ΕΛΛΗΝΙΚΗ ΔΗΜΟΚΡΑΤΙΑ
Εθνικόν και Καποδιστριακόν Πανεπιστήμιον Αθηνών
ΙΔΡΥΘΕΝ ΤΟ 1837
ΙΑΤΡΙΚΗ ΣΧΟΛΗ ΑΘΗΝΩΝ

ΚΑΡΔΙΑΓΓΕΙΑΚΗ ΑΠΕΙΚΟΝΙΣΗ

ΜΑΓΝΗΤΙΚΗ ΤΟΜΟΓΡΑΦΙΑ ΚΑΡΔΙΑΣ ΒΑΛΒΙΔΟΠΑΘΕΙΕΣ

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(CMR level 3, EACVI) – UZ Leuven, Belgium

https://cardiotherapis.gr

ASSESSMENT OF VALVULAR HEART DISEASE

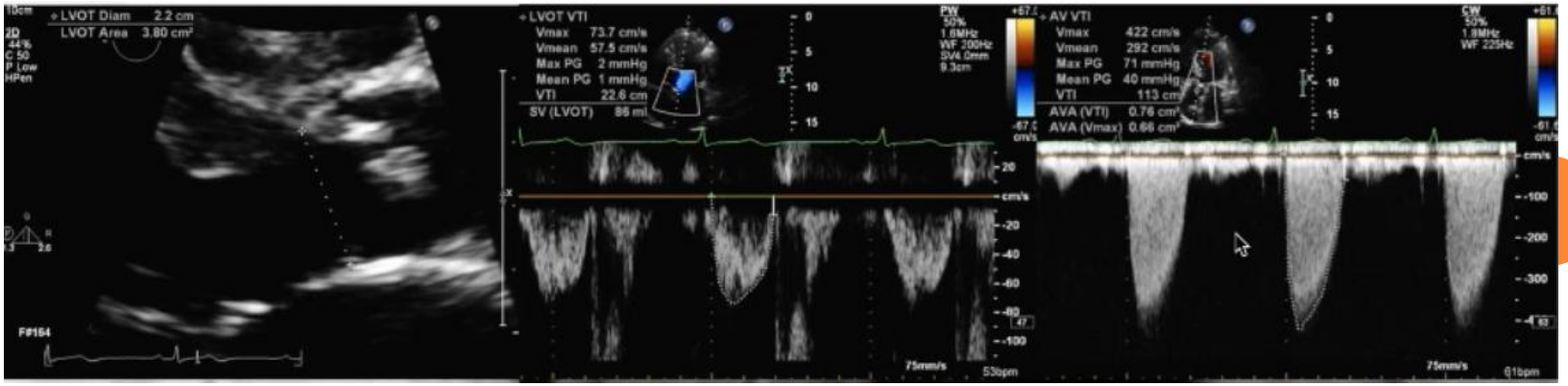
- Role of CMR
- Limitations
- Jet fluid dynamics
- Mechanisms and principles of CMR
- CMR protocols
  - Valvular stenosis
  - Valvular regurgitation

VALVULAR HEART DISEASE

- Echocardiography is the gold standard

"TTE is the standard diagnostic test in the initial evaluation of patients with known or suspected VHD." (ACC/AHA 2020)

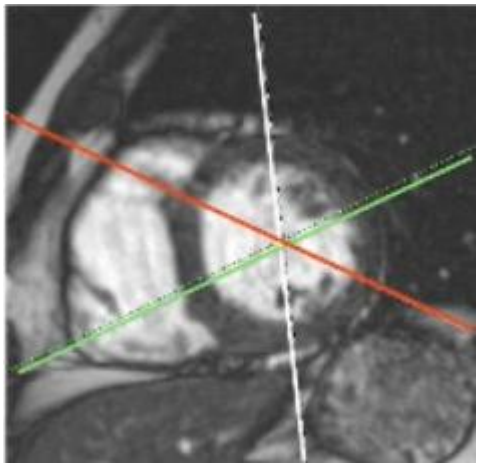
Echocardiography is the *"the key technique used to confirm the diagnosis of VHD, as well as to assess its severity and prognosis."* (ESC 2017)

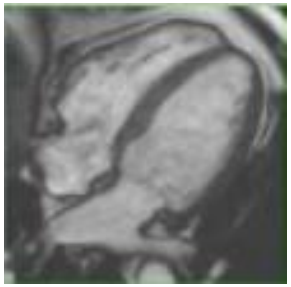


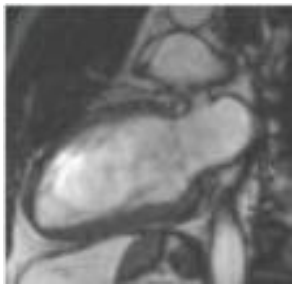
Complementary role of CMR

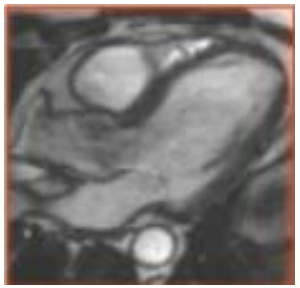
- When CMR?
  - Inadequate echo quality or discrepant results (e.g. eccentric jets)
  - Assess severity of valvular disease (especially regurgitation)
  - Assess mechanism
  - Assess ventricular volumes and systolic function
  - Higher reproducibility

Strengths	Weaknesses
Unlimited windows	Regurgitant jet visualisation not as good as echo
Excellent image quality	Low through-plane spatial resolution / Low temporal resolution
Flow quantification (reproducible)	Average of multiple RR
Gold standard for LV/RV assessment	Underestimation of peak velocities
Multi-parametric comprehensive assessment (LGE / ischaemia)	Cost / availability





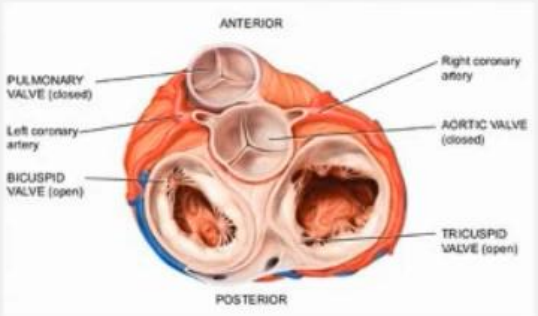




Comprehensive assessment

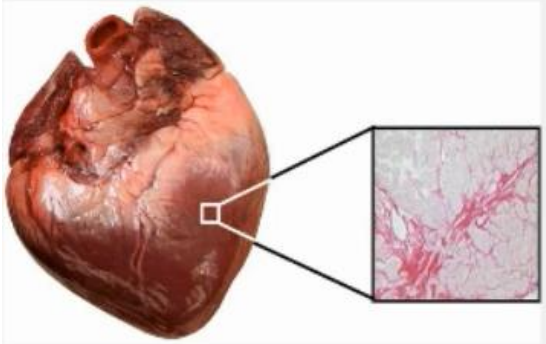
- A disease of the **valve** and the **myocardium**
- Chronic pressure / volume overload → cardiac remodeling
- Characterized by dilatation, hypertrophy and fibrosis

Valve



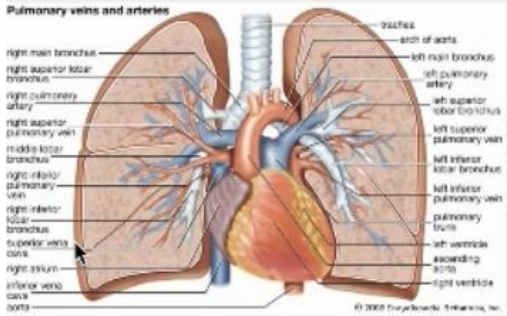
Morphology
Severity
Aetiology

Myocardium



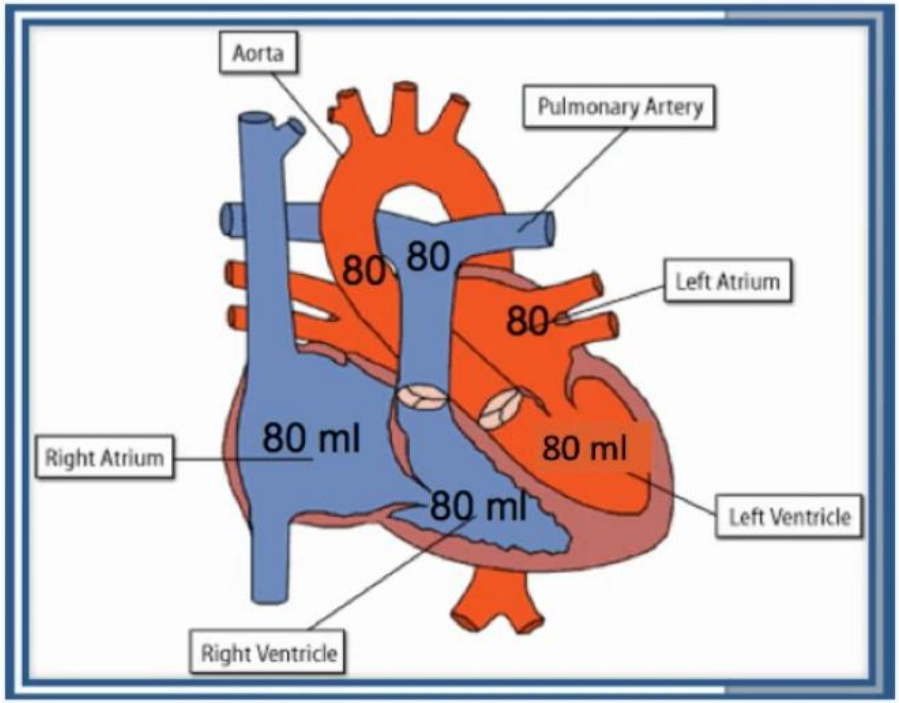
Mass, volume, function
Fibrosis
Ischaemia

