000Hz
WF 300Hz
2.5MHz
TIS1.0 MI 1.0
M3 M4
+46.2
-46.2
cm/s
1.3 2.6


Adult Echo
X5-1
50Hz
17cm
2D
64%
C 50
P Low
HPen
TIS0.4 MI 1.2
M3
1.3 2.6

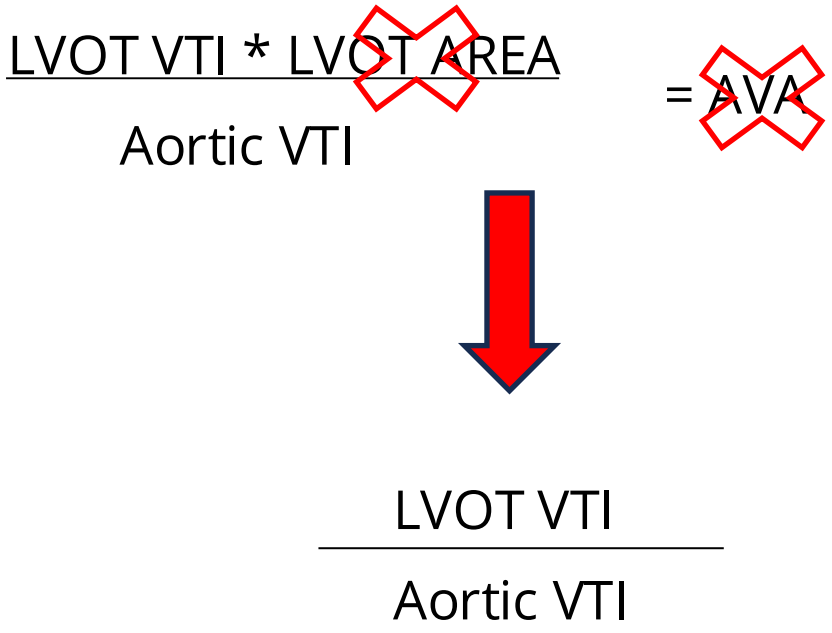

Adult Echo
X5-1
16Hz
24cm
2D
64%
C 50
P Low
HPen
CF
50%
3045Hz
WF 304Hz
2.5MHz
PW
50%
WF 150Hz
SV4.0mm
1.6MHz
13.1cm
TIS0.7 MI 0.6
M3 M4
+46.9
LVOT VTI
Vmax 81.7 cm/s
Vmean 56.6 cm/s
Max PG 3 mmHg
Mean PG 1 mmHg
VTI 10.4 cm
AV VR 0.27
SV (LVOT) 36 ml
AVA (VTI) 0.88 cm²
AVA (Vmax) 0.94 cm²
SI (LVOT) 16.7 ml/m²
CO (LVOT) 4.6 l/min
AVA(VTI)/BSA 0.41
SV Index 16.7 ml/m²
-80
-120
-160
100mm/s
129bpm


Adult Echo
X5-1
16Hz
24cm
2D
64%
C 50
P Low
HPen
CF
50%
3045Hz
WF 304Hz
2.5MHz
CW
60%
WF 225Hz
1.8MHz
TIS0.6 MI 0.1
M3 M4
+46.9
AV VTI
Vmax 302 cm/s
Vmean 213 cm/s
Max PG 36 mmHg
Mean PG 21 mmHg
VTI 41.0 cm
-46.9
cm/s
100
cm/s
-100
-200
-300
-400
100mm/s
129bpm

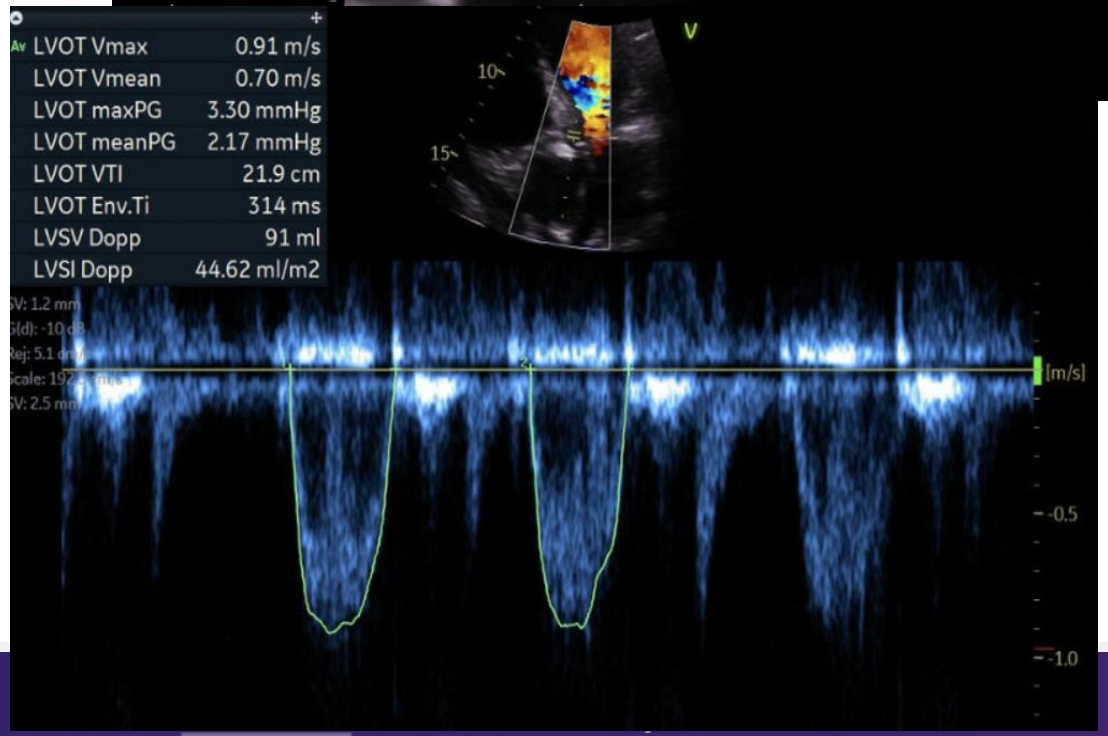
Doppler velocity (or VTI) index

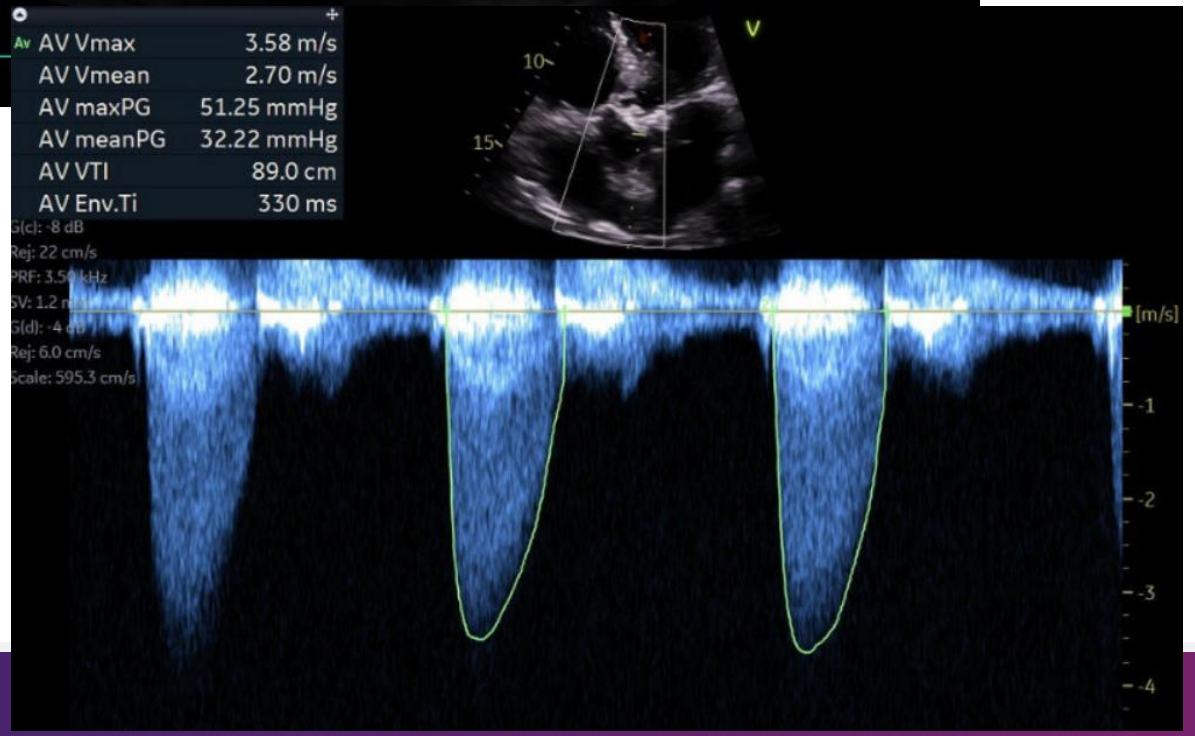
a.k.a. Why I don't use valve area

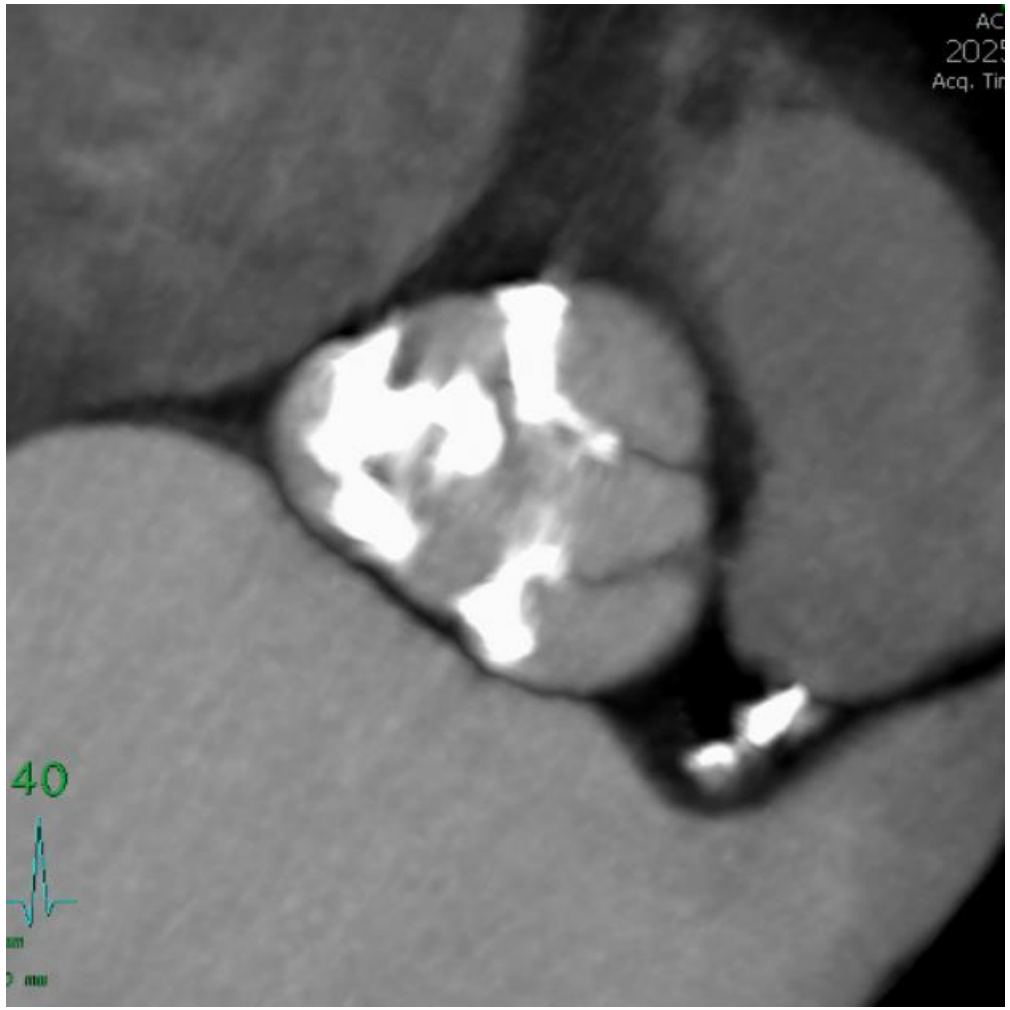
- Particularly valuable in differentiating high flow states from valve function.
- Removes LVOT diameter measurement, which is known to be highly variable.
- Easy to follow over time.



HD
Adapt
FPS: 44
f: 1.4 MHz/2.8 MHz
P: 0 dB
AG(t): 4 dB
Compr: 60 dB
D: 17.0 cm
HD
Adapt
FPS: 44
f: 1.4 MHz/2.8 MHz
P: 0 dB
AG(t): -3 dB
Compr: 60 dB
D: 19.0 cm
Soft


LVOT Vmax 0.91 m/s
LVOT Vmean 0.70 m/s
LVOT maxPG 3.30 mmHg
LVOT meanPG 2.17 mmHg
LVOT VTI 21.9 cm
LVOT Env.Ti 314 ms
LVSV Dopp 91 ml
LVSI Dopp 44.62 ml/m2
[m/s]
-0.5
-1.0


AV Vmax 3.58 m/s
AV Vmean 2.70 m/s
AV maxPG 51.25 mmHg
AV meanPG 32.22 mmHg
AV VTI 89.0 cm
AV Env.Ti 330 ms
Rej: 22 cm/s
Rej: 6.0 cm/s
Scale: 595.3 cm/s
[m/s]
-1
-2
-3
-4



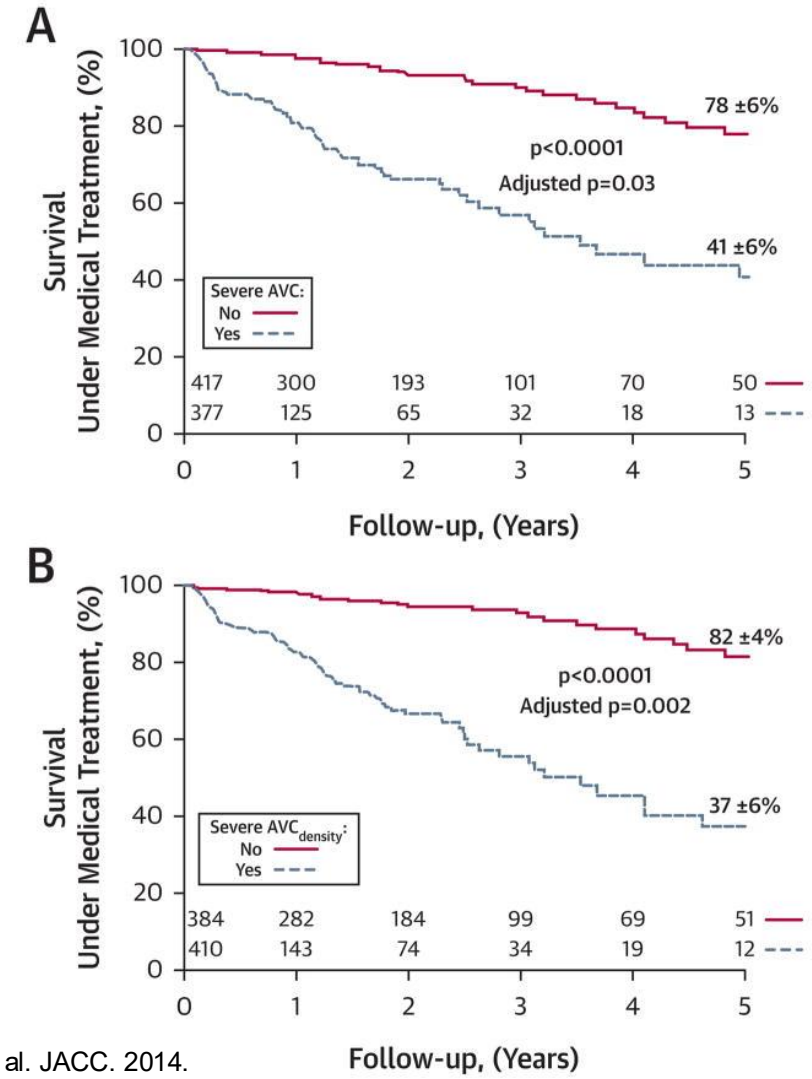
- Calcium score 2700 AU

- Calcium score 1350 AU

Aortic valve calcium scoring is a powerful adjunctive tool

Threshold of >2000 AU for men, >1200 AU for women

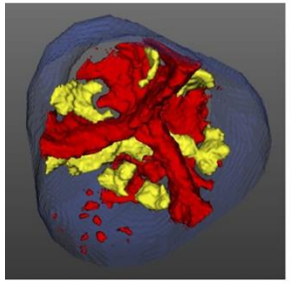
- Recommended in AHA/ACC and ESC guidelines for risk stratification in patients with calcific AS.
- Can be followed over time.
  - Should be performed on a non-contrasted scan.
  - Can be obtained at the time of TAVR CTA.
  - Well validated correlations to invasive hemodynamics.



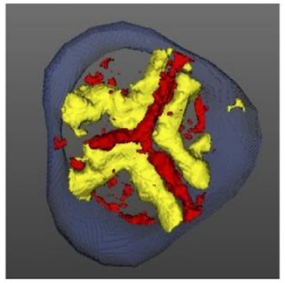
Clavel et al. JACC. 2014.