Surrounding structures



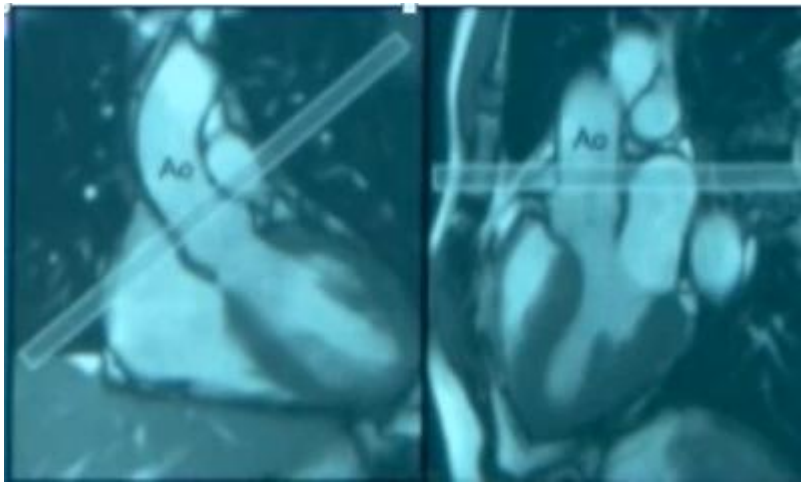
Aortopathy
Pulmonary hypertension
Concomitant disease/ decompensation

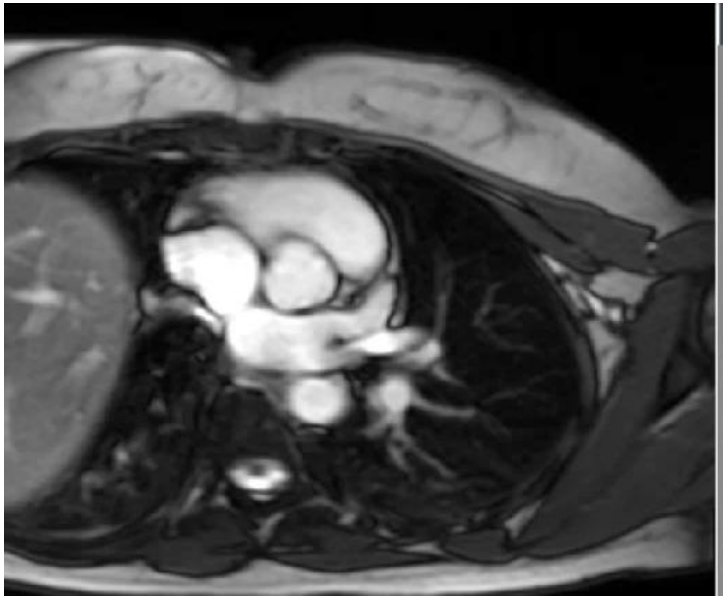
Conservation of Flow Principle

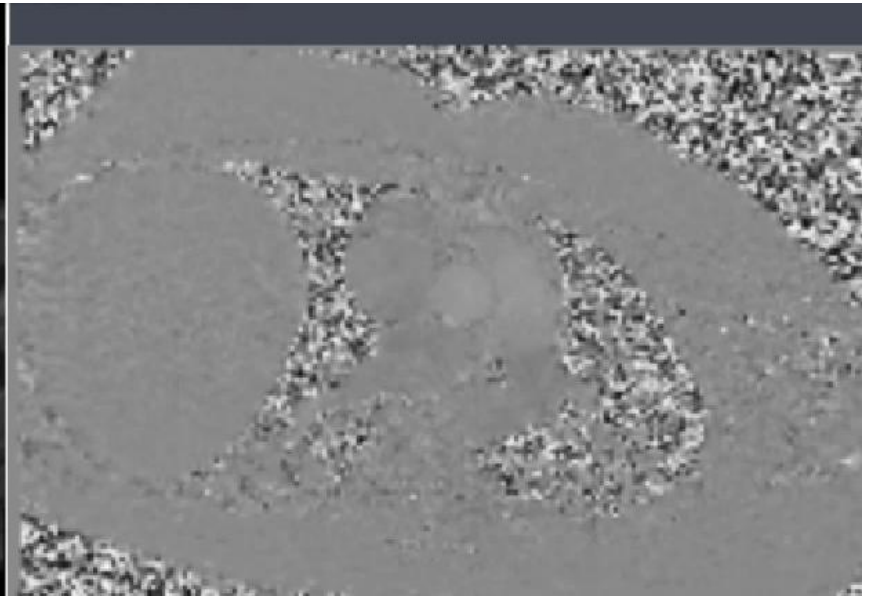


LVSV = RVSV = AO Flow = PA Flow

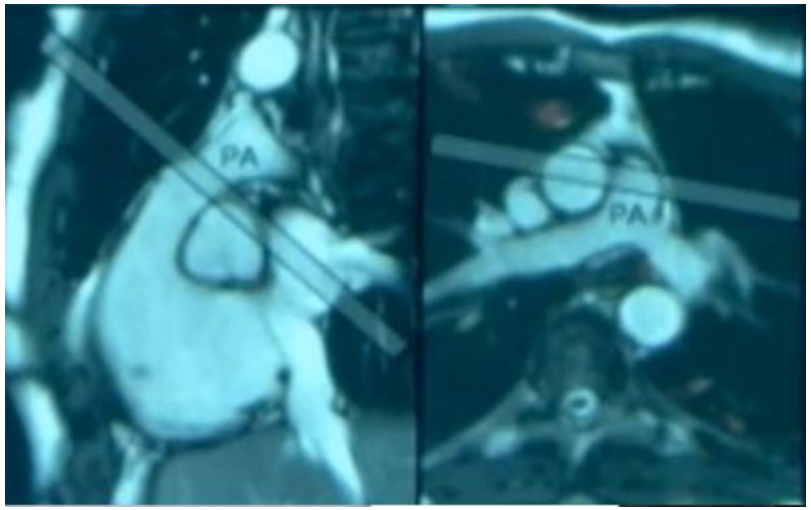
Aortic flow

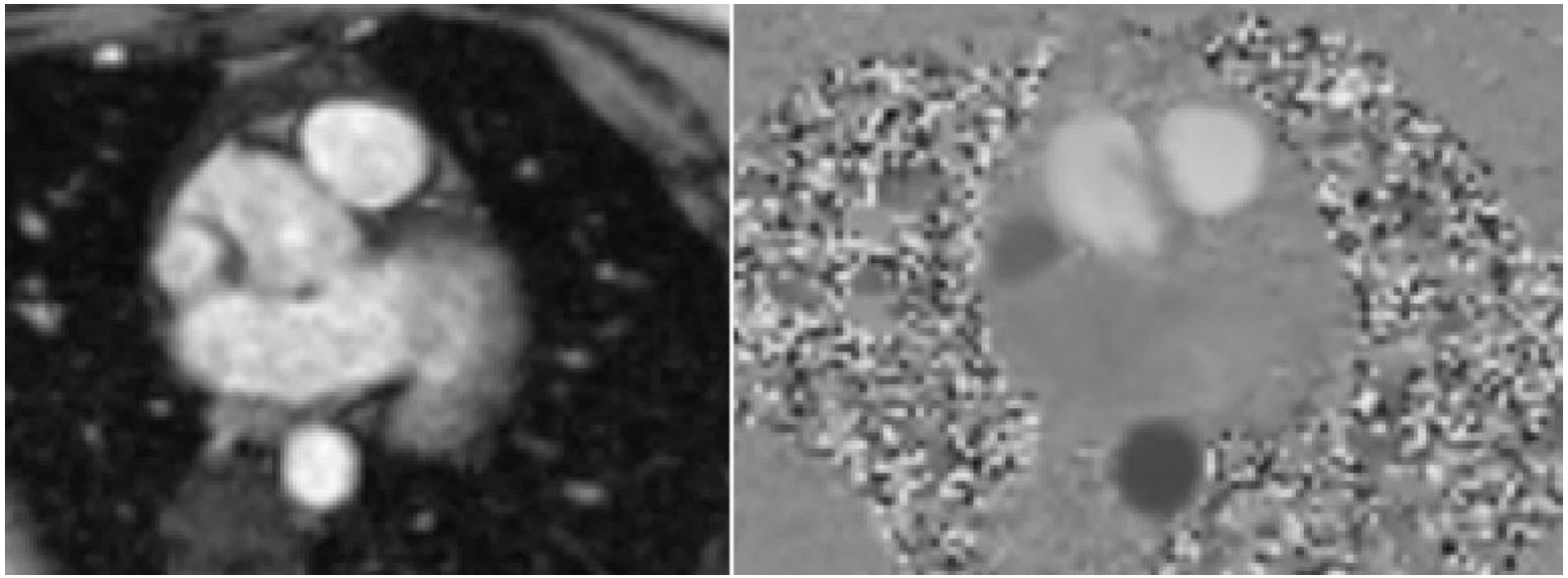




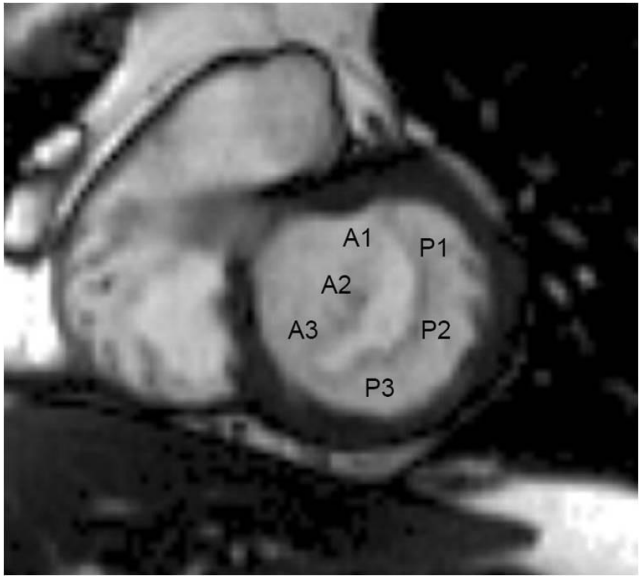


Pulmonary flow

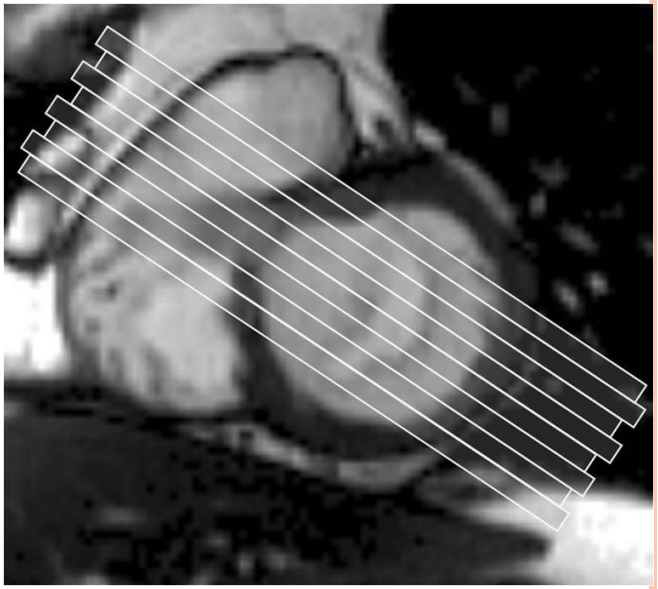




Mitral regurgitation



Valve morphology/ aetiology
Regurgitant volume/fraction
LV volume/ function
LA volume
Tissue characterization



Anatomy of the mitral valve. CMR short axis view of the mitral valve from a basal short axis slice showing its two leaflets (anterior and posterior) and the three scallops of each leaflet (A1, A2 and A3 in the anterior leaflet, P1, P2 and P3 in the posterior leaflet). The mitral valve is viewed from the LV looking towards the left atrium.

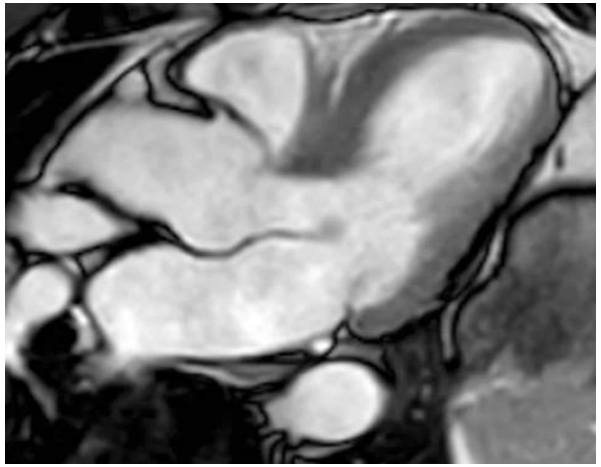
Slices taken across the mitral valve. 5 mm thick slices are taken starting from the superior (antero-lateral) commissure (A1-P1) and moving towards the inferior (postero-medial) commissure (A3-P3) at 5 mm intervals. The orientation of the slice is parallel to the LVOT slice.

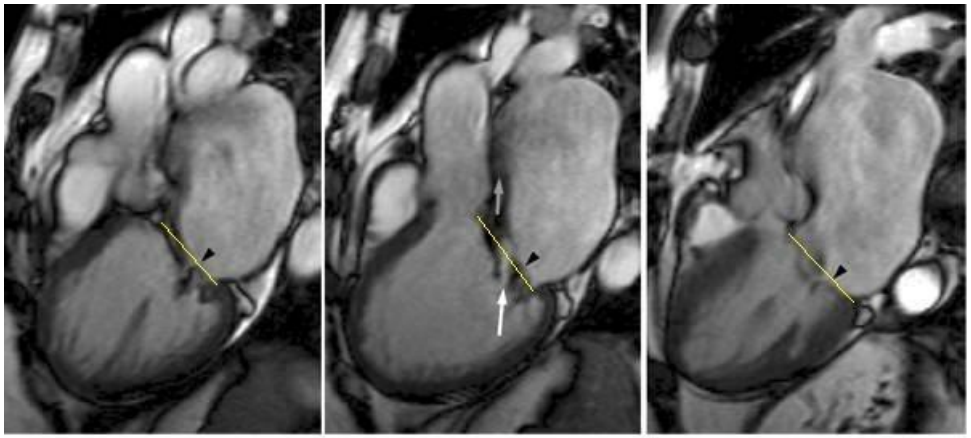
MR (MORPHOLOGY/AETIOLOGY)

Degenerative (primary) mitral regurgitation

Caused by a primary abnormality of components of the valve apparatus

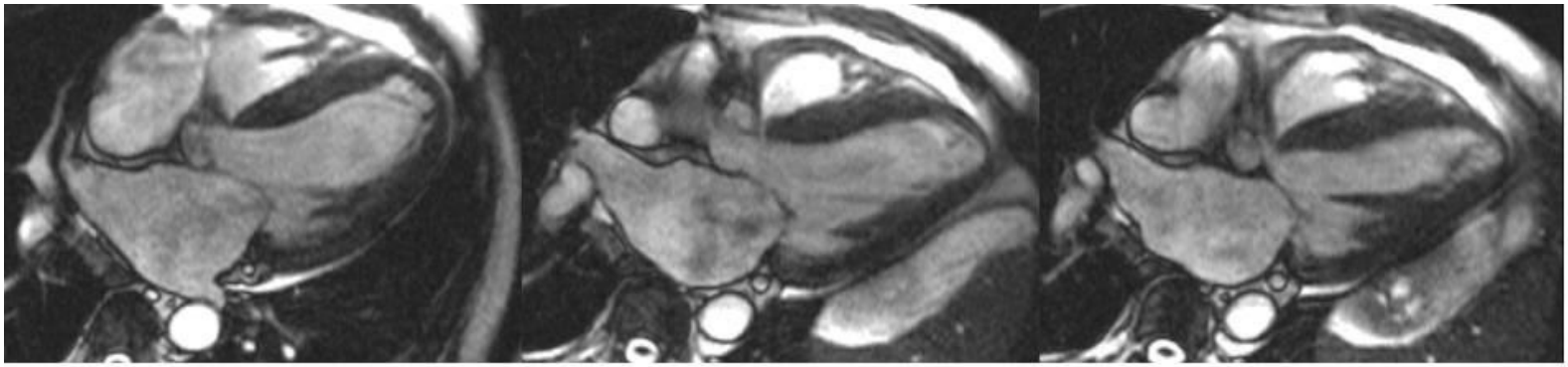
- Chordae tendineae rupture (fibroelastic deficiency: FED)
- Myxomatous degeneration with prolapse and/or flail (FED +, forme fruste, Barlow's disease)
- Rheumatic heart disease
- Endocarditis
- Papillary muscle rupture (eg. post MI)
- Mitral annular calcification





(a) A1-P1 (b) A2-P2 (c) A3-P3

MR (MORPHOLOGY/AETIOLOGY)



(a) A1-P1 (b) A2-P2 (c) A3-P3

Images obtained of each scallop of the mitral valve. Each scallop of both mitral valve leaflets is clearly visualised: (a) A1-P1, (b) A2-P2, (c) A3-P3.

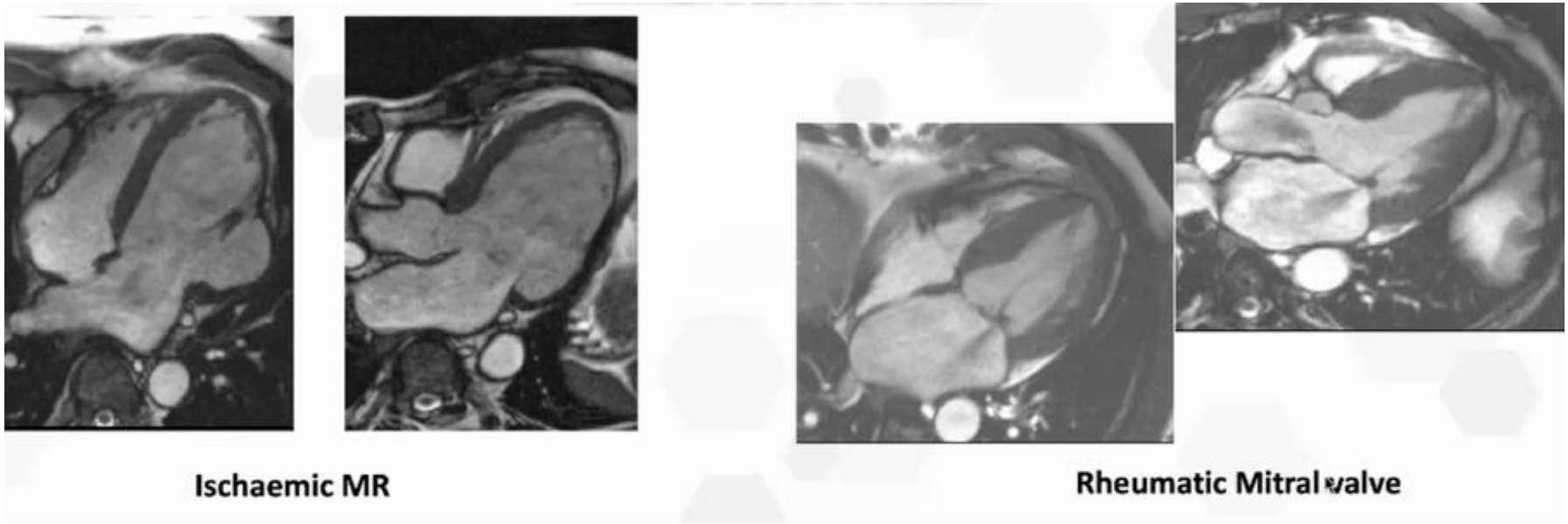
Moderate centrally directed mitral regurgitation is seen most marked at (b) A2-P2 due to ***leaflet restriction*** following ***myocardial infarction***.