ADVANCED AORTIC VALVE CHARACTERIZATION

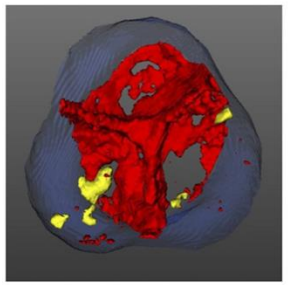
AORTIC VALVE PHENOTYPE



FIBRO-CALCIFIC



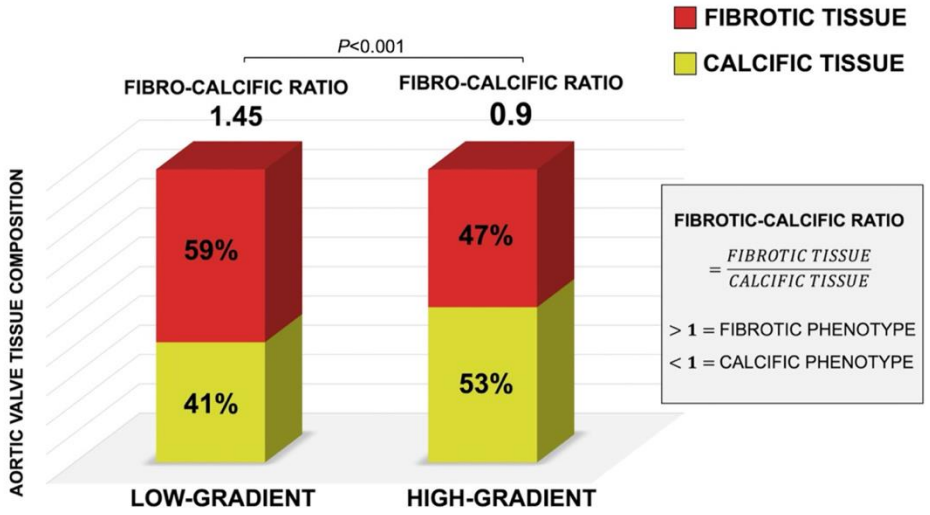
CALCIFIC



FIBROTIC

	♀	♂
FIBRO-CALCIFIC (mm^3/cm^2)	↓	↑
CALCIFIC (mm^3/cm^2)	↓	↑
FIBROTIC (mm^3/cm^2)	Similar	Similar
FIBRO-CALCIFIC RATIO	↑	↓

LOW-GRADIENT vs HIGH-GRADIENT



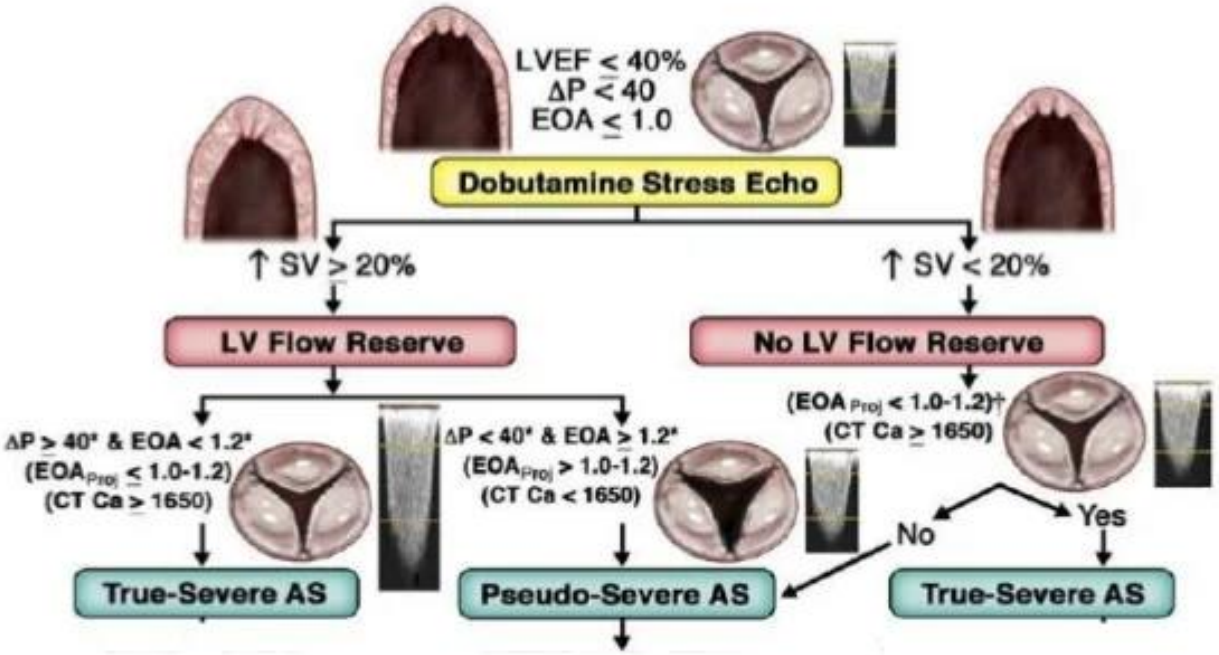
RACE/ETHNICITY

	♀	♂
FIBRO-CALCIFIC (mm^3/cm^2)	↑ in NH-White	Similar
CALCIFIC (mm^3/cm^2)	Similar	Similar
FIBROTIC (mm^3/cm^2)	↑ in NH-White ↓ in NH-Black	Similar
FIBRO-CALCIFIC RATIO	↑ in NH-White ↓ in NH-Black	Similar

What is the role of DSE?

- Several issues with DSE
  - Poor patient tolerance
  - Often limited by ectopy, hypotension
  - Dependent on sonographer acquisition skill, patient windows

Nobody seems to enjoy it.*



- ***Normal flow reserve:***

Medical followup every 6 months **vs** AVR (ESC class IIa)–based on the clinician's judgement

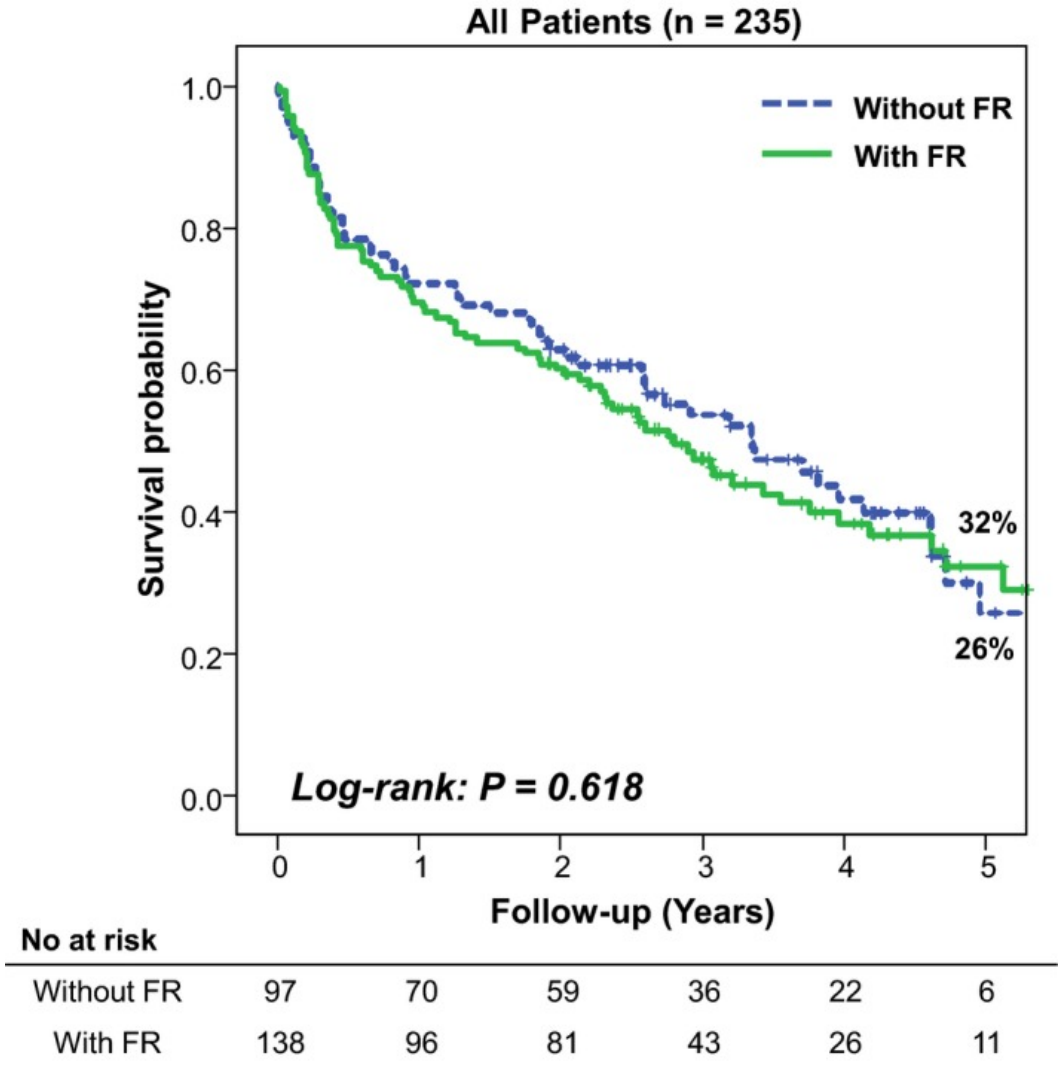
- ***Low flow reserve:***

1.IHD-OMT ± revascularisation
2.HTN- to be treated
3.Optimal heart failure management strategy
4.AVR (ESC class IIb)

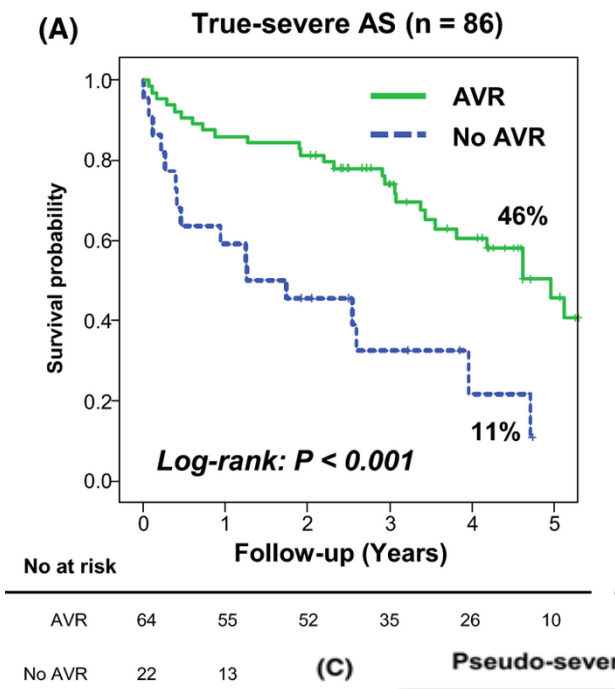
LOW FLOW LOW GRADIENT AS

*my opinion with no data

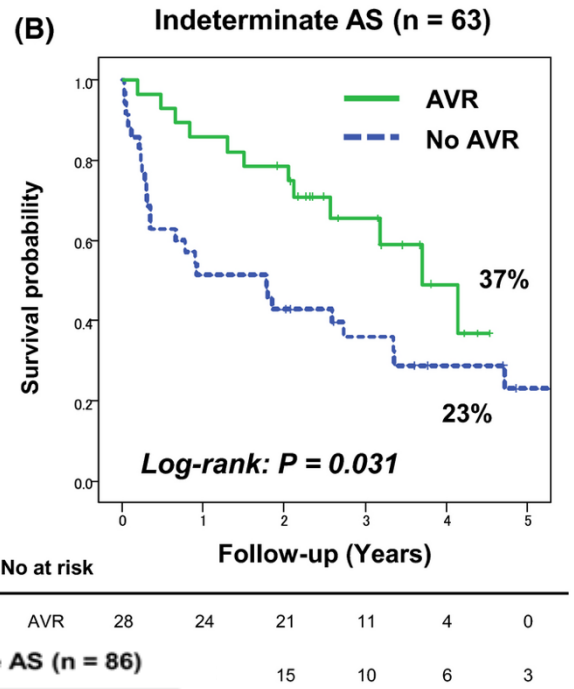
Flow reserve does not correlate to outcome



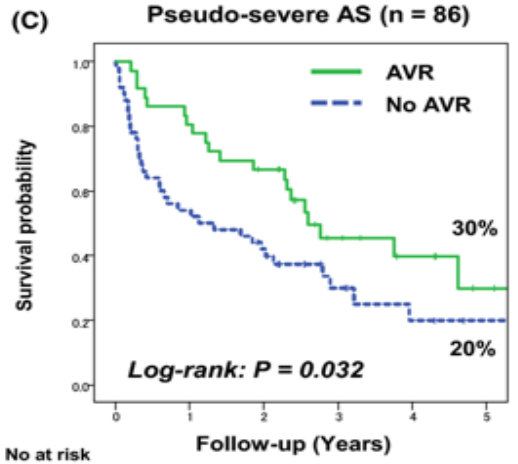
No at risk						
Without FR	97	70	59	36	22	6
With FR	138	96	81	43	26	11



No at risk						
AVR	64	55	52	35	26	10
No AVR	22	13			15	10



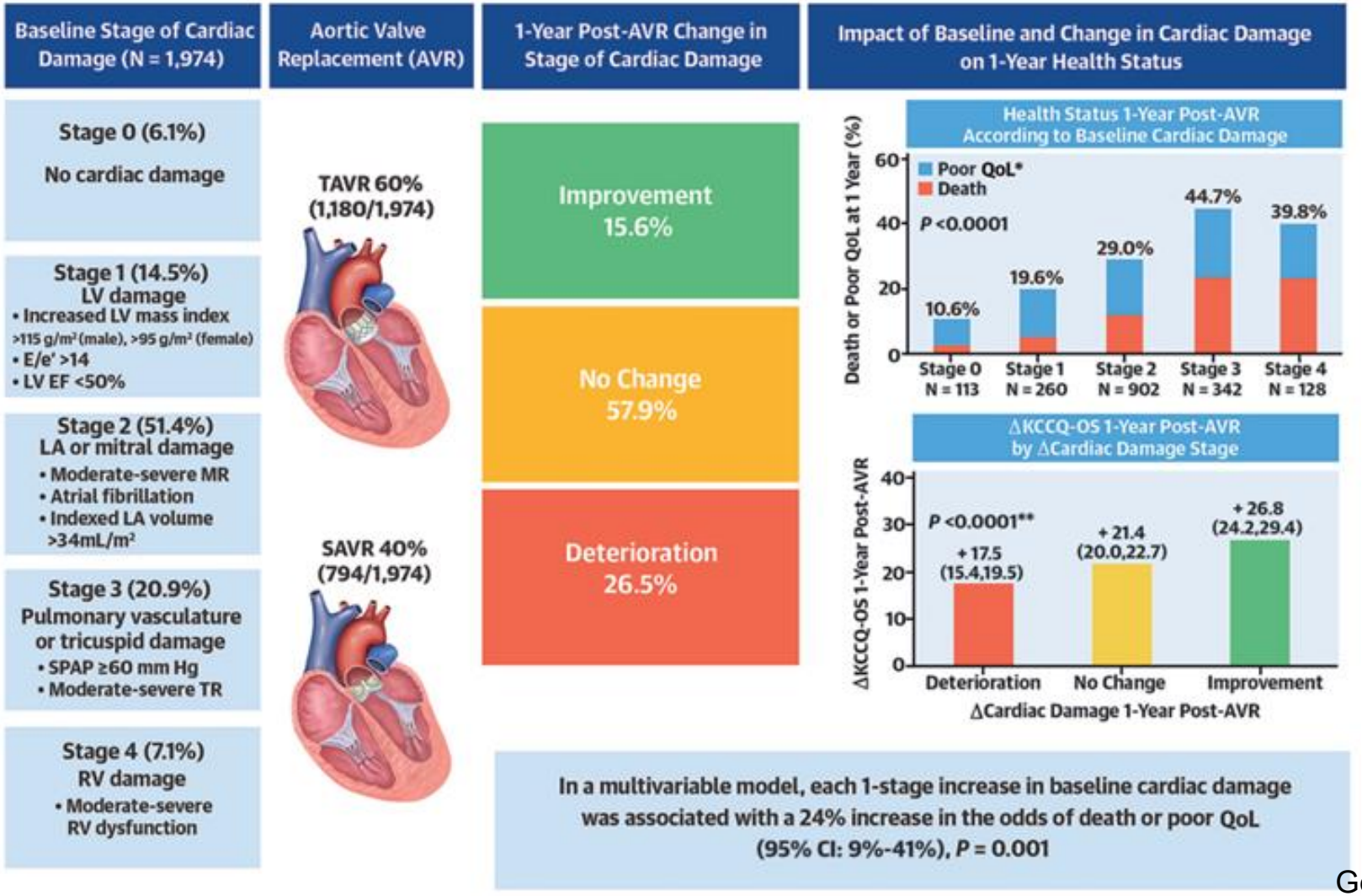
No at risk						
AVR	28	24	21	11	4	0
					6	3



No at risk						
AVR	36	29	23	10	6	2
No AVR	50	27	20	8	4	2

Sato K et al. JAHA. 2014.

Increasing cardiac damage predicts poor response to therapy



Genereux P et al. JACC 2023.

My questions

- Should we intervene in low EF patients with moderate disease?
    - TAVR UNLOAD trial
- What do we do with VERY low gradient patients?
- How can we better evaluate fibrotic leaflet changes in day to day practice?
- What do we do with discordant data?

Aortic regurgitation

Aortic regurgitation is often overlooked and undermeasured

Among asymptomatic AR patients with LV dysfunction...