Functional (secondary) mitral regurgitation

Occurs despite a structurally normal mitral valve

- Ischaemic cardiomyopathy
- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Severe left atrial dilation

MR (morphology/aetiology)

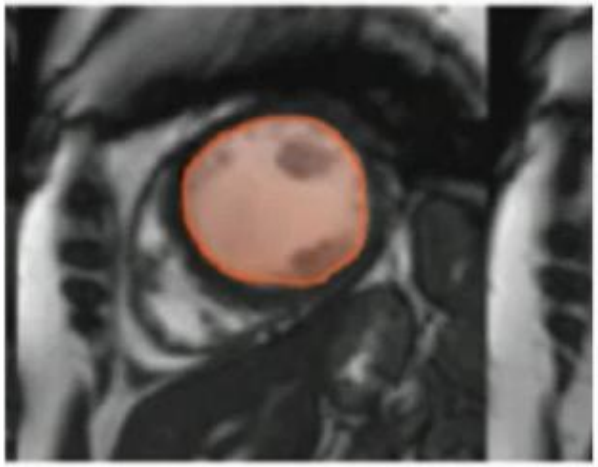


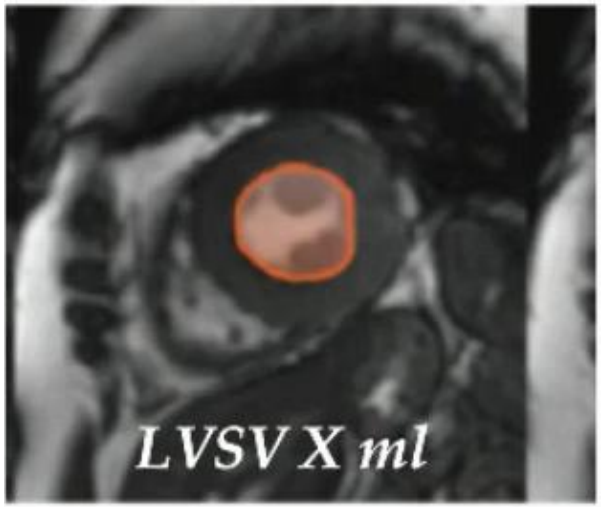
Ischaemic MR

Rheumatic Mitral valve

Quantification of MR severity

End-diastole

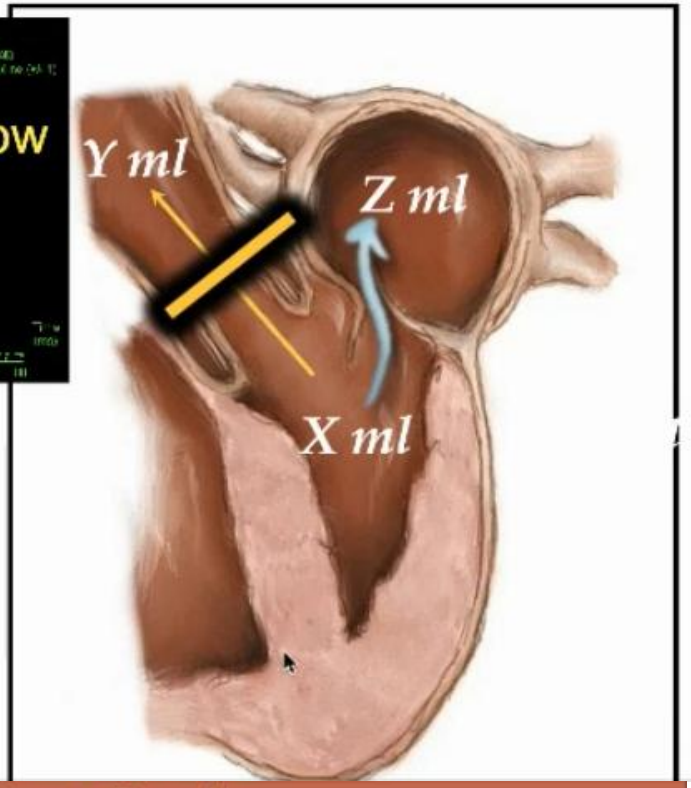




End-systole

Mitral Reg Vol = LV stroke volume – Aortic forward volume

Z = X - Y



MR fraction = MR volume / LVSV (X100, for %)

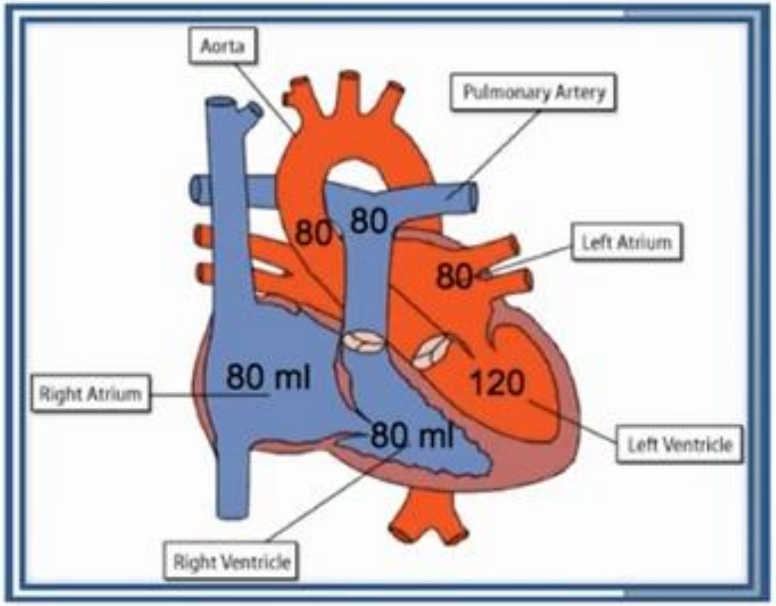
This technique is not affected by:

1. Presence of changing degrees of MR during systole.
2. Eccentric jets
3. Mobile mitral regurgitant jets.

Quantification of MR severity

- **Mitral Regurgitant Volume =**
  1) LVSV – Aortic Forward Flow
     - Applies even in presence of AI
     - more practical and reproducible than the other methods
  2) LVSV – Net Pulmonary SV
     - AoSV is within 5% of PA SV (in absence of intra-cardiac shunt)
     - Useful in patients with AS, where asc aortic flow may have aliasing
  3) LVSV – RVSV*
     - RVSV less reproducible due to extensive trabeculation of RV
     - Significant concomitant regurgitant lesions invalidates use
     - Useful in setting of atrial fibrillation.
- **Regurg Fraction (%) = MR Rvol / LVSV***

* In absence of concomitant left sided valve lesions

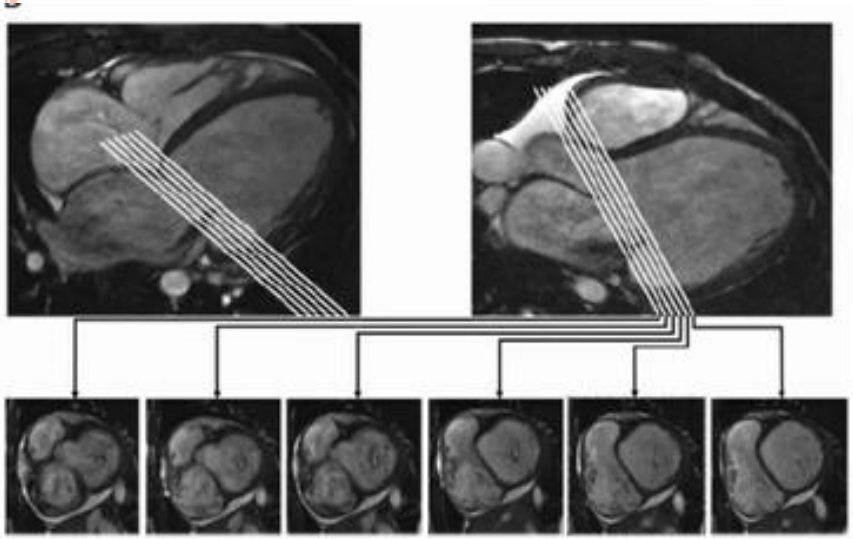


Quantitative Definitions of Severity of Mitral Regurgitation

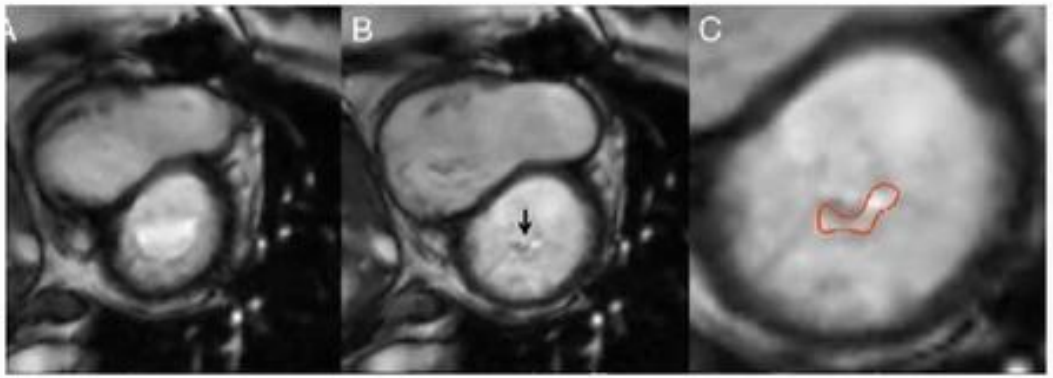
Degree of Regurgitation	Regurgitant Volume (mL/beat)	Regurgitant Fraction
Mild	<30	<30
Moderate	30–59	30–49
Severe	>60	>50