> **25%** of them develop symptoms within 1 year[6]

Once symptoms arise:

Annual mortality rises to **25%**[1]

Death usually occurs within

4 years

of the onset of angina and within

2 years

of heart failure onset if there is no surgical intervention[7]

1 in 4 ssAR patients

were treated with SAVR within 1 year of diagnosis (26%)

1. Galusko V. Eur Heart J Clin Qual Care. 2022.
6. Bekeredjian R. Circ. 2005.
7. Aksoy O. Manual of CV Medicine. 2013.

Numerical quantification is challenging

Echocardiographic Criteria to Grade Aortic Regurgitation

		Mild	Moderate		Severe
			Mild to moderate	Moderate to severe	
PHT	ms	>500	500-200		< 200
Vena contracta	mm	< 3	3 – 6		> 6
Jet / LVOT	%	< 25	25-45	46-64	>65
Regurgitant Volume	ml	< 30	30-44	45-59	≥ 60
Regurgitant Fraction	%	< 30	30-39	40-49	≥ 50
EROA	cm^2	< 0.10	0.10-0.19	0.20-0.29	≥ 0.30
Aortic Backflow	cm	Early diastolic		Holodiastolic > 15	

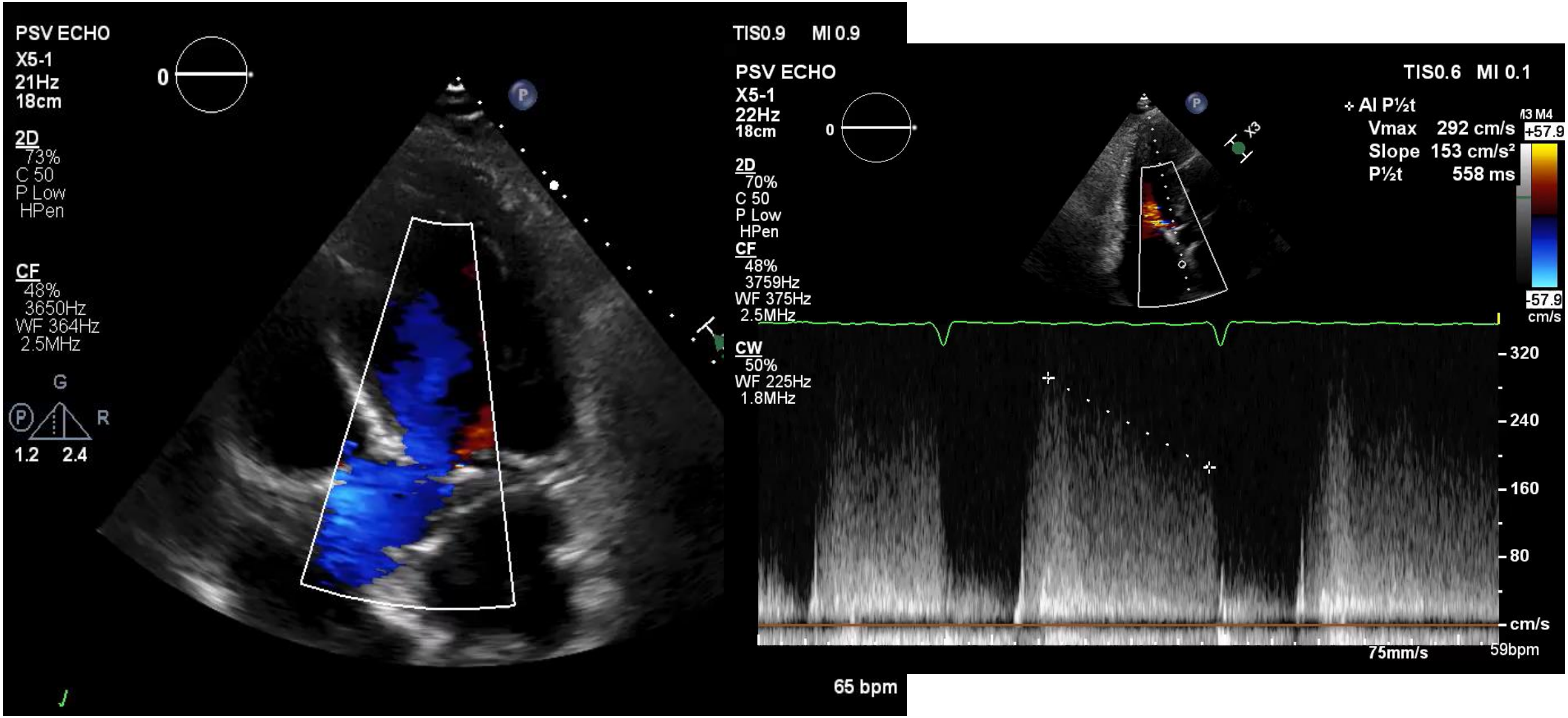
ACC/AHA Guidelines

LVEDD 50 mm

LVESD 25 mm

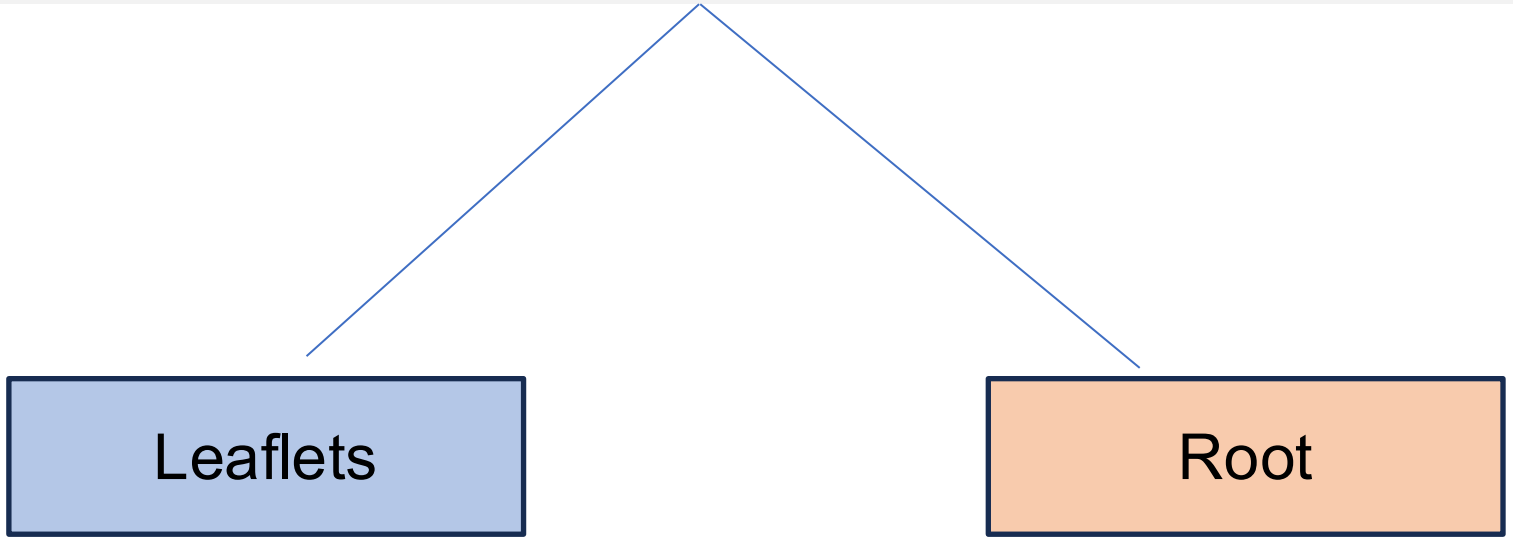
(some data suggests 20mm)

EF < 55%



NYHA Class II symptoms. LVEF 53%. Normal dimensions.

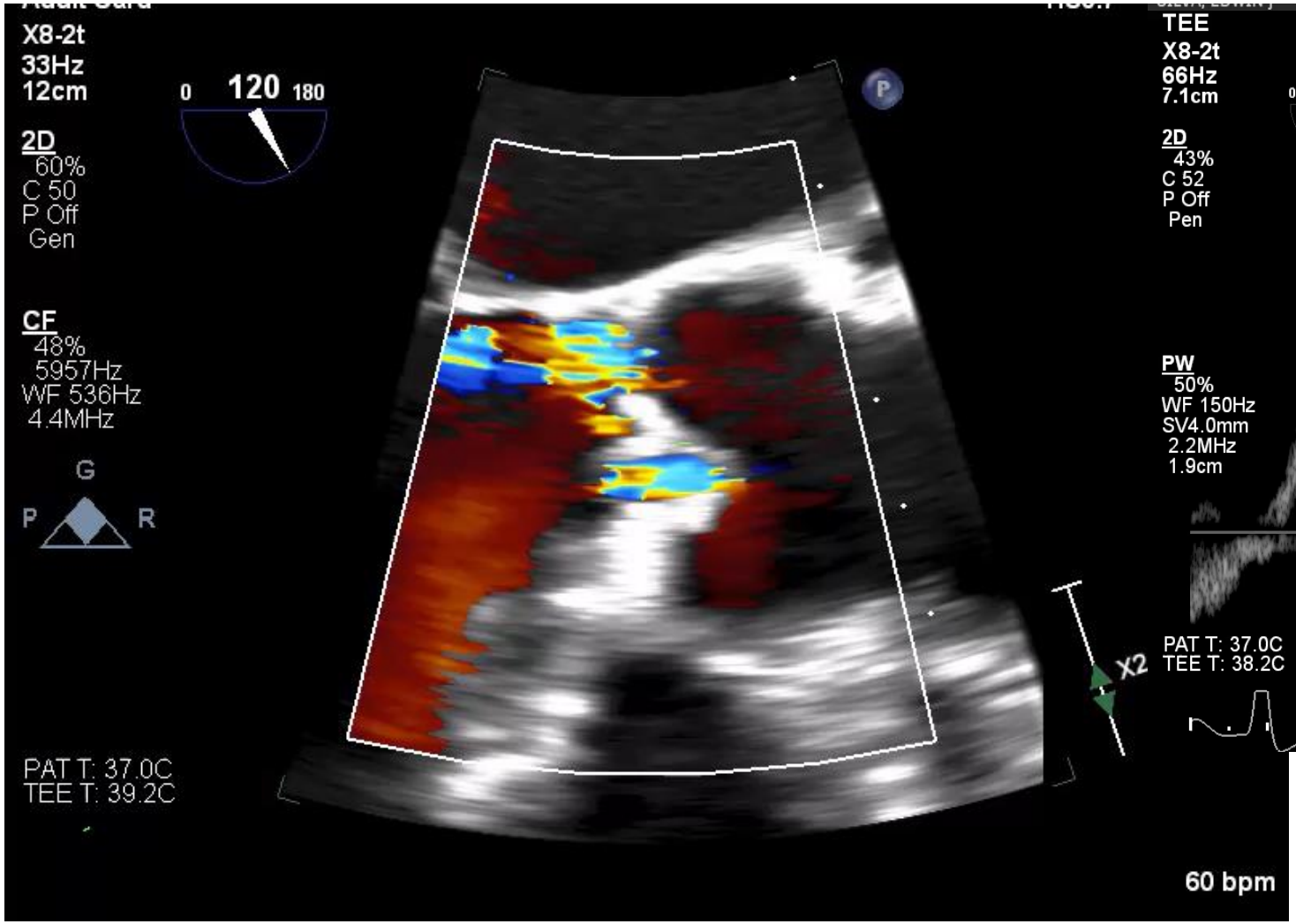
Thorough evaluation of etiology is needed

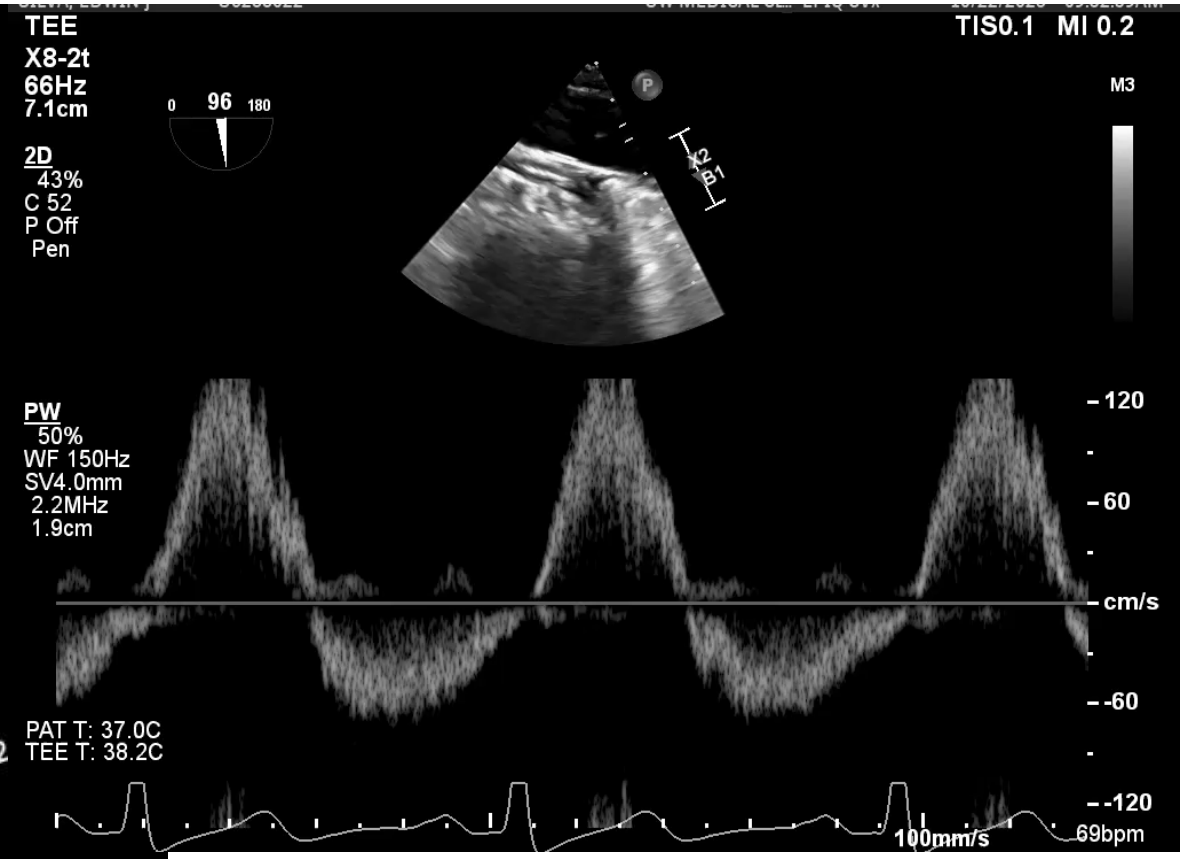


- Calcific disease
- Endocarditis
- Rheumatic
- Congenital (bicuspid)

- Root aneurysm
- Dissection
- Connective tissue disease
- Aortitis

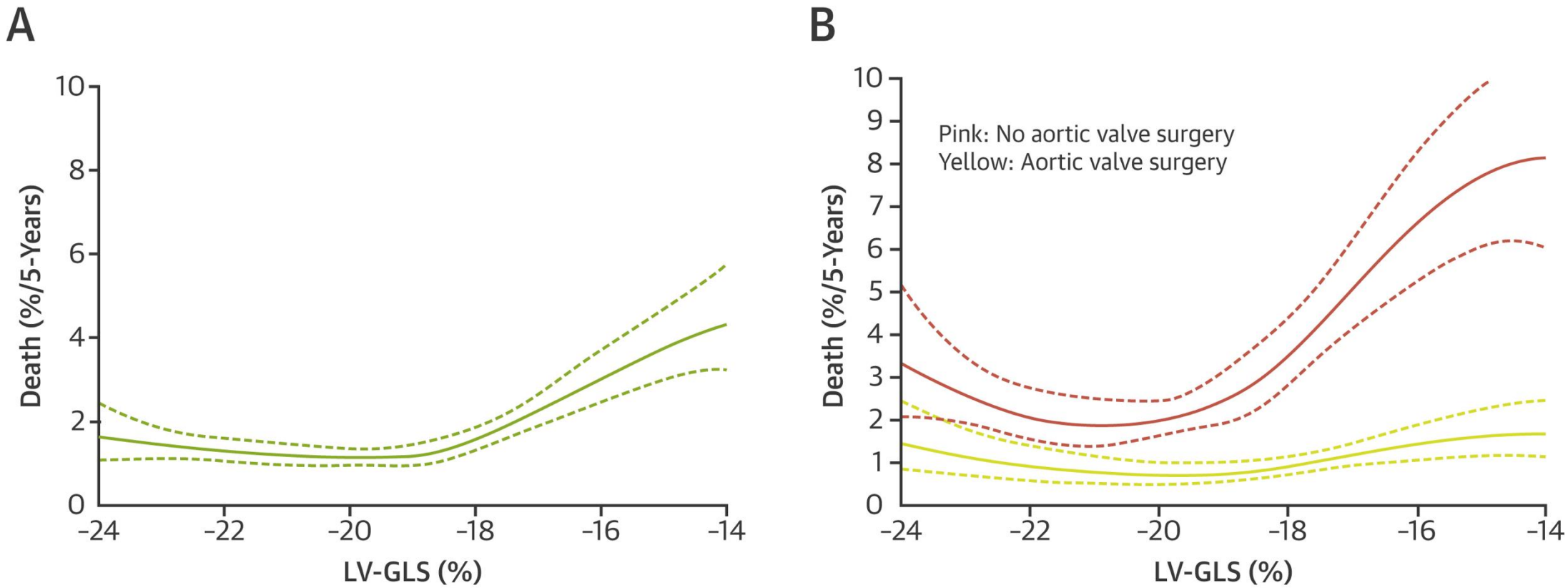
I maintain a low threshold for TEE in <u>non calcified</u> AR





Severe AR: holodiastolic flow reversal, EROA 0.32 cm^2

Abnormal GLS can help identify struggling ventricles



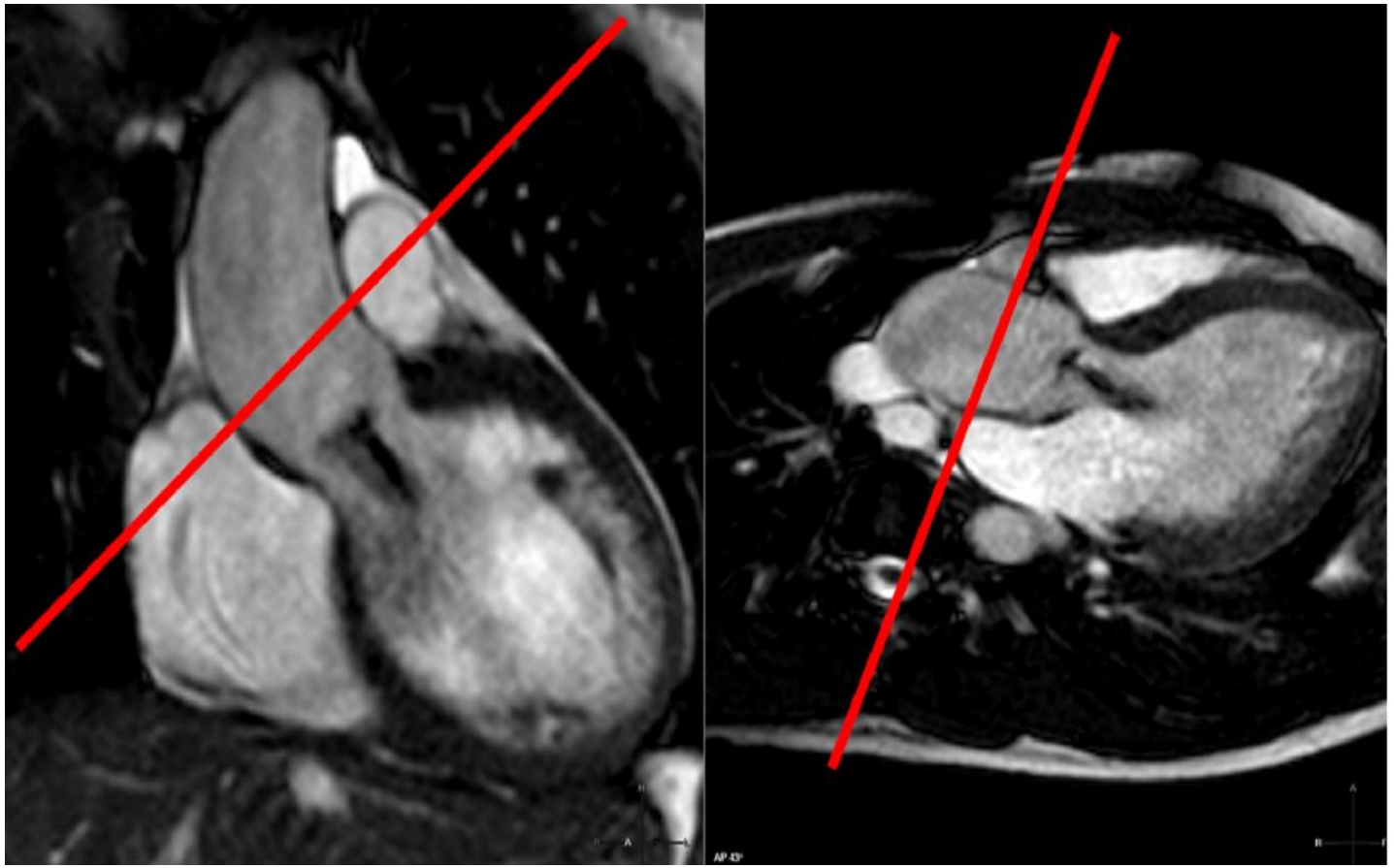
1,063 patients with moderate or greater aortic regurgitation and normal LV dimensions and function stratified by GLS.