Quantification of MR severity

Direct Planimetry of the AROA Using CMR

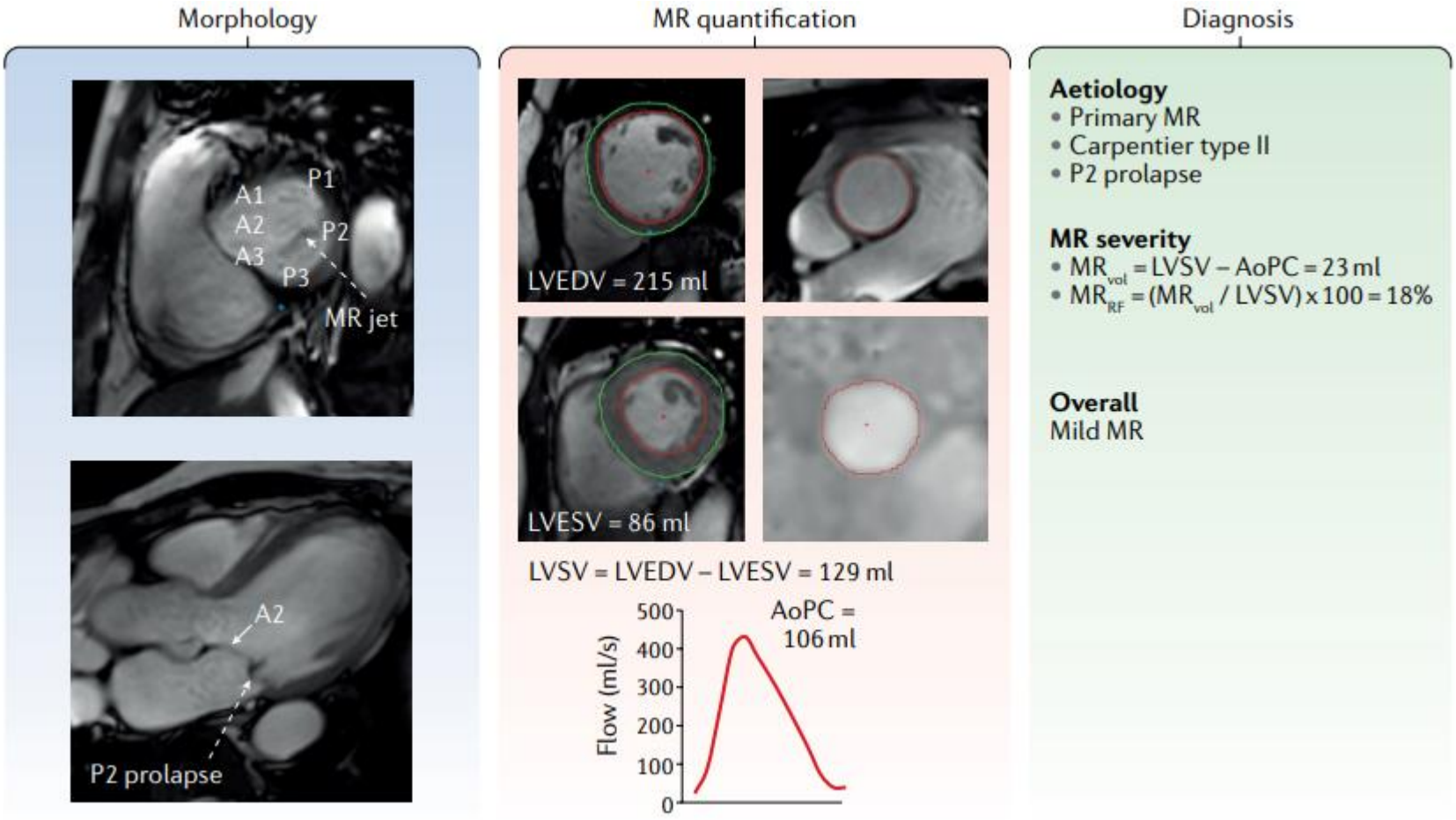


En face view of the regurgitant orifice



CMR-AROA > 0.40 cm2 had a 94% sensitivity and specificity for identification of Sellers angiographic grade 3 or 4 MR

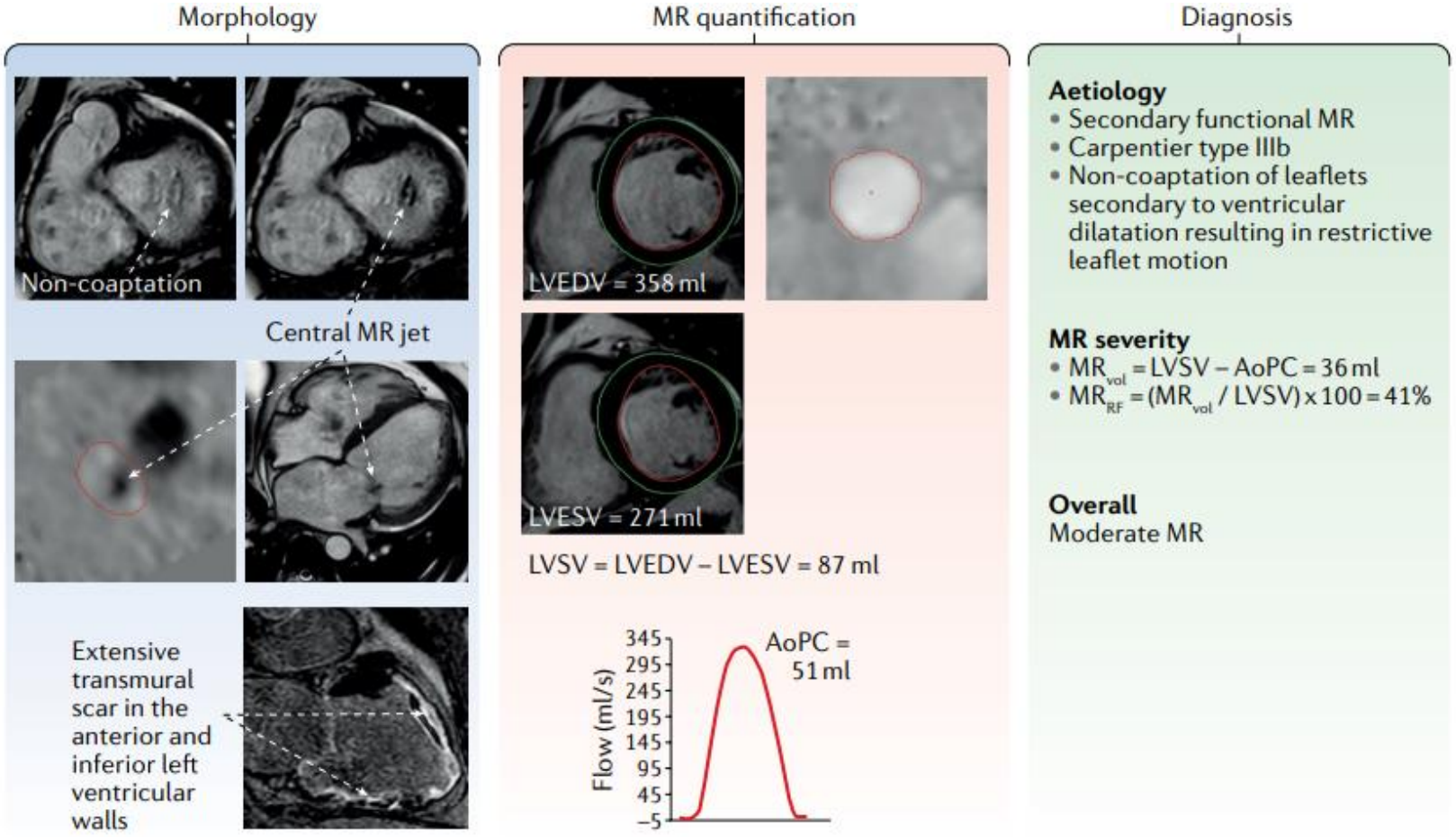
Mitral regurgitation (primary)



P2 prolapse (mid-systole)

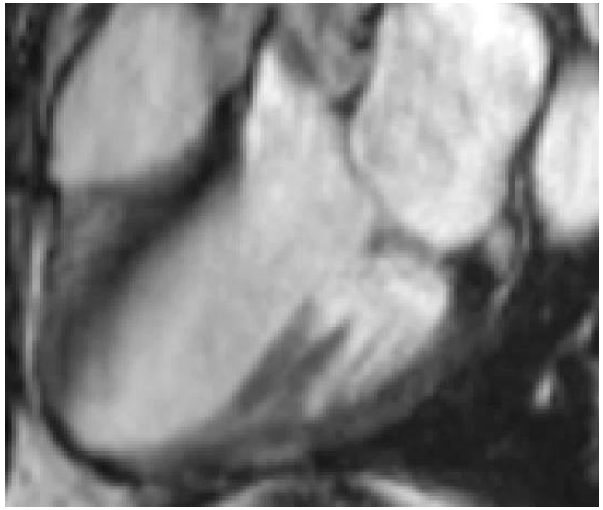
MITRAL REGURGITATION (SECONDARY)

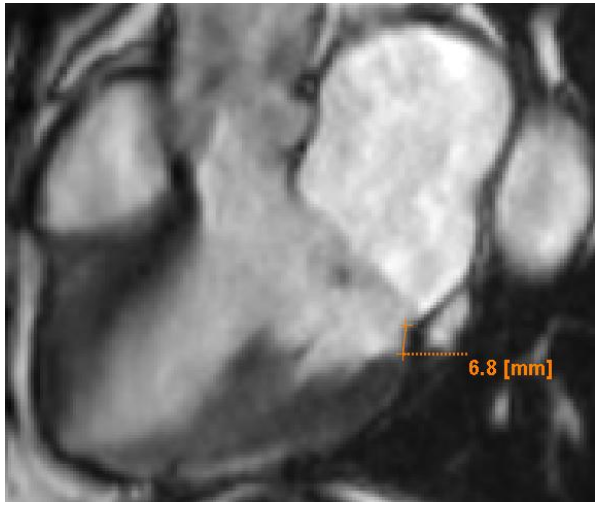
Ischaemic cardiomyopathy



Non-coaptation due to ventricular dilatation

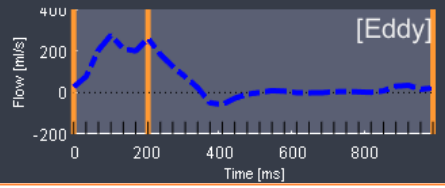
MR PROLAPSE





LV	Stack #14
ED/ES LVM [g]	100 / 98
EDV [ml]	157
ESV [ml]	59
SV [ml]	98
EF [%]	62
CO [L/min]	6.1
HR [bpm]	62

Flow	Stack #58
ROI-1	
Net vol [ml]	56.33
Forward vol [ml]	61.35
Regurg. vol [ml]	-5.02
Regurg. frac. [%]	8
FlowCO [L/min]	3.3
HR [bpm]	59



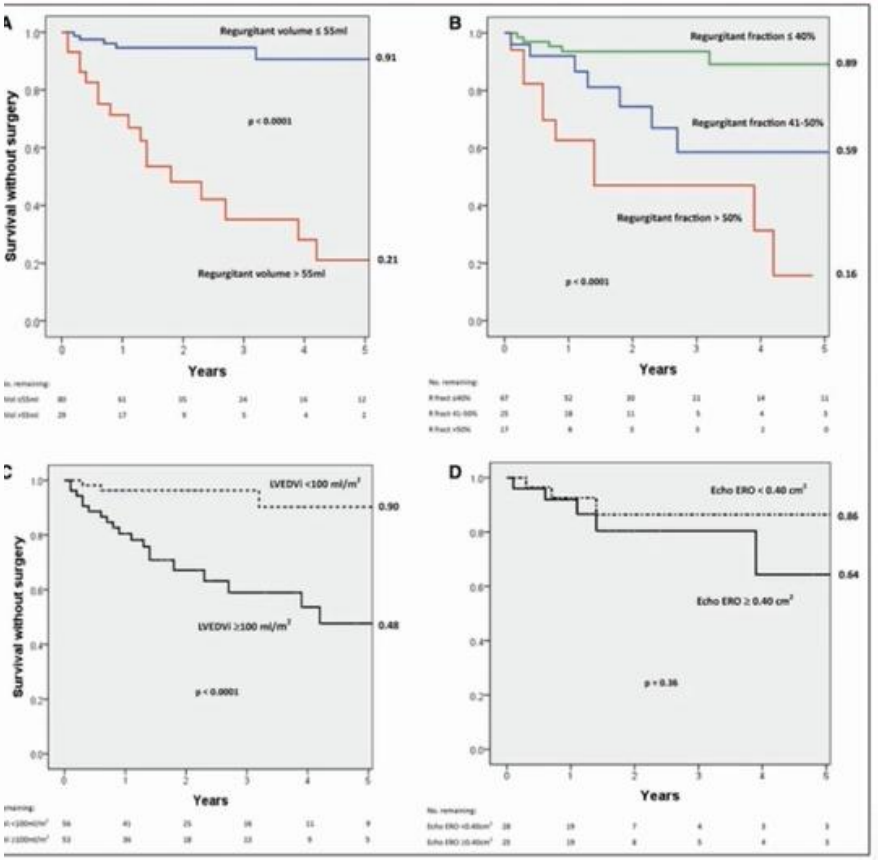
Mitral RV = LVSV – AoFF = 98 – 61 = 37
RF = Mitral RV / LVSV x 100 = 37/98 x 100 = 38%

Mitral Annular Disjunction (MAD) > 8.5 mm is related with higher arrhythmia risk

Mitral regurgitation

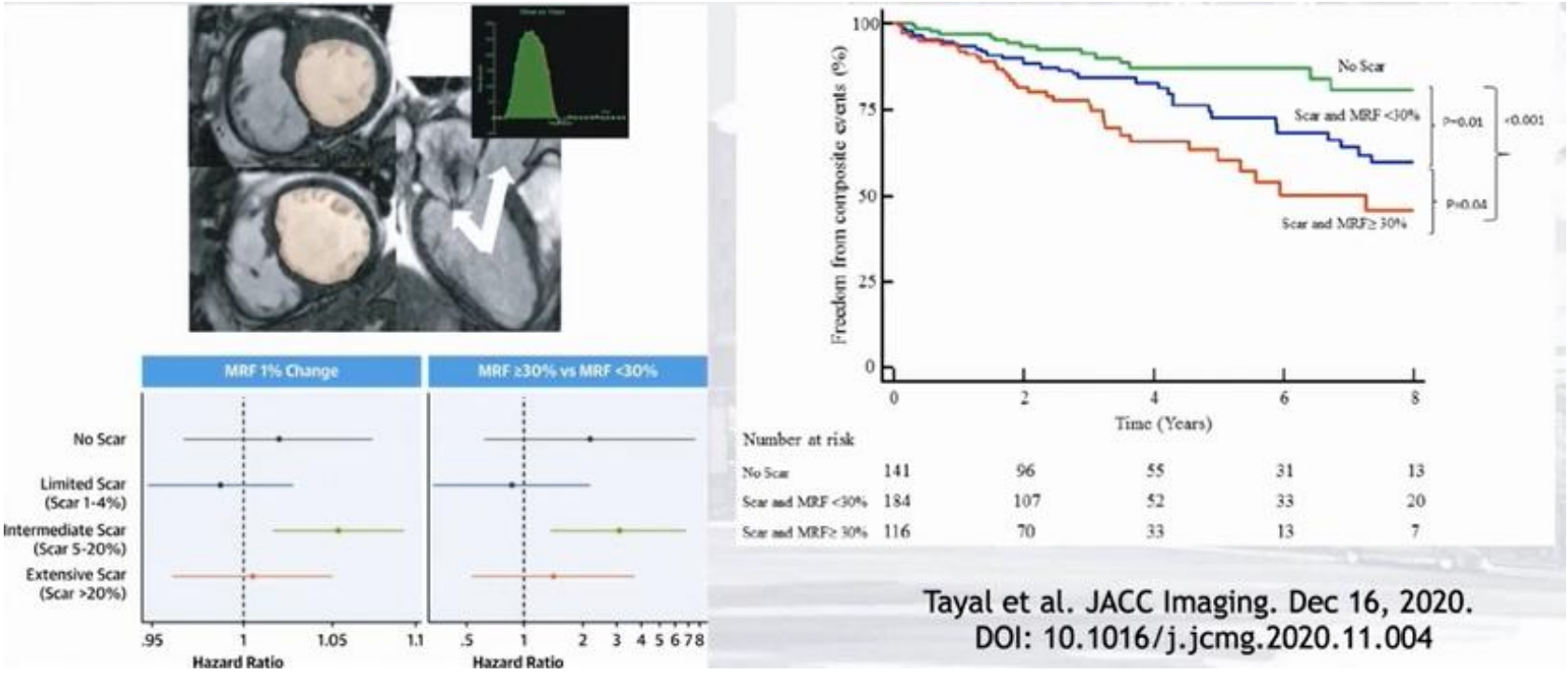
- CMR quantification of regurgitant volume >55ml or regurgitant fraction >40% accurately identified patients who progressed to surgery

- CMR-derived end-diastolic volume index showed a weaker association with outcome and added little to the discriminatory power of regurgitant fraction/volume alone.



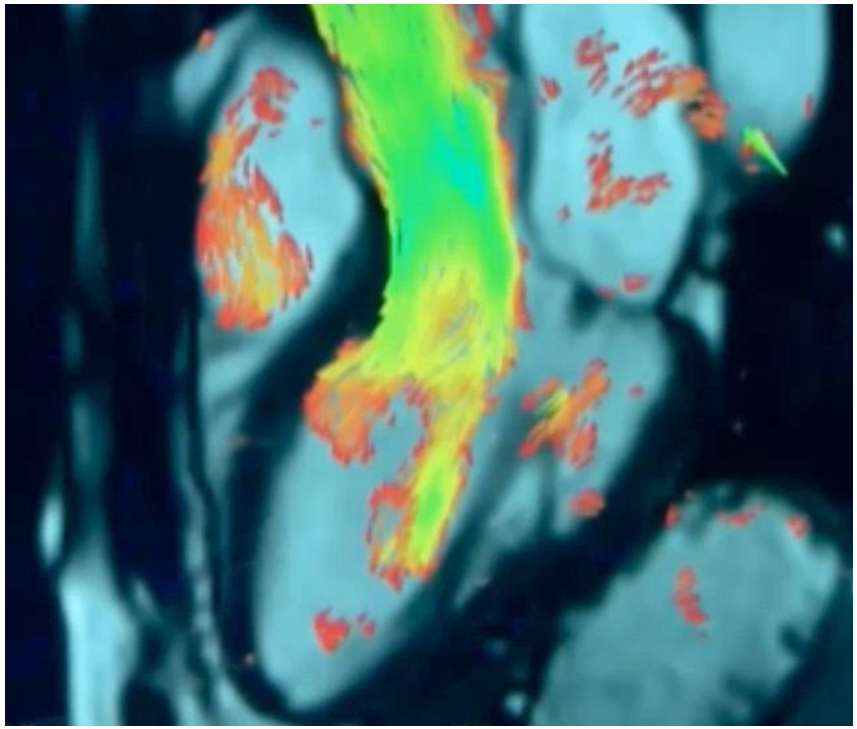
Type of MR	Grading of severity			
	Mild	Moderate	Severe	Very severe
Primary	MR_{RF} <20%[a]	MR_{RF} = 20–39%[a]	MR_{RF} 40–50%; MR_{vol} >55–60 ml	MR_{RF} >50%
Secondary	MR_{vol} <30 ml	MR_{vol} = 30–60 ml	MR_{vol} ≥60 ml	–

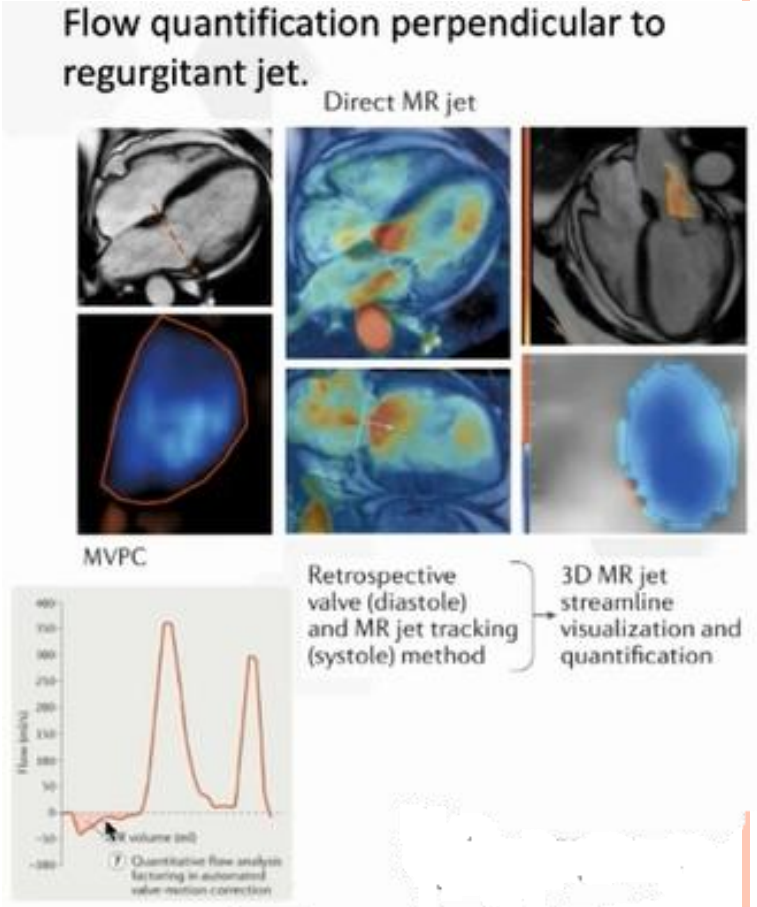
Impact of scar on prognosis of secondary MR in HF