Alashi A et al. JACC CV Imaging. 2018.

Cardiac MRI can help quantification in complex cases

Beware “garbage in, garbage out”

- Location of measurement – annulus, STJ, ascending aorta, etc.
  - Must be consistent
- Translational motion of the valve annulus
- Arrhythmia
- Dense LV trabeculations
- Sources of artifact
- Decreased validity with shunt lesions or multivalve disease

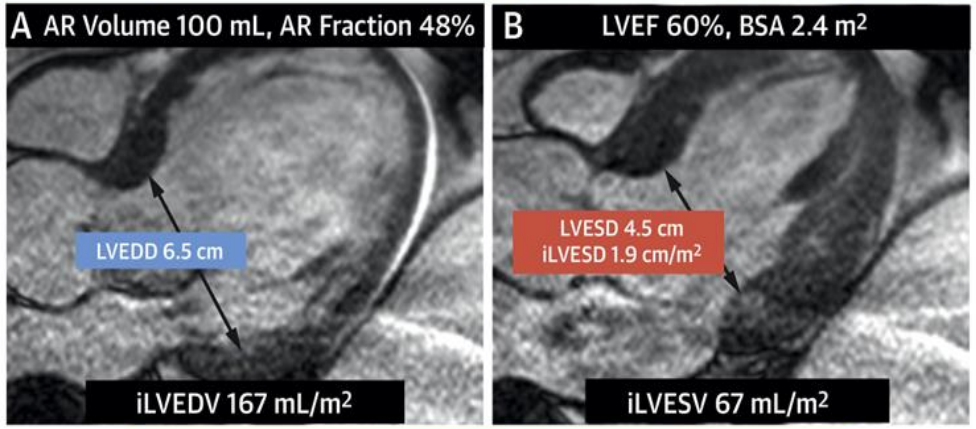


CENTRAL ILLUSTRATION: Parameters Associated With Adverse Events in Asymptomatic Aortic Regurgitation Patients

Asymptomatic Chronic AR Patients With Preserved LV Function

Quantitative CMR Findings to Predict Development of Symptoms, Decrease in Ejection Fraction, Surgery for Established LV Remodeling Thresholds, or Death Under Medical Management

Regurgitant Volume ≥47 mL Regurgitant Fraction ≥43%	Indexed LVESV ≥43 mL/m²	Indexed LVEDV ≥109 mL/m²	LVESD ≥4 cm Indexed LVESD ≥2 cm/m²



Use of LVESV Threshold Performed Favorably Compared to Diameter Measures, Which Can Introduce Higher Variability

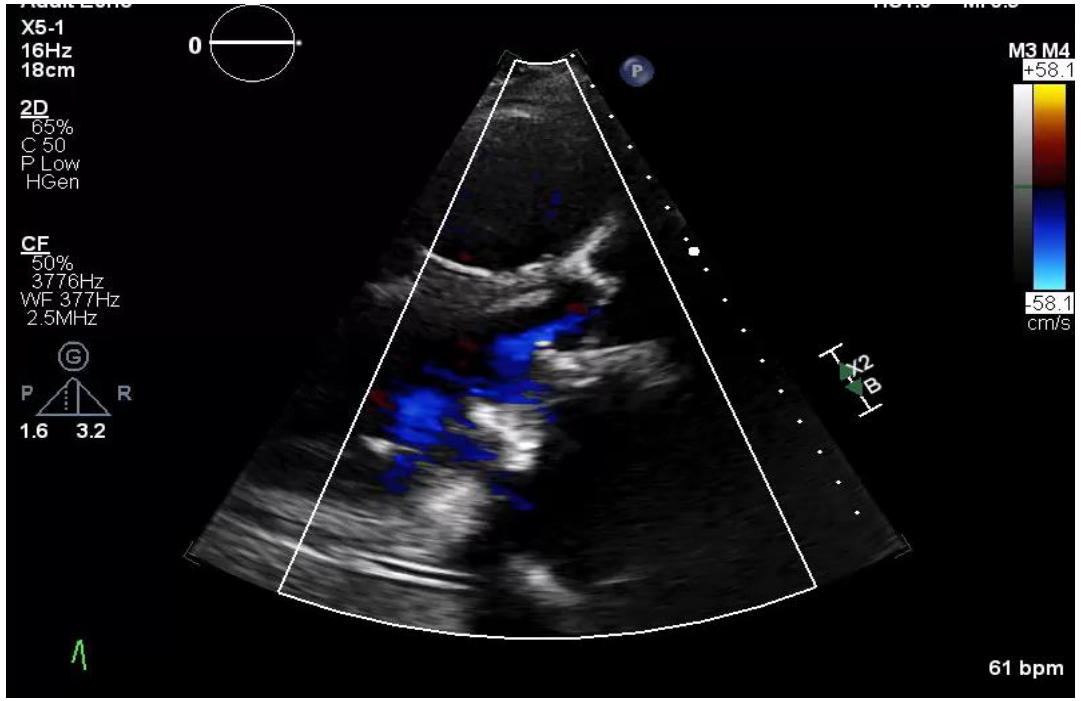
Malahfji M, et al. J Am Coll Cardiol. 2023;81(19):1885-1898.

This does not include many other studied parameters such as cMR based strain and myocardial fibrosis, which have been correlated with outcomes.

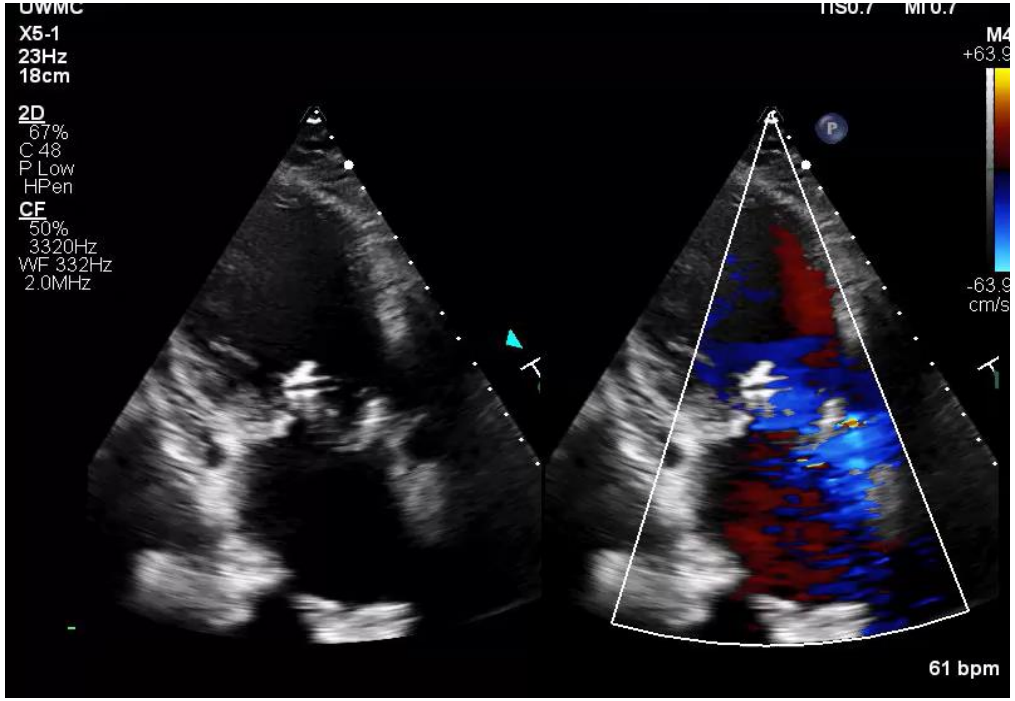
Mitral and Tricuspid Imaging

When is surface echo inadequate

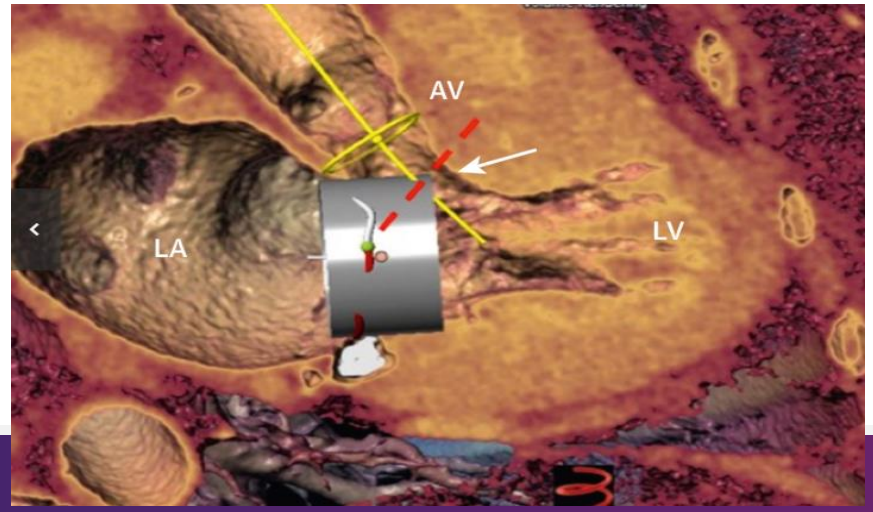
Uncertain Severity

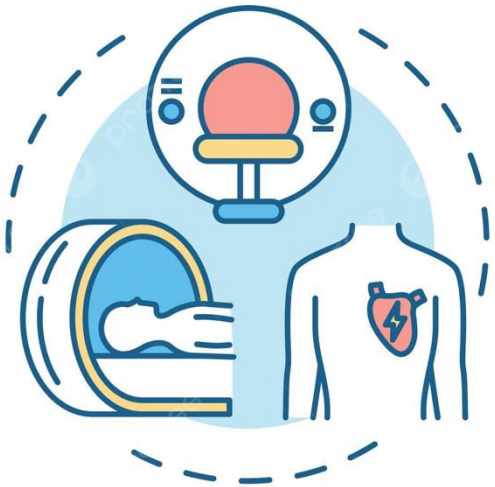


Uncertain Mechanism



Planning an intervention



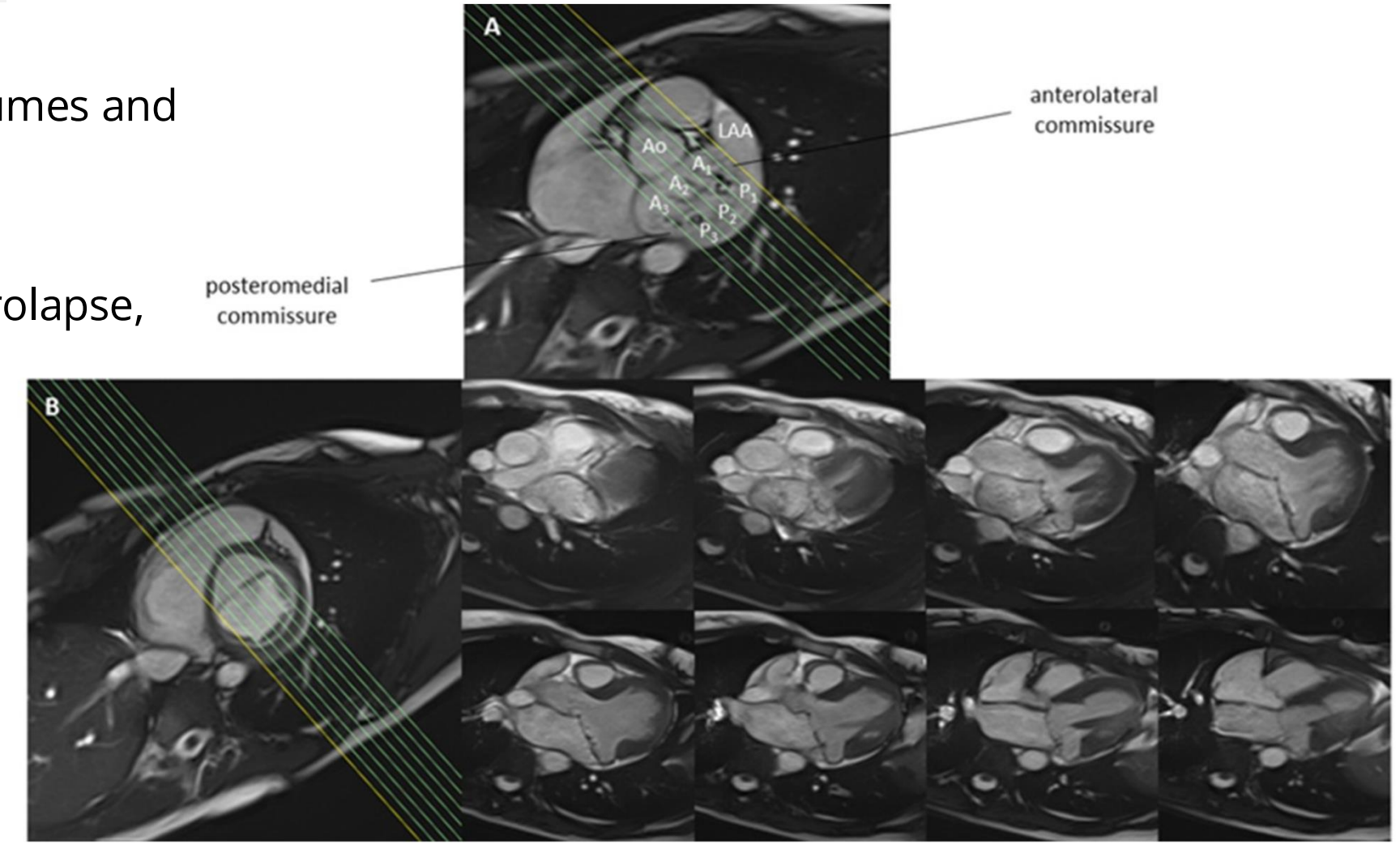

Cardiac CT

MRI

classroomclipart.com
http://classroomclipart.com

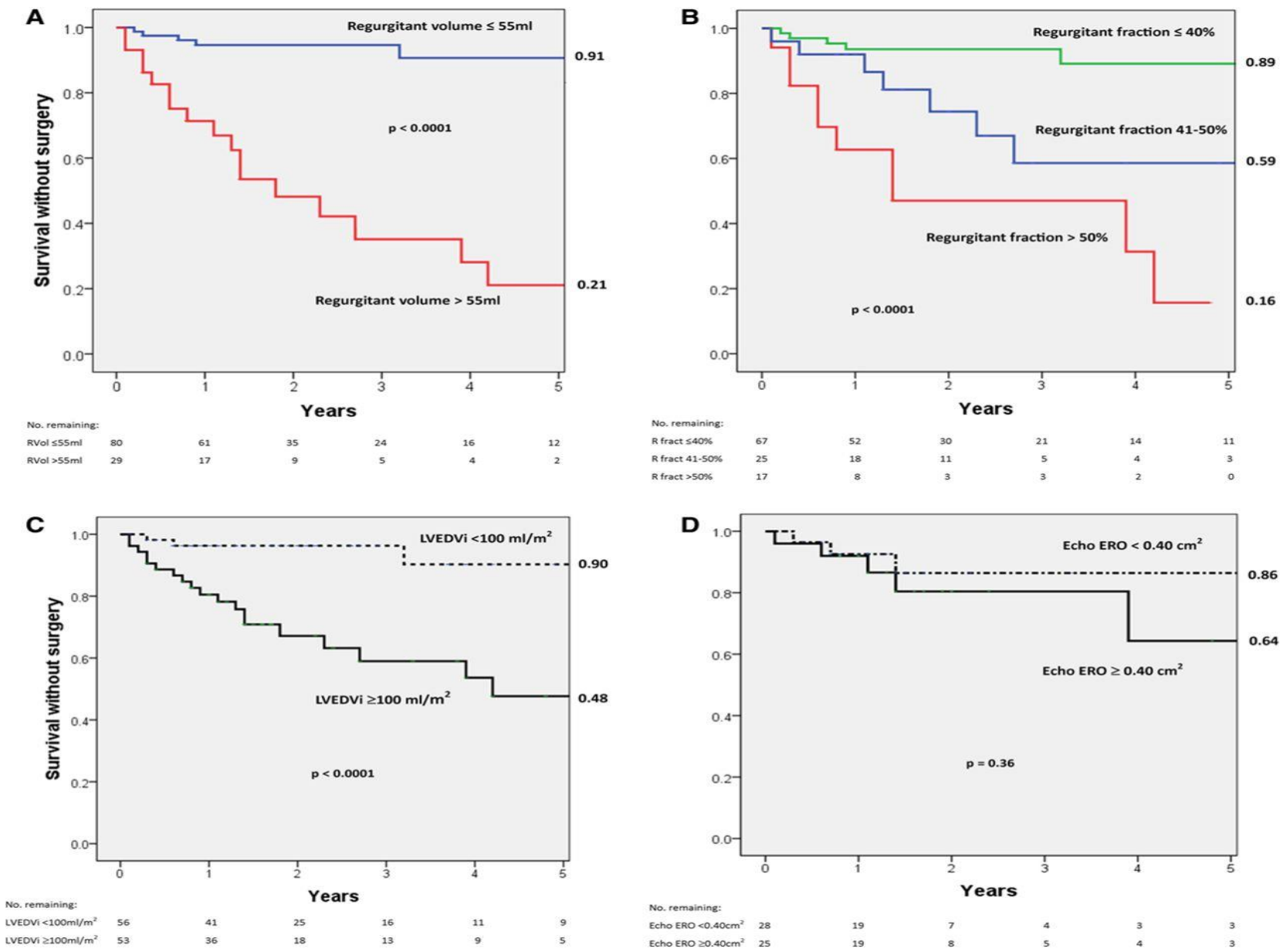
cMR for mitral regurgitation

- Calculation of LV volumes and function.
- Directionality of jet
- Leaflet coaptation, prolapse, flail.
- Left atrial size
- Quantification



Consistent predictor of poor clinical outcome

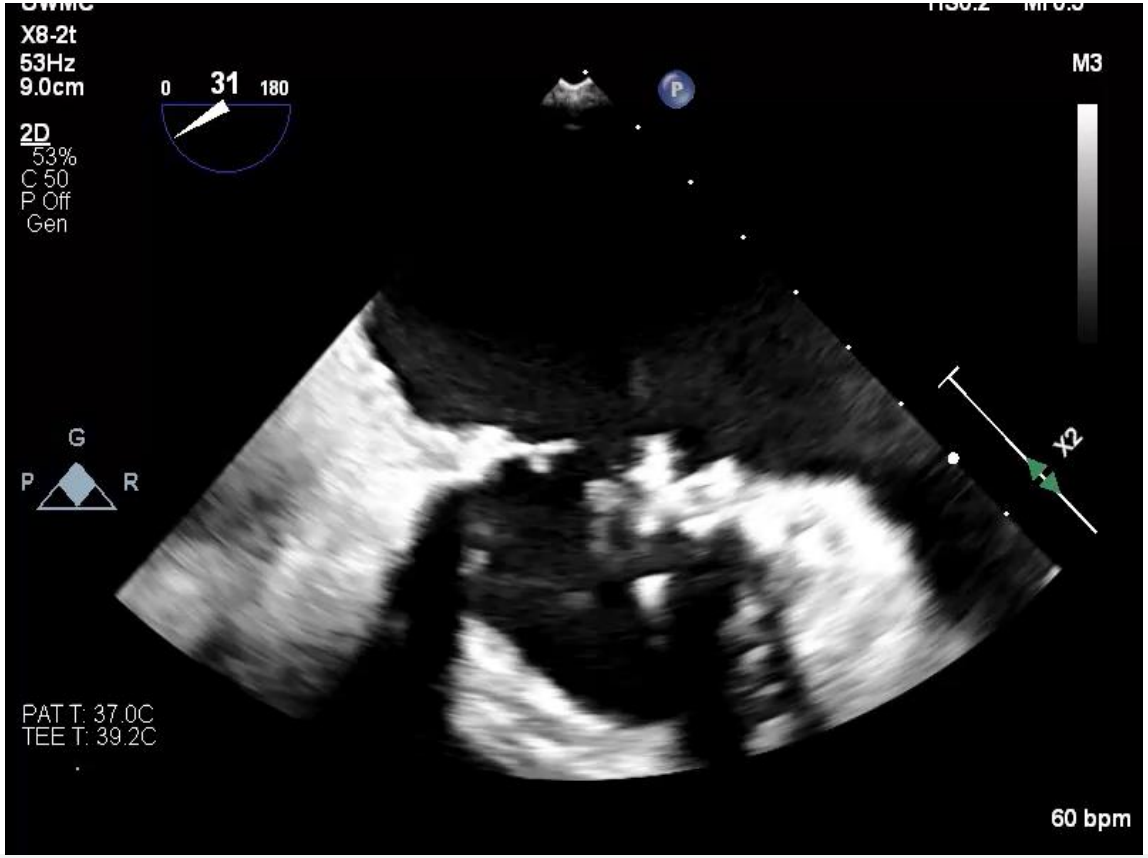
*I don't do a lot of cMR for mitral patients

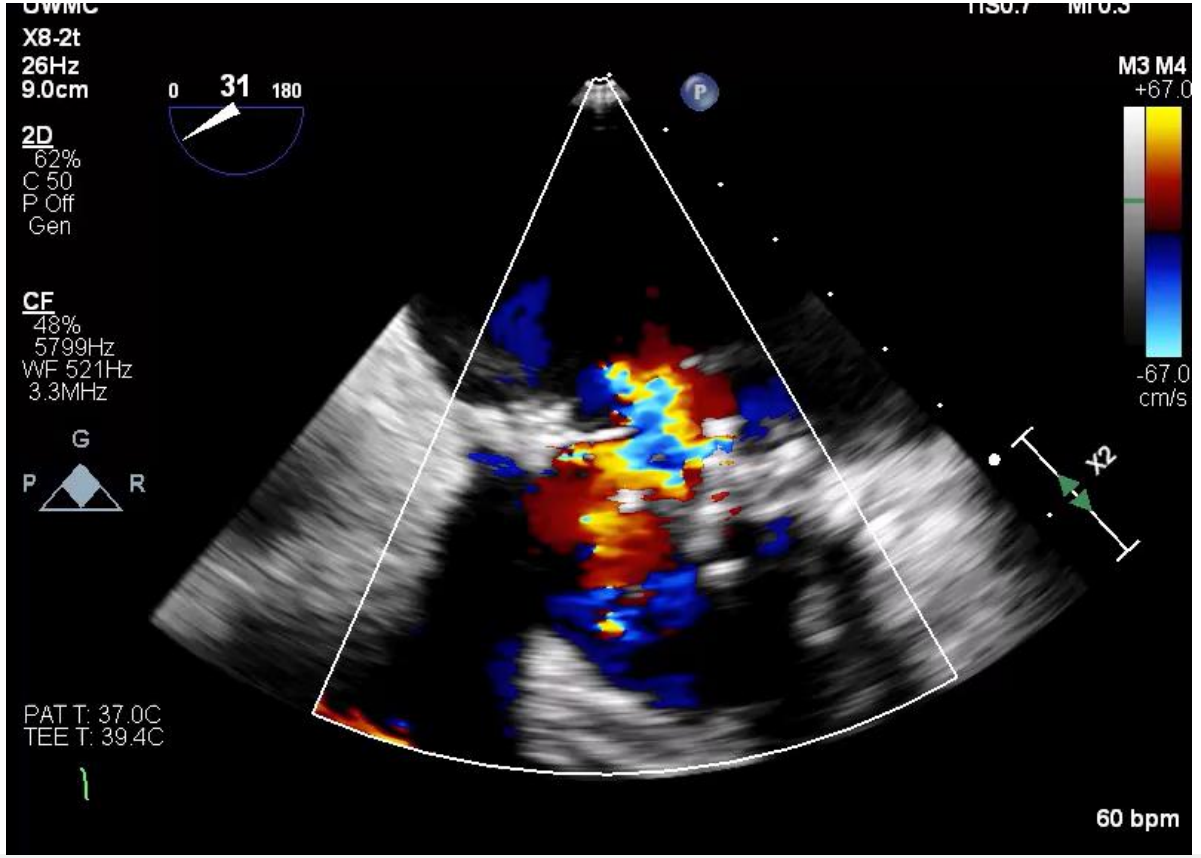


Myerson et al. Circ. 2016.

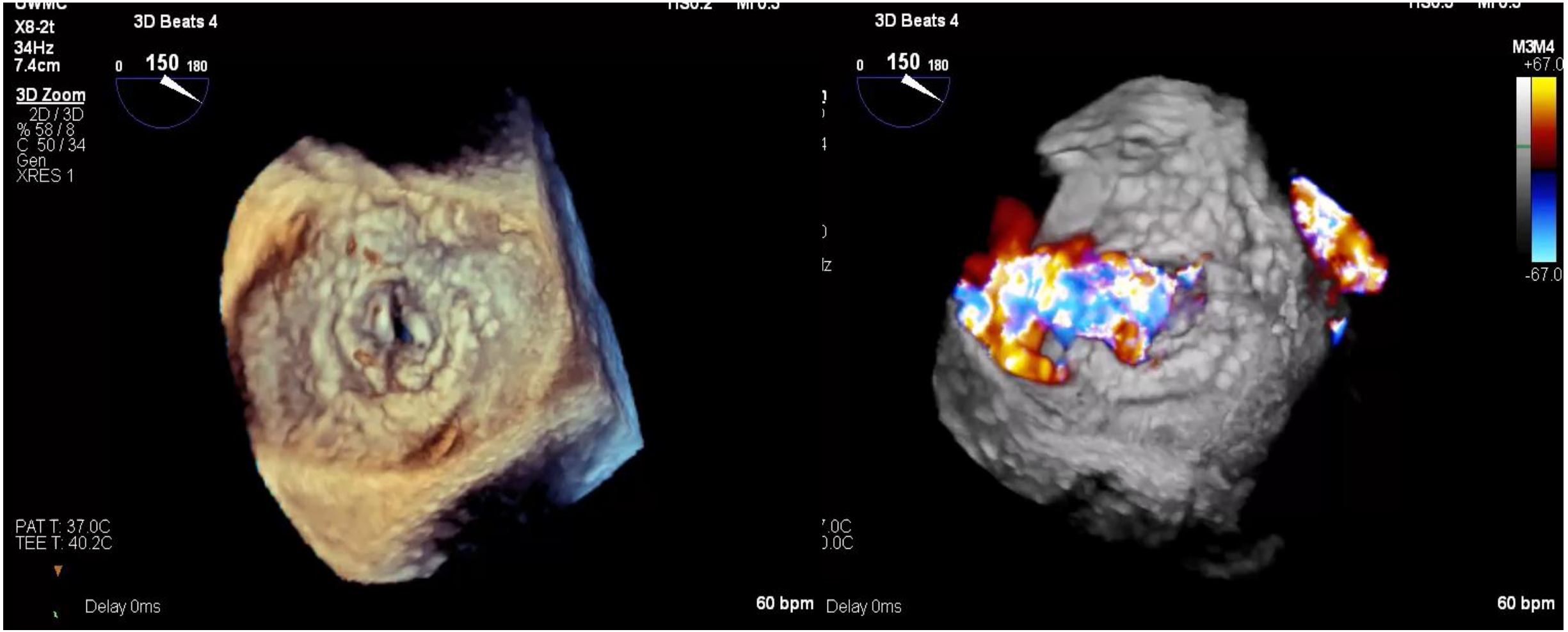
TEE is my “go to” for full delineation

- Complete information regarding mechanism and quantification
- Procedural planning and guidance



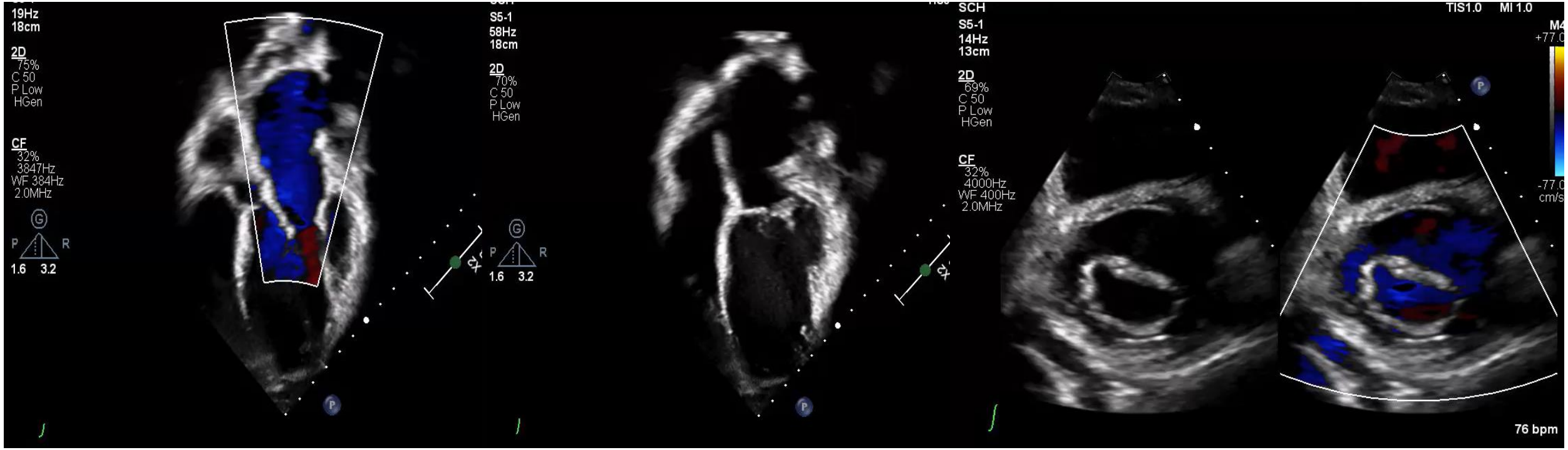


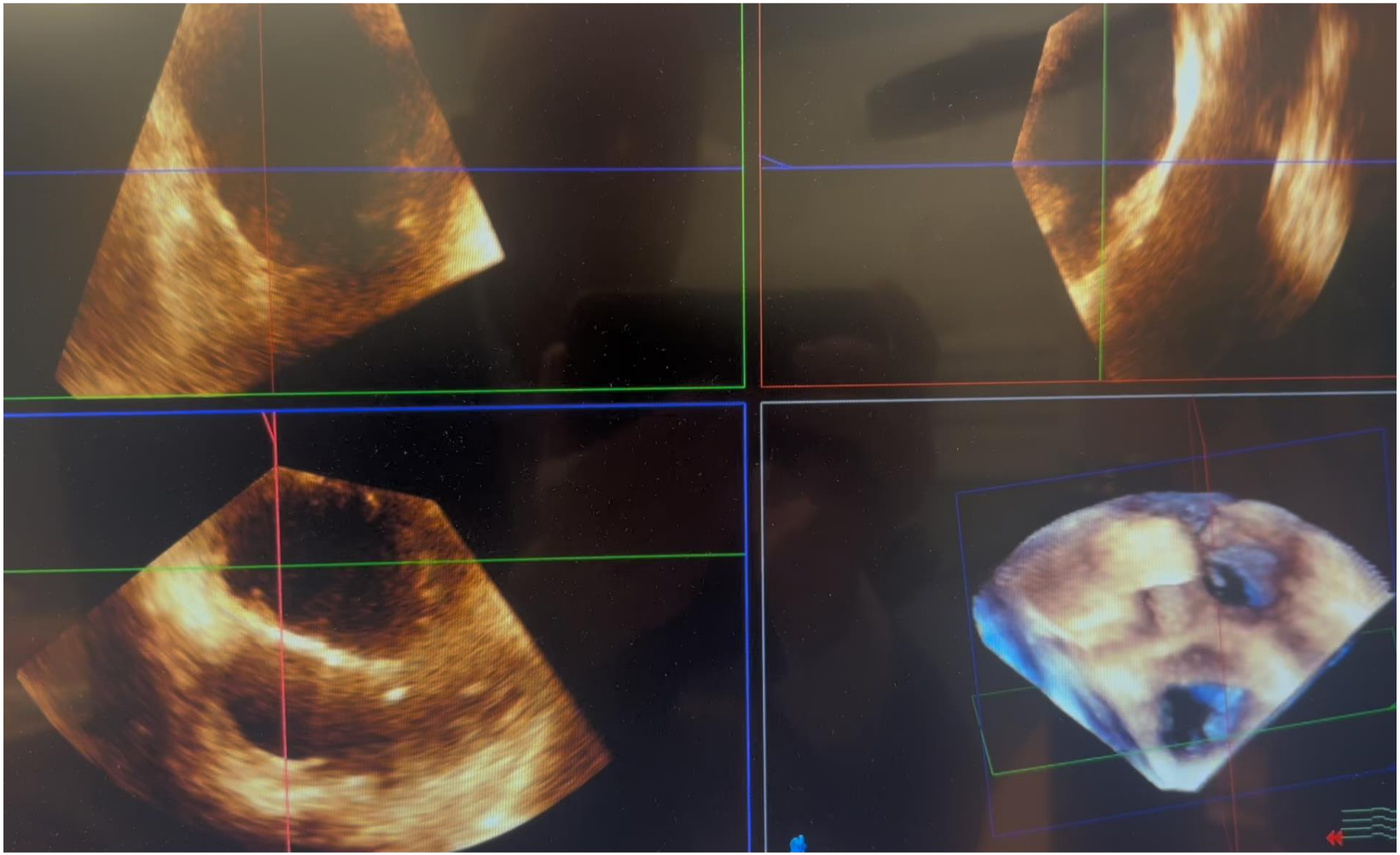
3D data set reconstruction



TEE 3D data can help understand mechanism

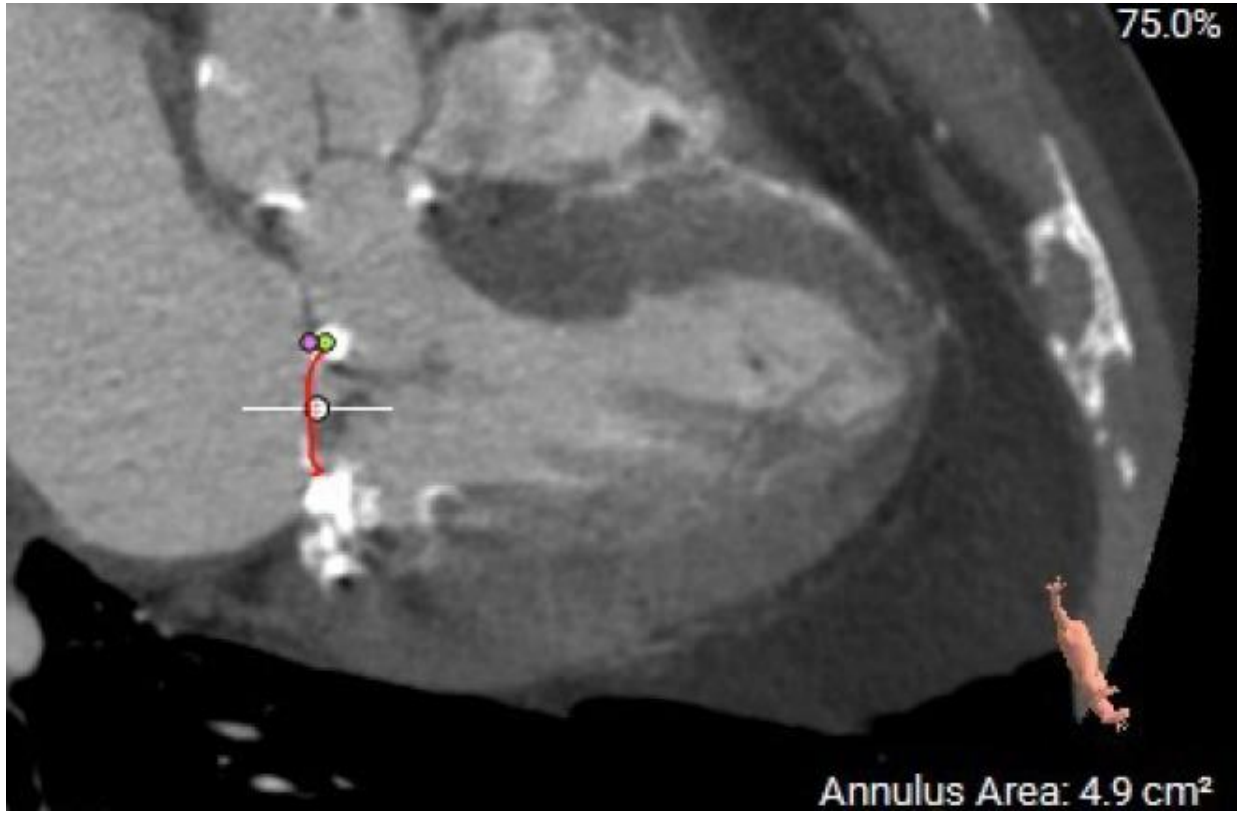
14 yo girl with heart transplant in infancy for LV noncompaction. Now with MR.