Results: Among patients (n = 441) included in the study (age 59 ± 14 years, 43% with ischemic etiology), 85 (19%) experienced an adverse event. MRF ≥30% was associated with increased risk of events among the study group (hazard ratio: 1.74; 95% confidence interval: 1.10 to 2.76; p = 0.02). When stratified by presence or absence of scar, MRF ≥30% was associated with events only among patients with scar (hazard ratio: 1.67; 95% confidence interval: 1.02 to 2.76; p = 0.04) but not among patients without scar. On further classification of patients with scar, the prognostic significance of secondary MR was observed primarily among patients with intermediate scar burden.

4D FLOW





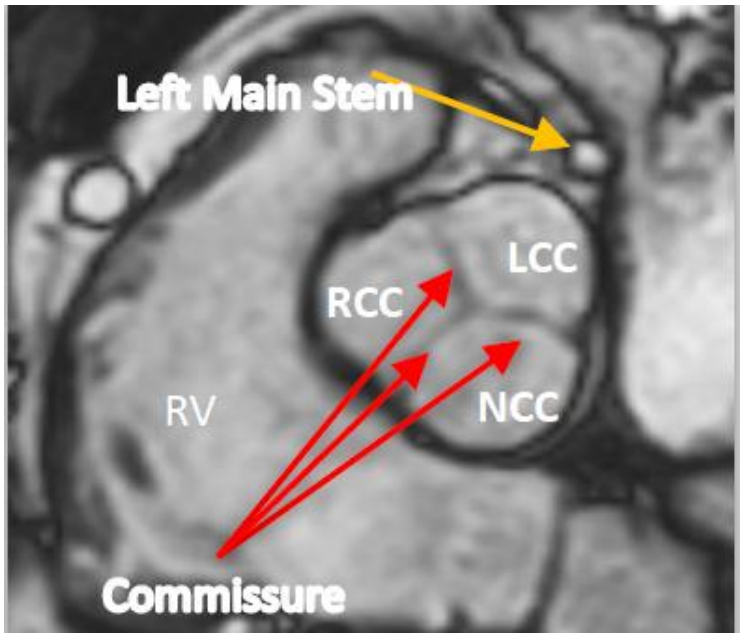
4D flow MRI is an advanced imaging technique that captures three-dimensional (3D), time-resolved (4D) data of blood flow velocity across the entire cardiac cycle. It enables comprehensive visualization and quantification of complex cardiovascular hemodynamics, such as wall shear stress and turbulent kinetic energy. It is primarily used for diagnosing complex congenital heart disease, thoracic aorta diseases, and valvular disorders.

Aortic valve imaging

Anatomy

1. **AV components**
   - Three equal cusps: left coronary cusp (LCC), right coronary cusp (RCC), non-coronary cusp (NCC)
   - These cusps overlap by 2-3mm to form commissures
2. **Aortic Sinus**

   The 3 AV cusps attach to three adjacent aortic sinuses which have the same anatomical names: right, left, non-coronary aortic sinuses



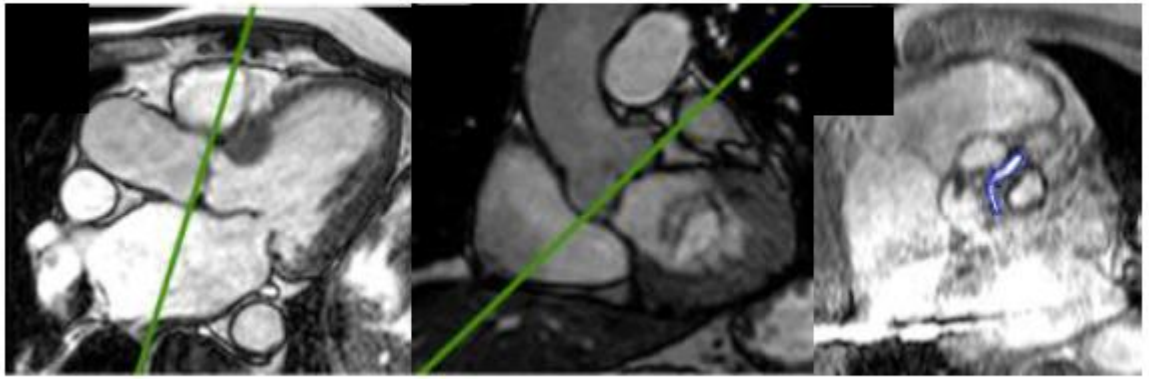
Aortic stenosis

Causes of AS

- Degenerative (most common with age)
- BAV (most common in age < 40yrs)
- Rarely rheumatic (in the developed world)

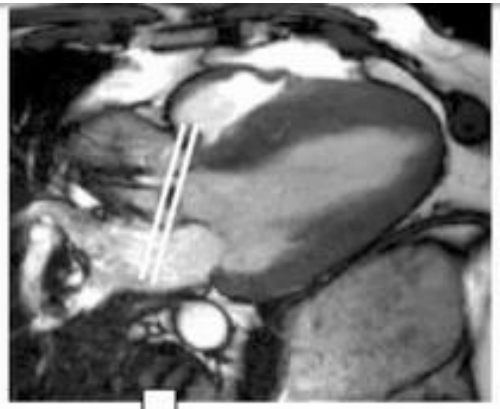
Quantification

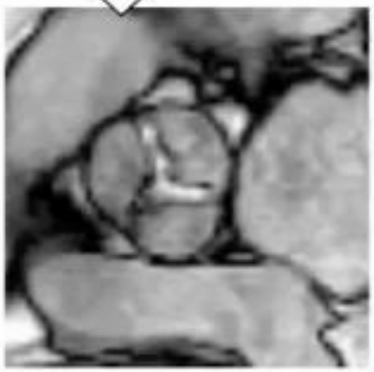
- AV orifice area planimetry (cm^2)
- Peak and mean velocity assessment (m/sec)



Aortic valve area planimetry: cine stack parallel to the annulus

Aortic stenosis





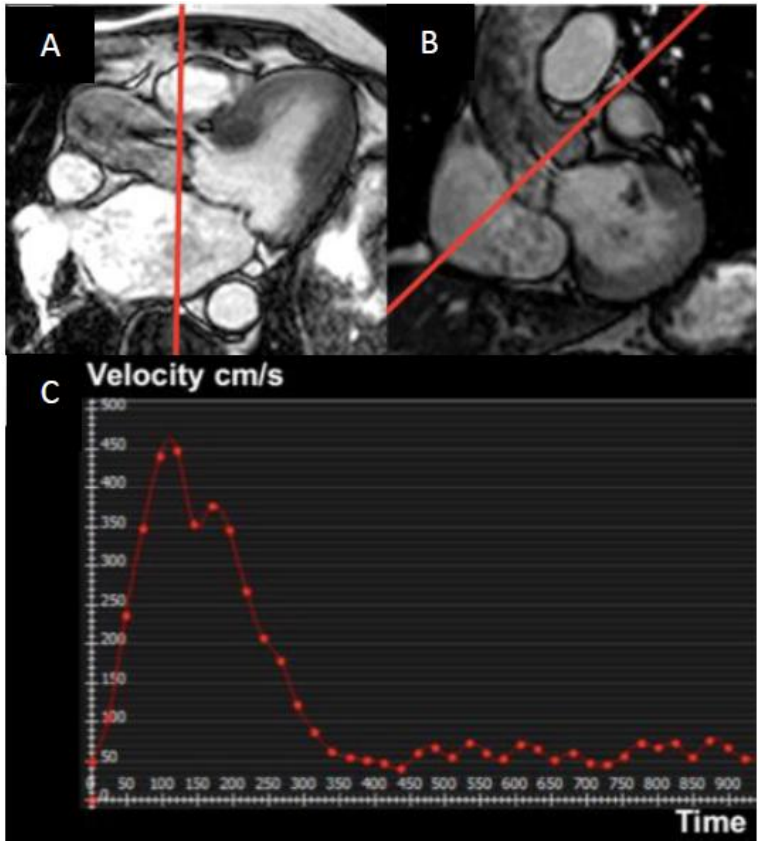
AVA by planimetry (good correlation with echo)

Peak velocity can be underestimated:

- Partial volume effects
- Lower temporal resolution
- ↓ accuracy at high velocities

- Multiple planes- a series of short-axis cuts of the valve, with direct visualisation of the valve orifice (minimal orifice area) and morphology.
- Thin slices (4-5 mm) with no gap, perpendicular to the valve.
- Standard is bSSFP sequence, but GRE sequence can be used.