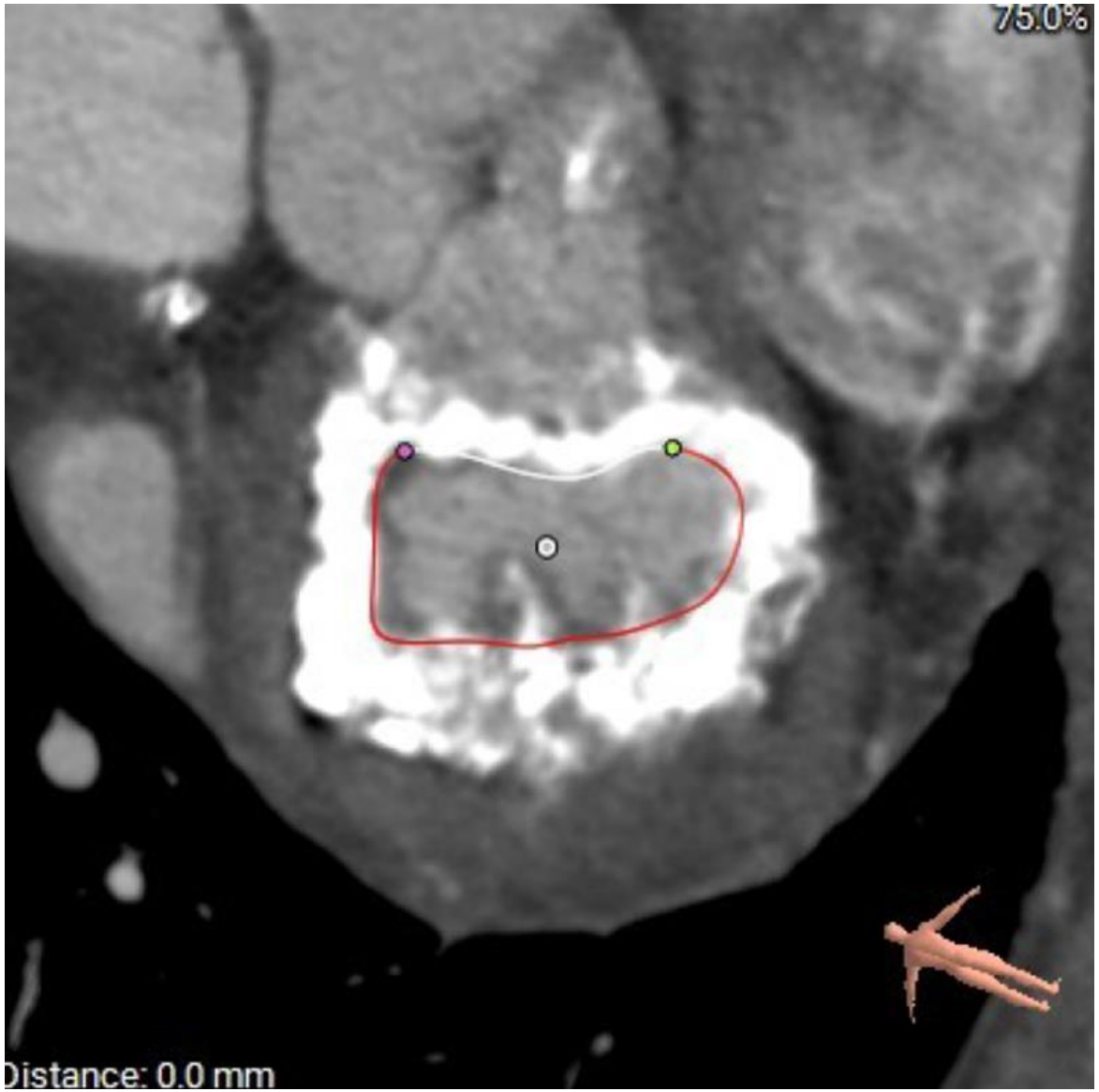




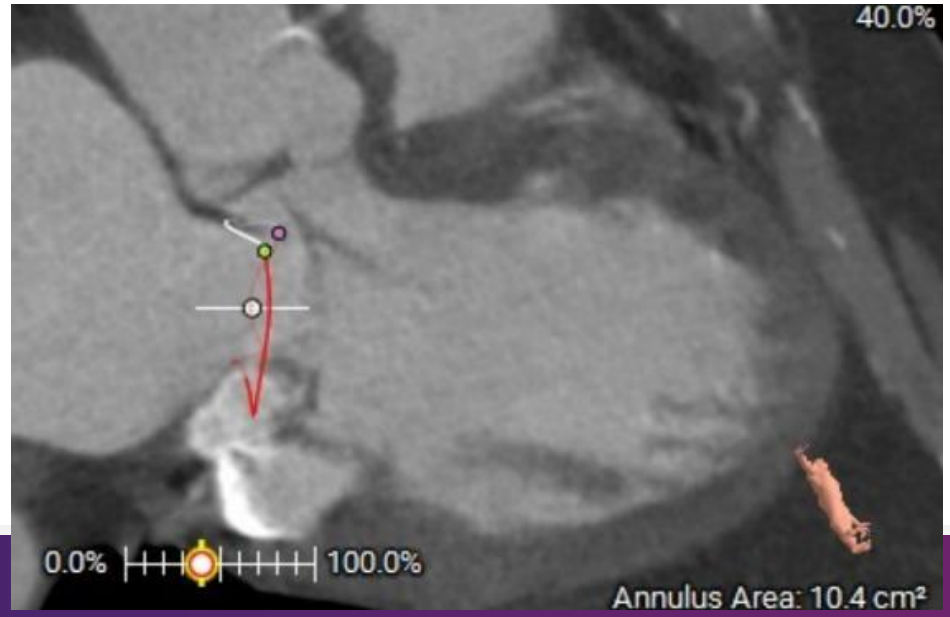
Cardiac gated CTA is critical for procedural planning

- Many third party software platforms for reconstruction exist
  - 3Mensio, TerraRecon, Circle, etc
- High fidelity measurements of annular sizing, LVOT area, etc.
- Calcium distribution
- Additional structures
  - Pseudoaneurysms
  - Large papillary muscles
  - Accessory cords



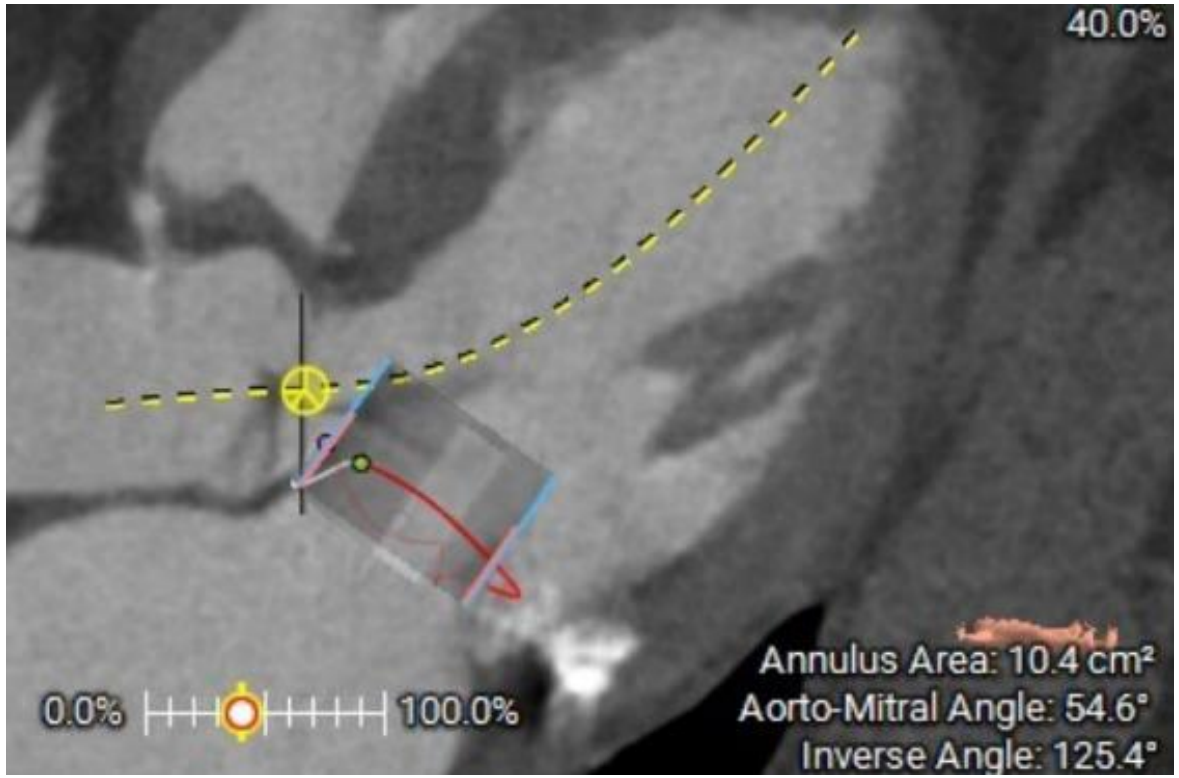

75.0%
Distance: 0.0 mm

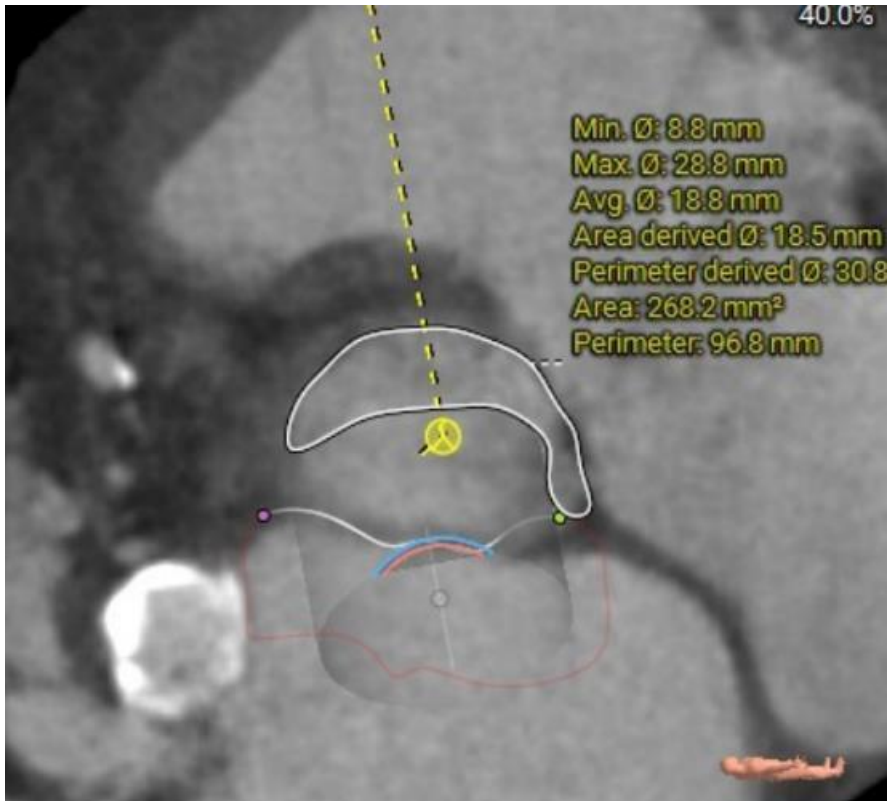
Distance: 0.0 mm


40.0%
0.0%
100.0%
Annulus Area: 10.4 cm²

Understanding “neoLVOT” risk for obstruction is primary concern in TMVR cases

Generally acceptable threshold <200 mm^2 at risk for hemodynamically significant LVOTO





Expanded categories of tricuspid regurgitation

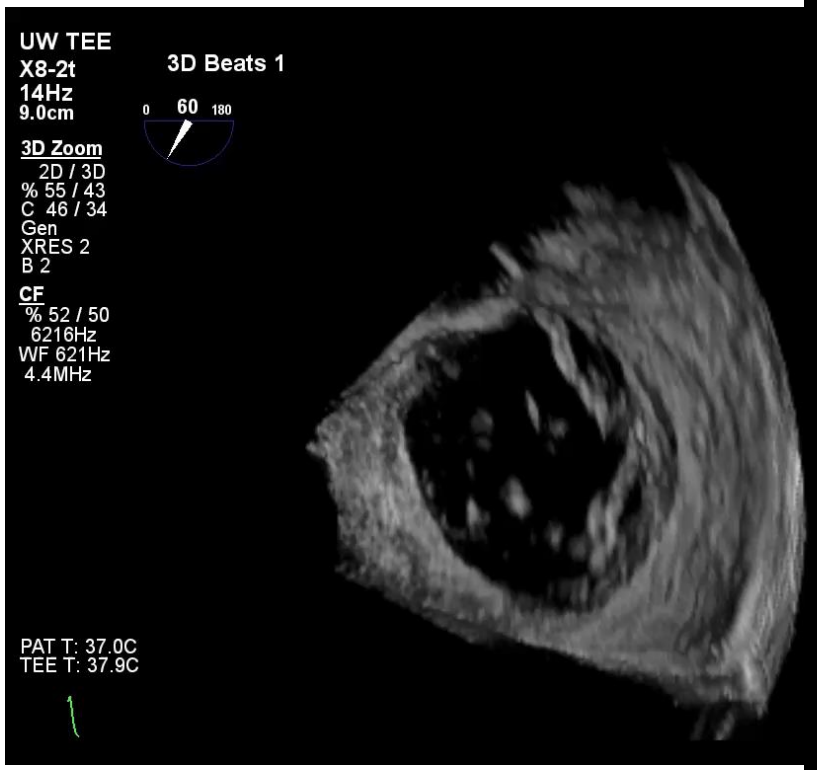
	Mild	Moderate	Severe	Massive*	Torrential*
QUALITATIVE					
TV morphology	Normal/abnormal	Normal/abnormal	Abnormal/flail/large coaptation defect		
Color Doppler of TR jet	Small, central	Intermediate	Very large central jet or eccentric wall impinging jet		
CW signal of TR jet	Faint/parabolic	Dense/parabolic	Dense/triangular with early peaking	Peak TR velocity <2 m/s	–
SEMIQUANTITATIVE					
VC width (mm)§	<3	3–6.9	7–13.9	14–20	>21
PISA radius (mm)	≤5	6–9	>9	–	–
Hepatic vein flow	Systolic dominance	Systolic blunting	Systolic flow reversal		
Tricuspid inflow	Normal	Normal	E wave dominant (≥1 cm/s)		
QUANTITATIVE					
EROA (mm^2) by PISA	<20	20–39	40–59	60–79	≥80
EROA (mm^2) by quantitative Doppler	–	–	75–94	95–114	≥115
EROA (mm^2) by 3D	–	–	75–94	95–114	≥115
R Vol (ml) by PISA	<30	30–44	45–59	60–74	≥75

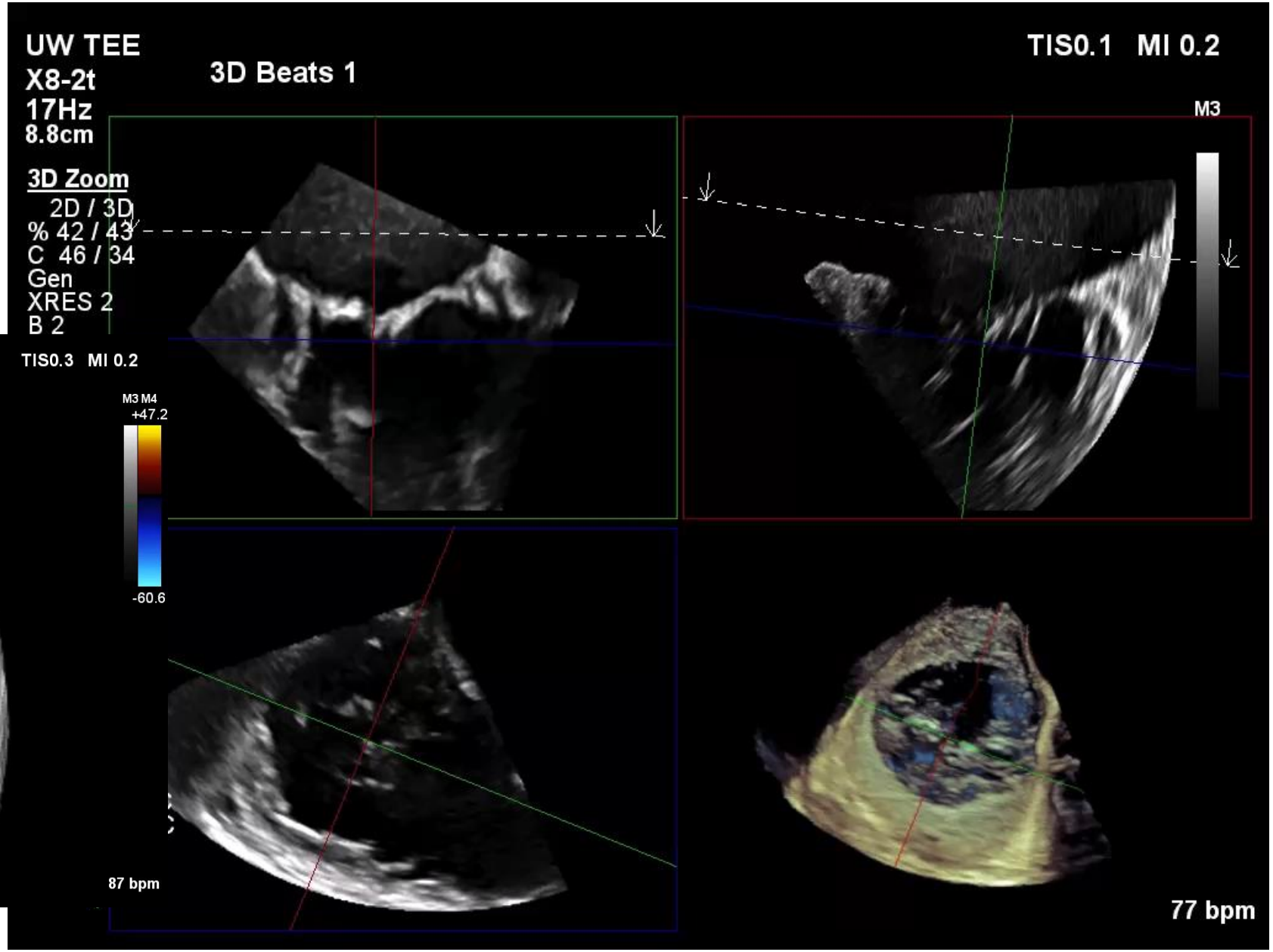
TV, tricuspid valve; TR, tricuspid regurgitation; CW, continuous wave; VC, vena contracta; PISA, proximal isovelocity surface area; EROA, effective regurgitant orifice area; R Vol, regurgitant volume.

**further data required.*

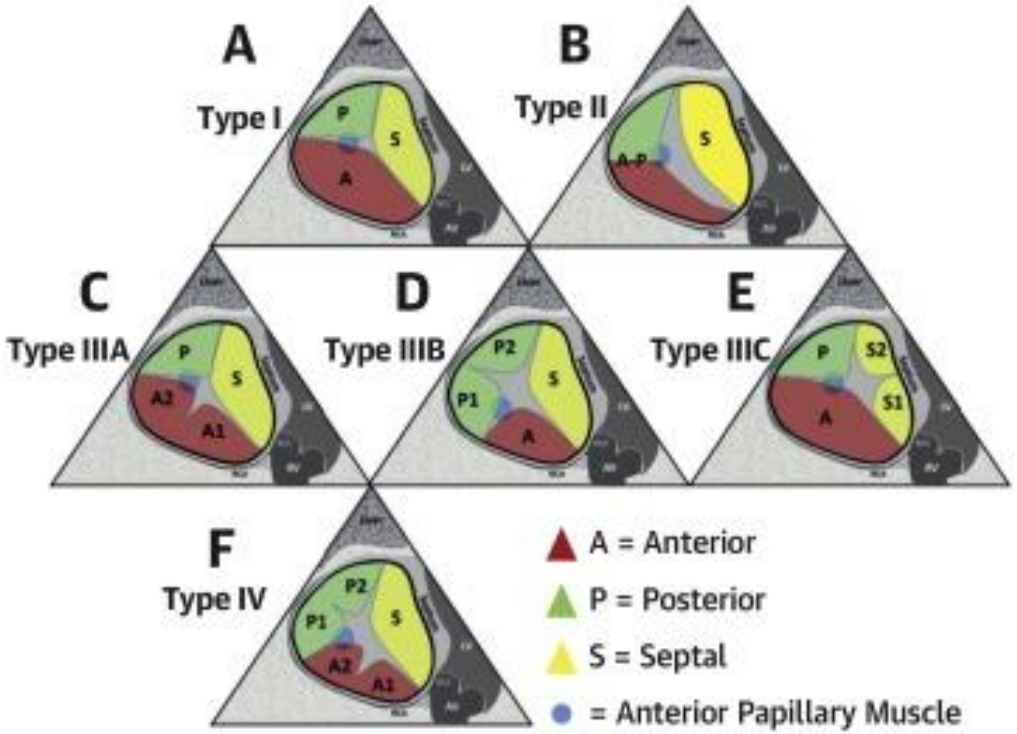
§preferably biplane.

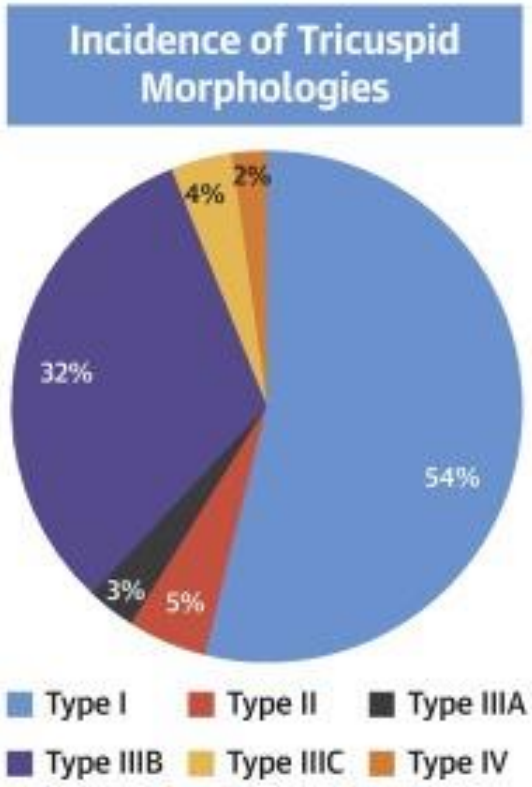
Multiplanar reconstruction and gastric short axis images on TEE





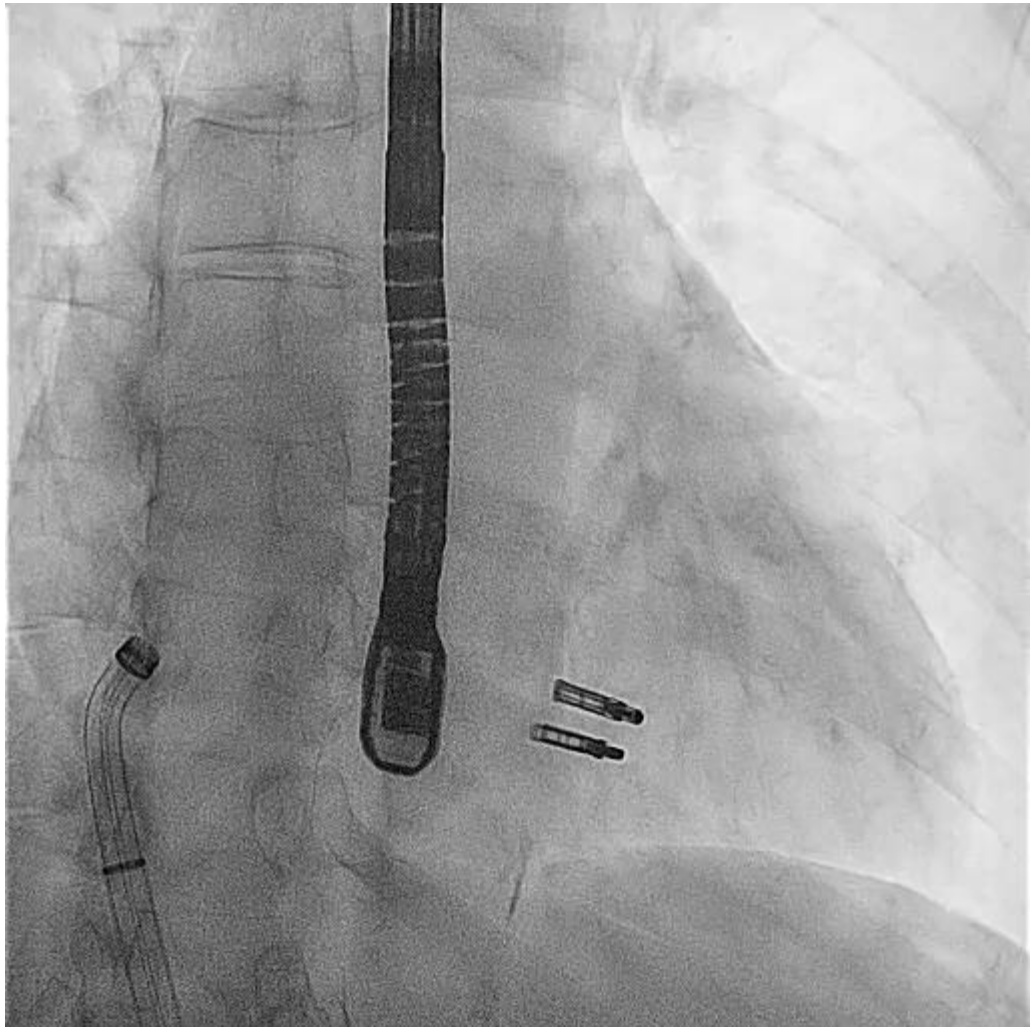
CENTRAL ILLUSTRATION: Tricuspid Valve Nomenclature Classification Scheme

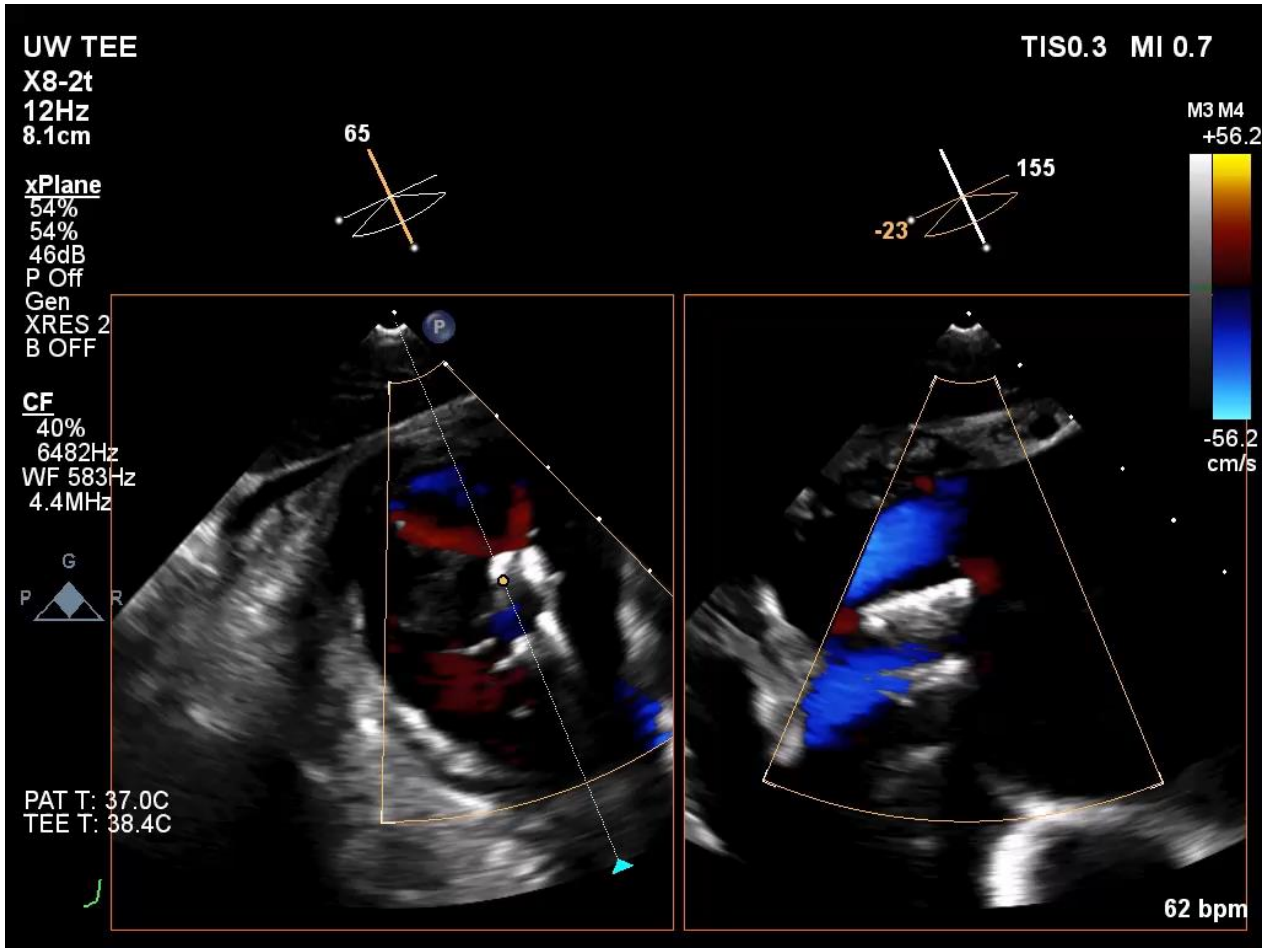




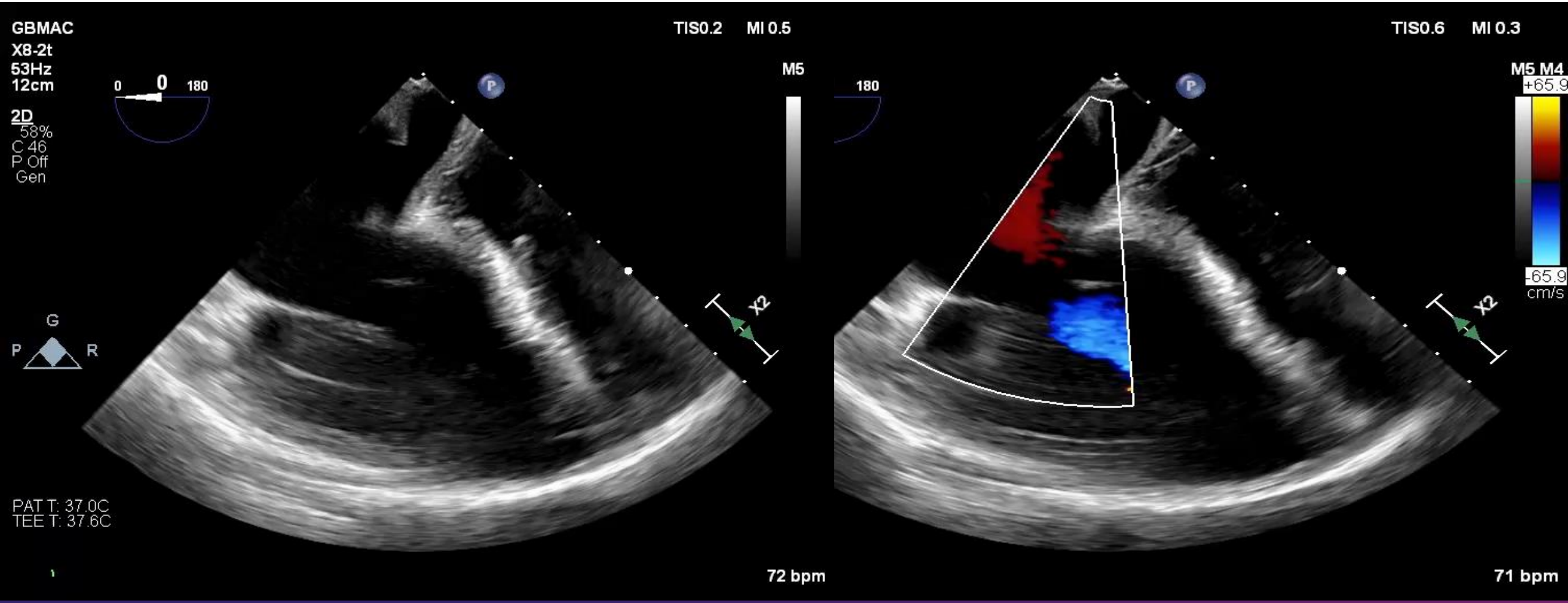
Hahn, R.T. et al. J Am Coll Cardiol Img. 2021;14(7):1299-305.

Anatomically a good candidate for tTEER

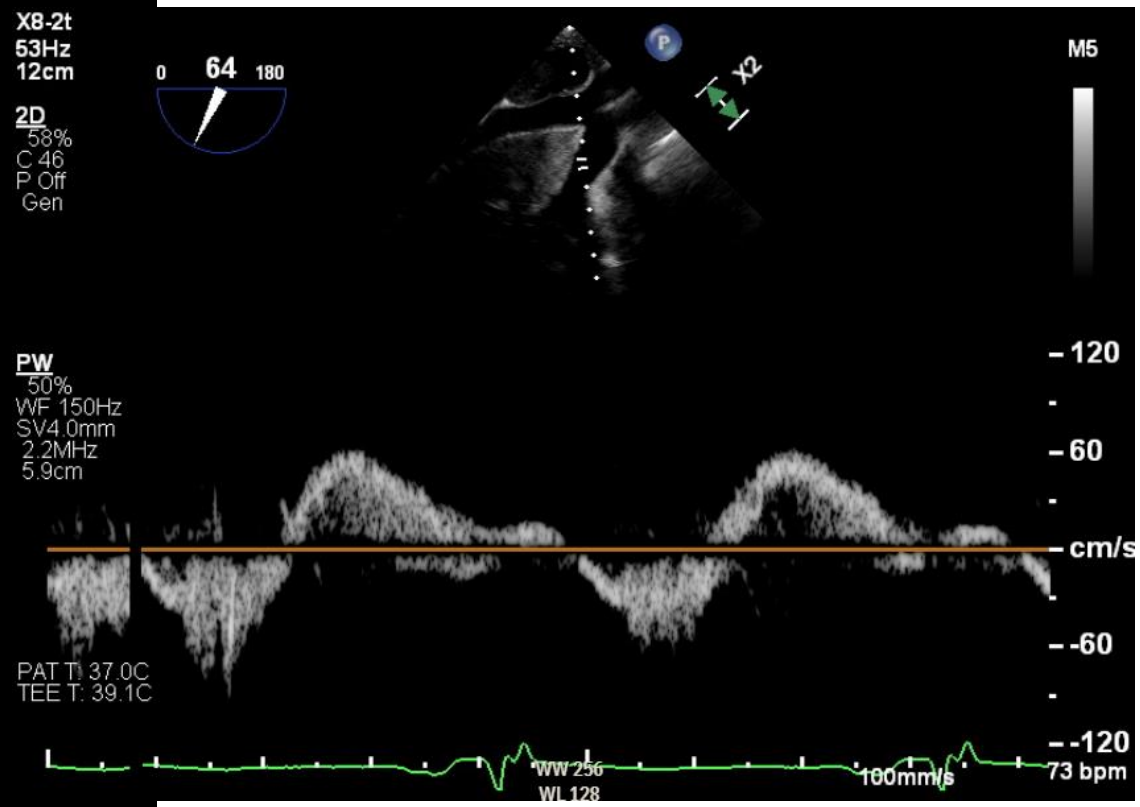




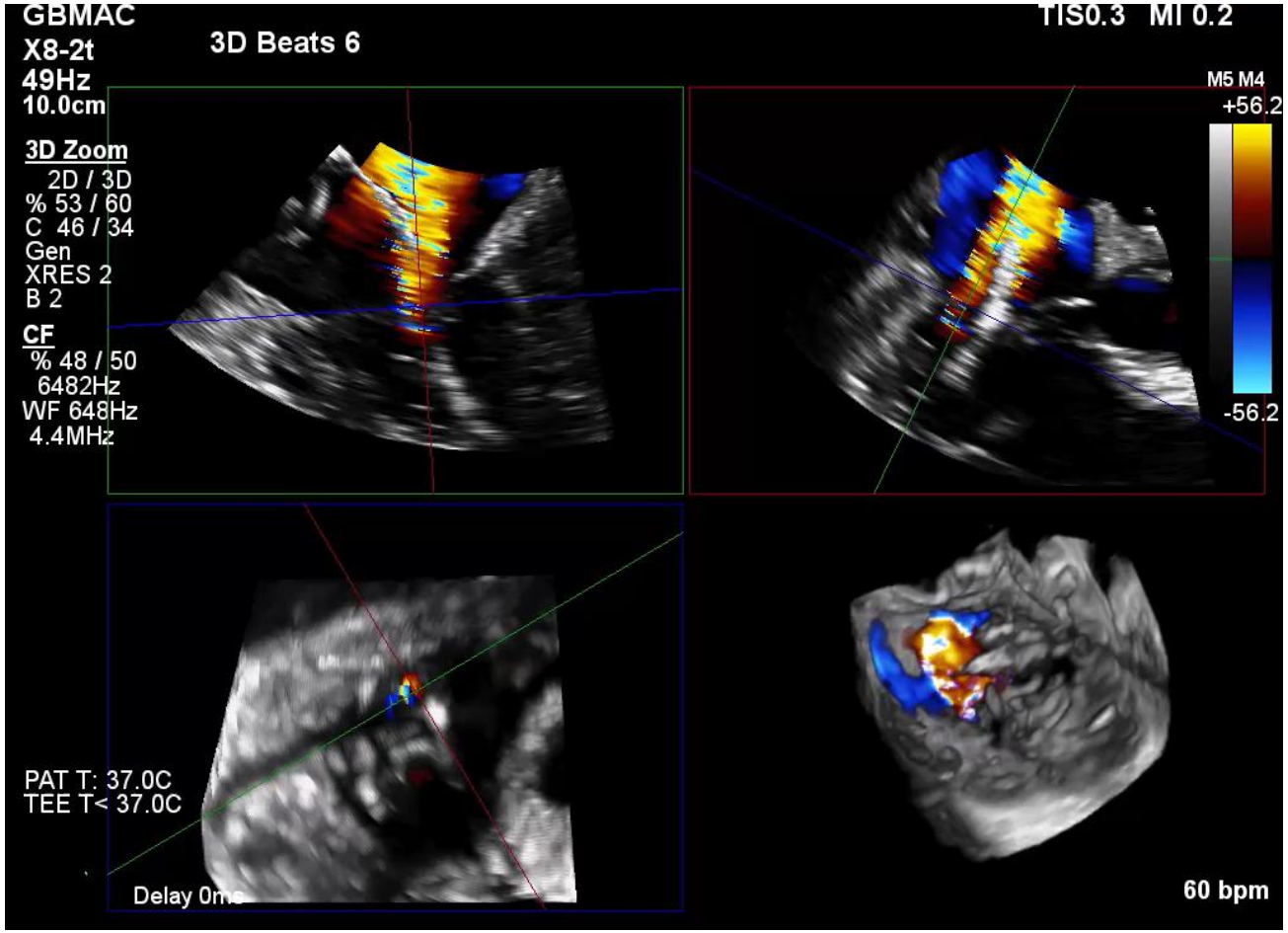
Clear images of leaflet pathology are possible

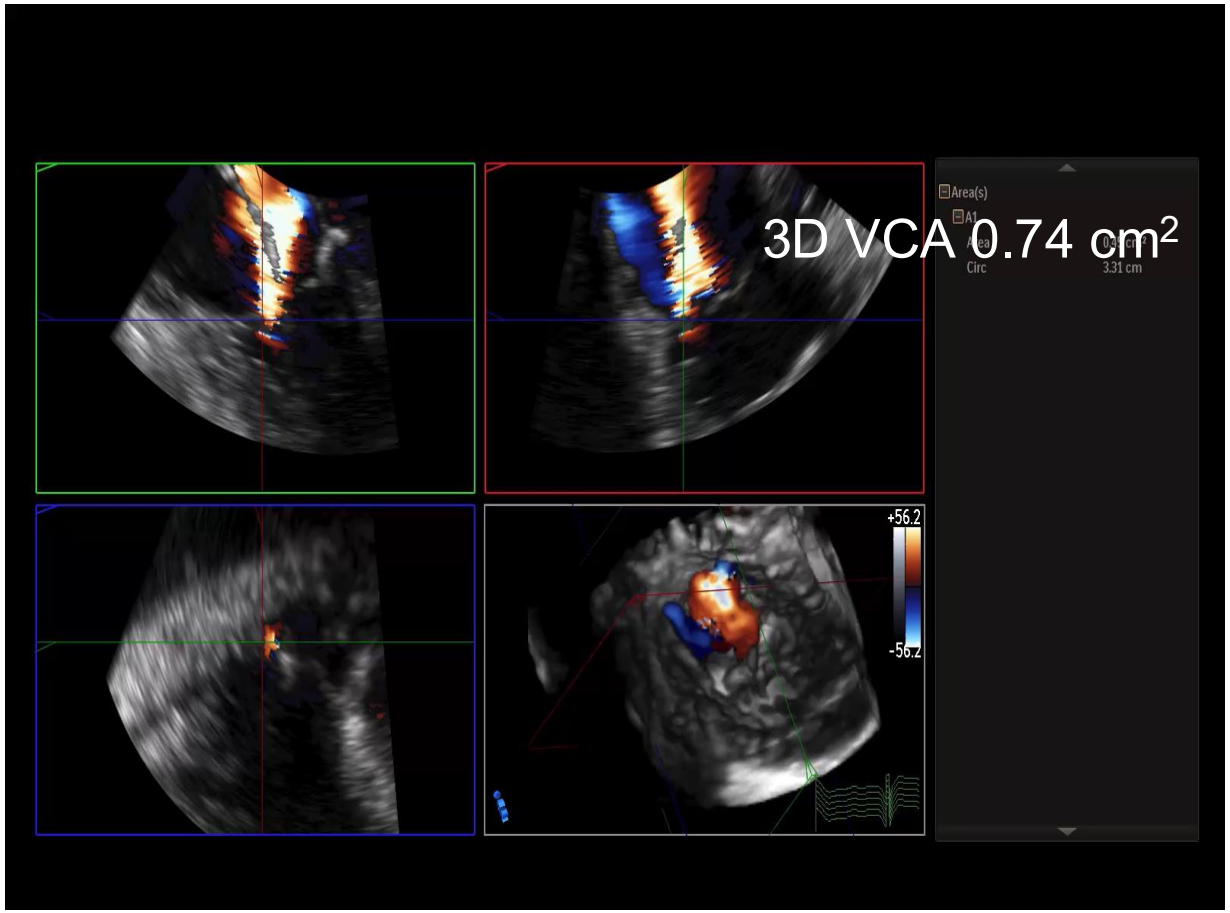


GBMAC
X8-2t
21Hz
9.1cm
3D Beats 1
0 60 180
3D Zoom
2D / 3D
% 62 / 43
C 46 / 30
Gen
XRES ON
TIS0.2 MI 0.2
PAT T: 37.0C
TEE T: 38.7C
71 bpm

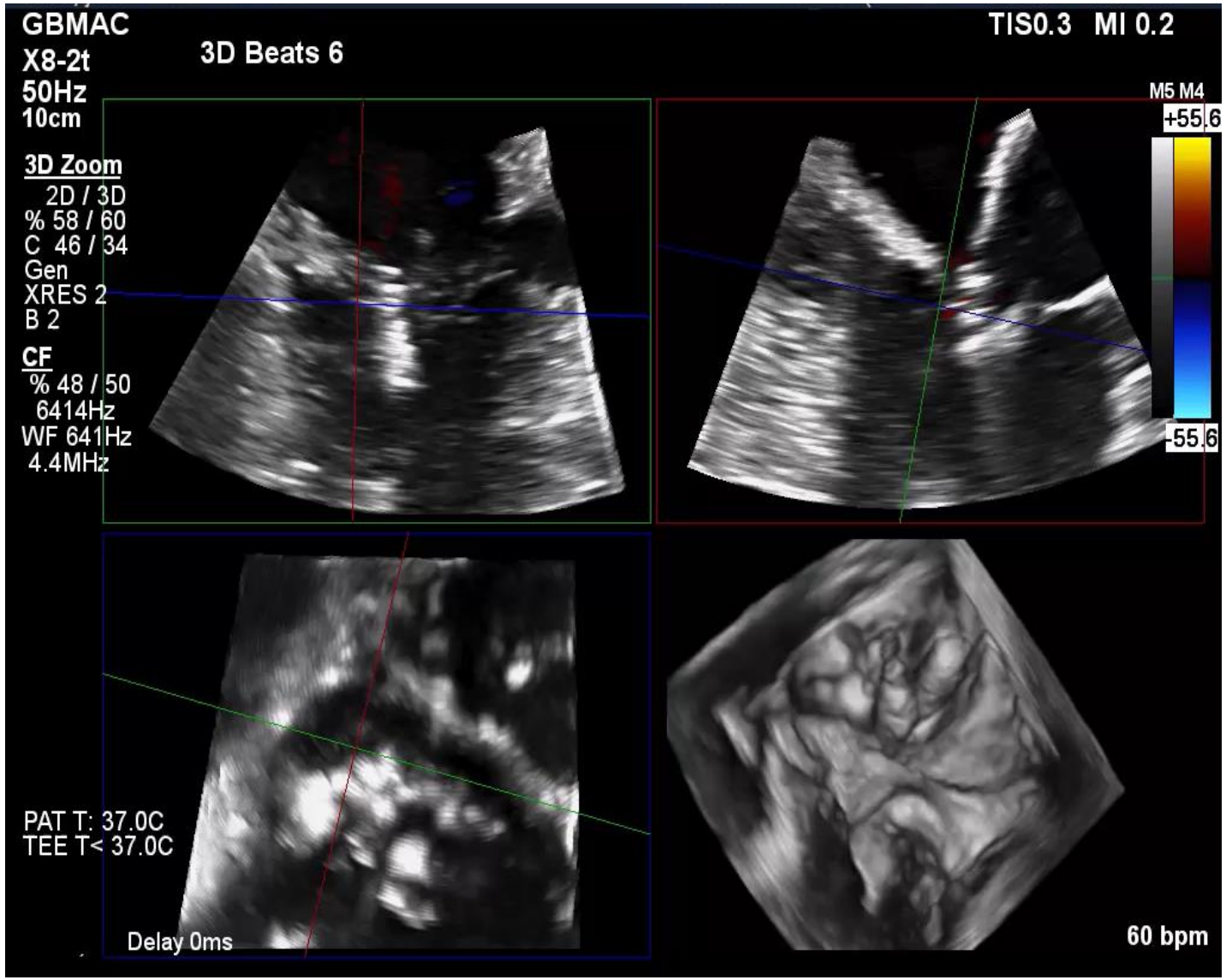

X8-2t
53Hz
12cm
0 64 180
2D
58%
C 46
P Off
Gen
M5
PW
50%
WF 150Hz
SV4.0mm
2.2MHz
5.9cm
120
60
cm/s
-60
-120
PAT T: 37.0C
TEE T: 39.1C
WW 256
WL 128
100mm/s
73 bpm

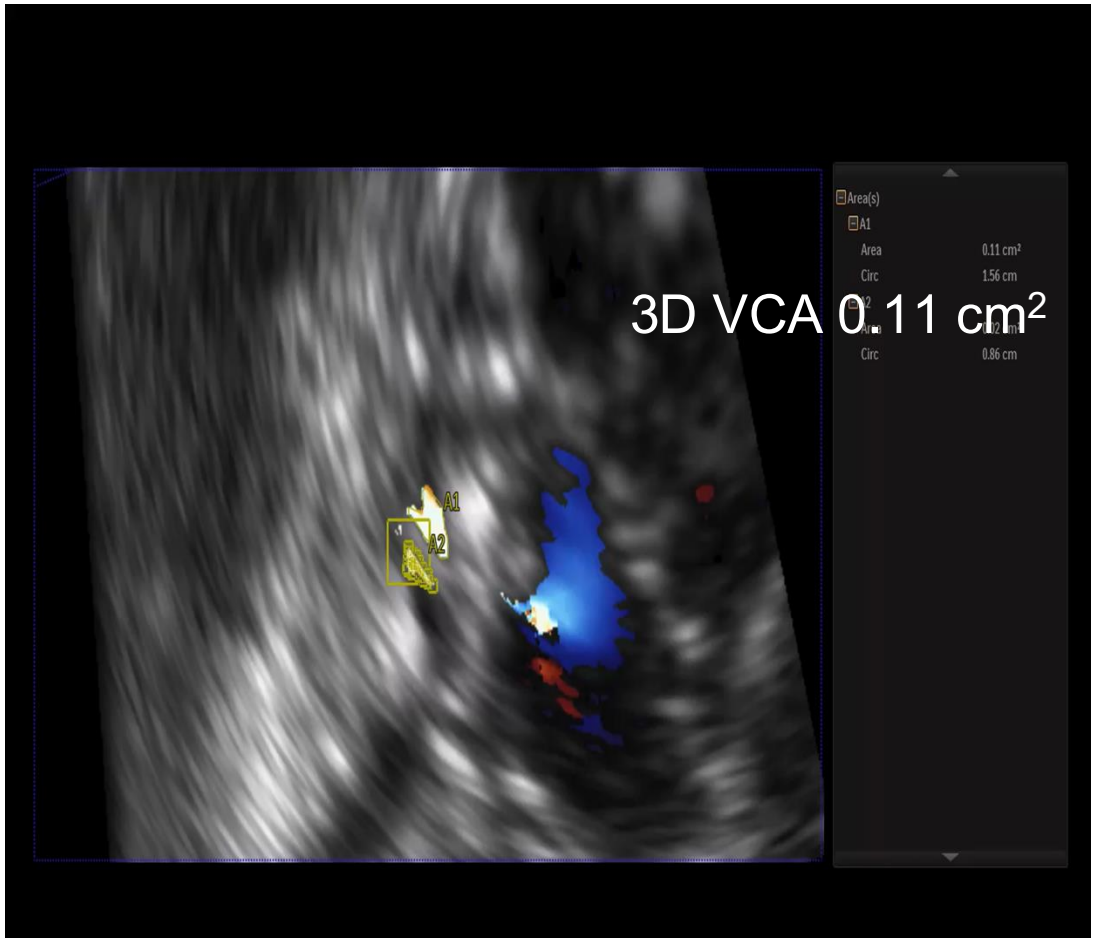
Severe TR to severe TR?
Understanding hemodynamic change beyond color Doppler?





High velocity jet?
Blooming?

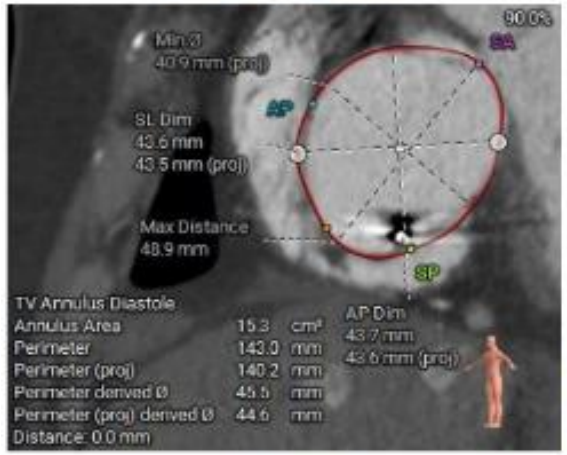




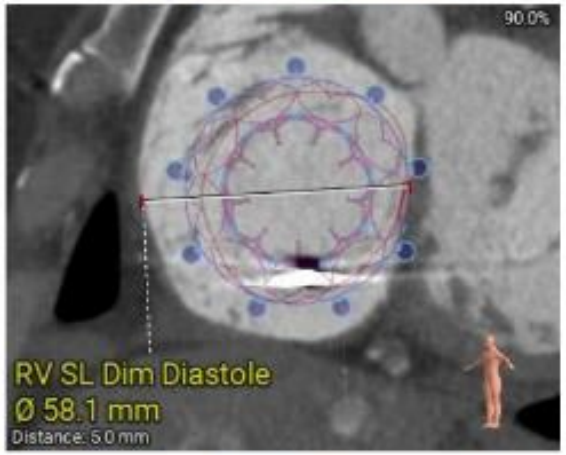
….much to learn

CT Preplanning in tricuspid valve replacement cases

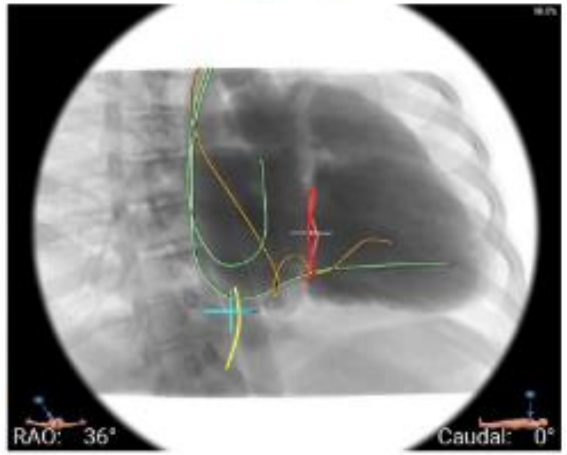
(1) TVA Diastole:



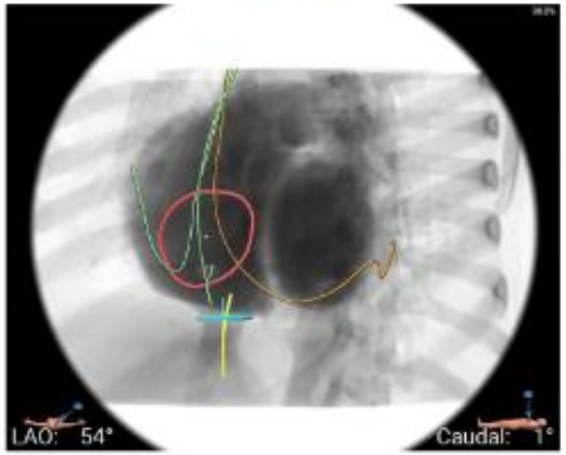
(2) Basal RV Diastole:



Implant:



En Face:



Anatomical "Working Room" - Delivery System

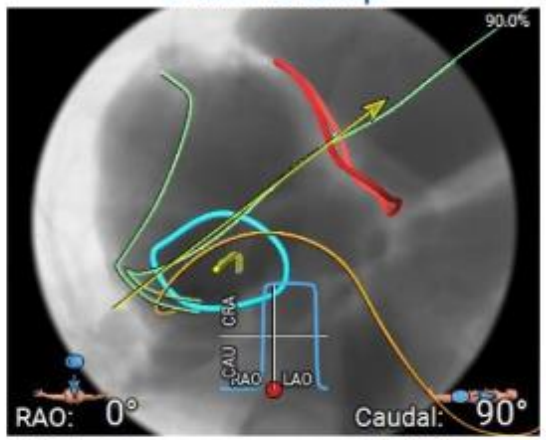
RA + RV Room
(CG height consideration)

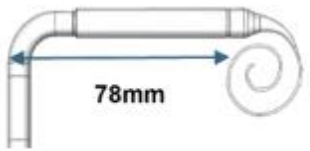
Anatomical Working Room - Diastole:

Anatomical Working Room - Systole:

IVC : TV Annulus Overlap
(Secondary flex consideration)

IVC:TVA Overlap:





Conclusions

1. Multimodality imaging for heart valve disease is rapidly expanding and is vital to the evaluation and treatment of these patients.
2. Aortic valve calcium scoring is an easily acquired, well validate metric. Get it with your TAVR CTA!

   --- Because of this, I have seen a reduction in the role of DSE.
3. Low threshold to obtain TEE in non calcified aortic regurgitation.

   --- Look for all etiologies including the aortic root.
4. cMRI measures of LV function and size are well validated predictors of mortality in aortic and mitral disease. Consistently protocols are needed.
5. Mitral and tricuspid valve assessment typically requires invasive imaging for complete understanding of severity and leaflet pathology, as well as to guide procedural plan.
6. Gated cardiac CTA is required for valve sizing and fit, as well as neoLVOT measurements in mitral cases.

Thanks!

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