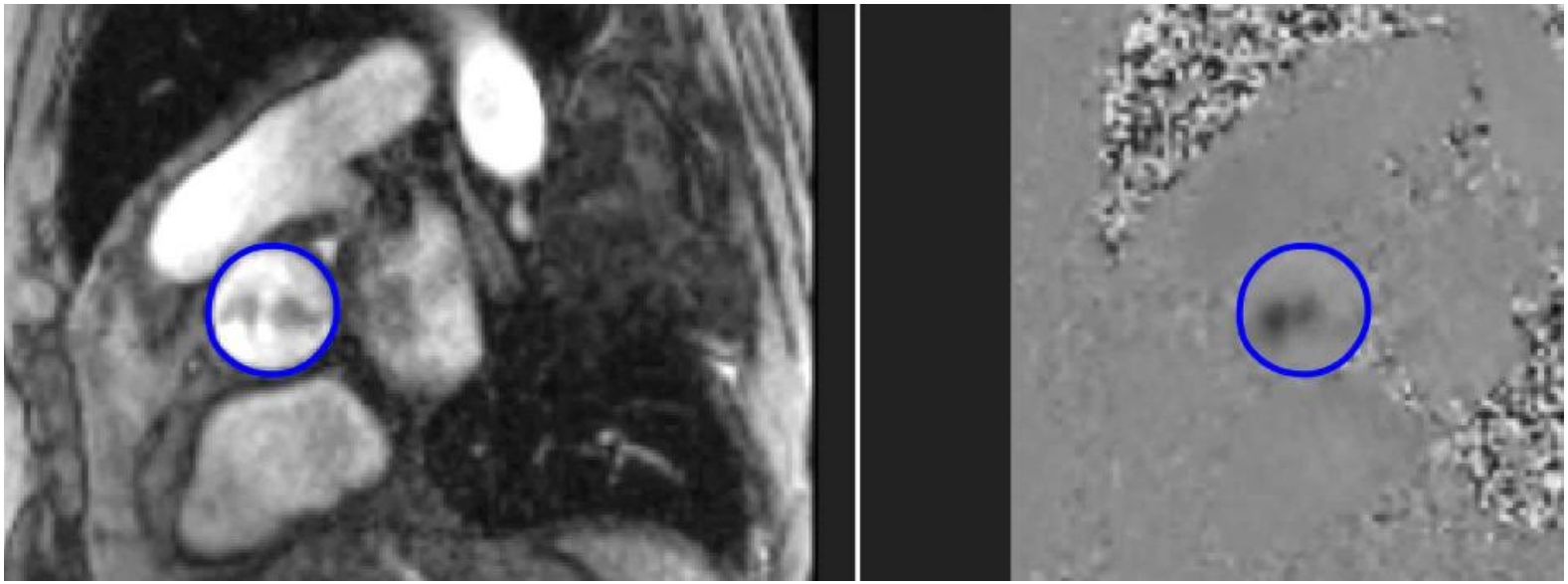
Aortic stenosis

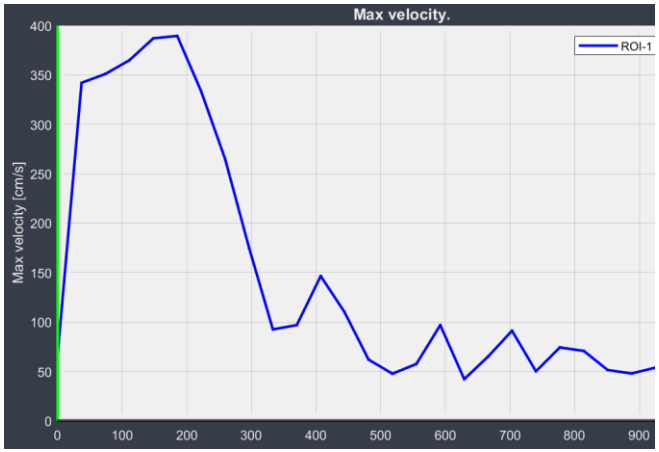


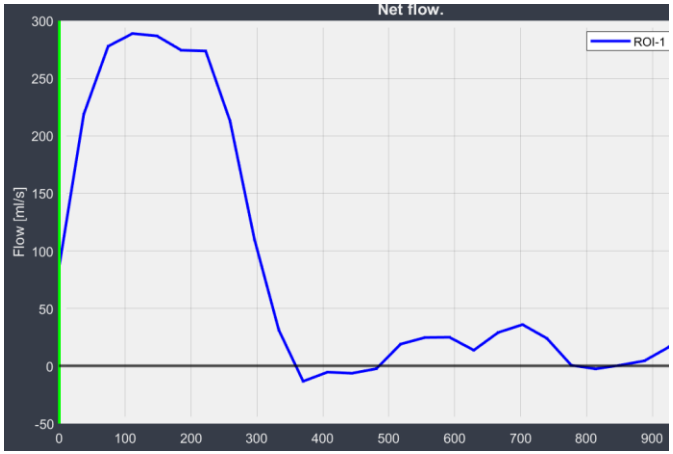
Maximal velocity assessment in a case of degenerative aortic stenosis: Projection plane in **3CH (A)** and **coronal LVOT (B)** orthogonal to the aortic stenosis jet. **Velocity time curve (C)** with a maximal velocity of 4.5 m/sec.

- Phase-contrast velocity mapping
- Free-breathing or breath-hold sequence
- Perpendicular to jet
- Adjust Venc to avoid aliasing
- Underestimates peak velocity CF Doppler

Aortic stenosis

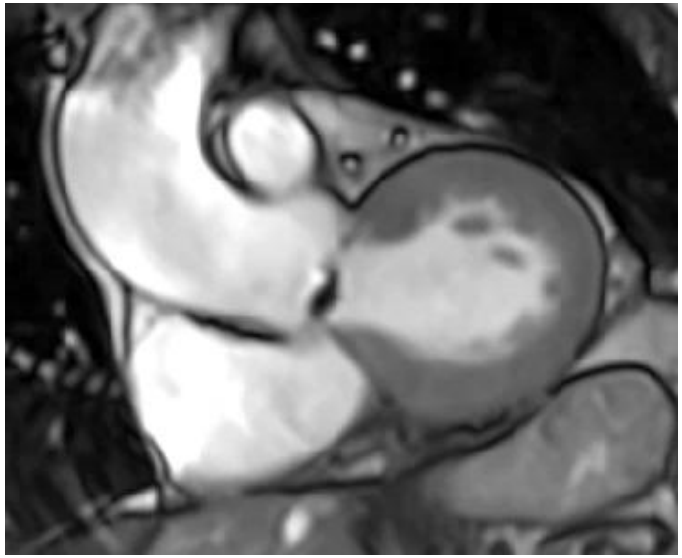


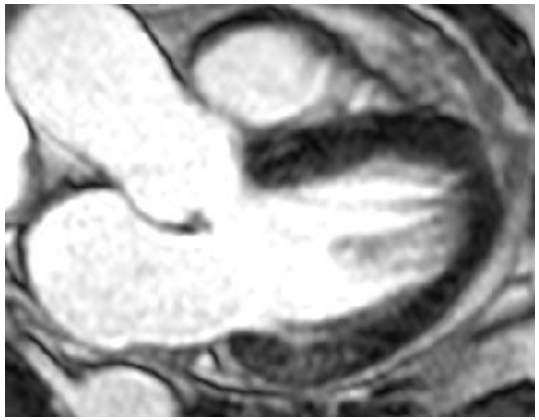


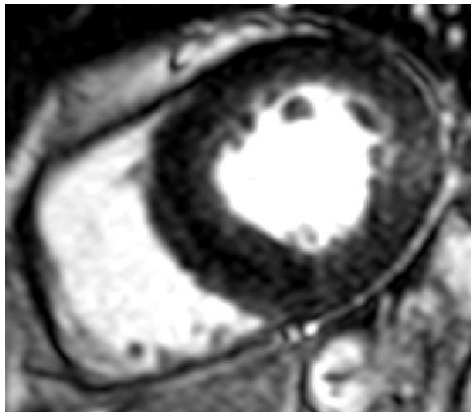


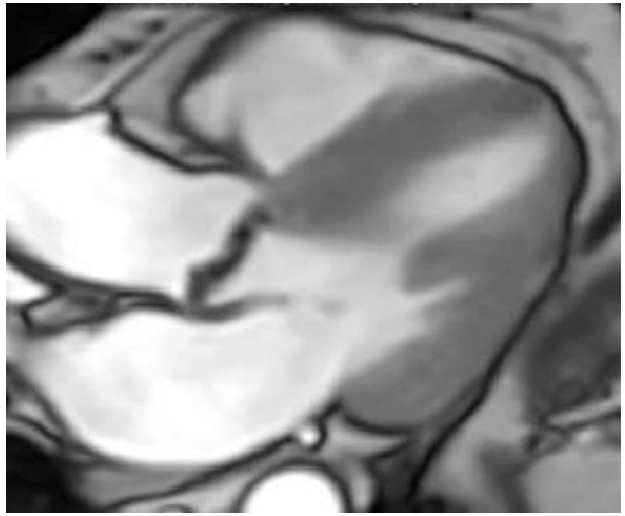
Aortic stenosis

- Calcification of the valve
- Dilatation of aorta
- LV hypertrophy
- LGE





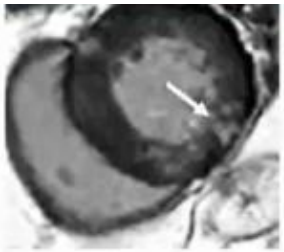




AS and LGE

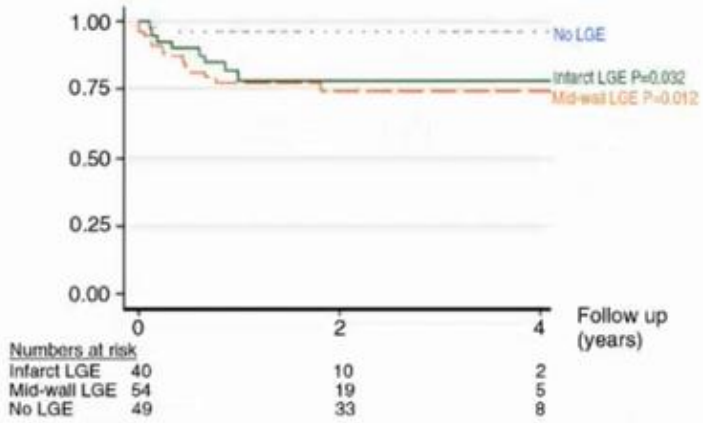
Cardiac mortality in 143 patients with moderate or severe AS

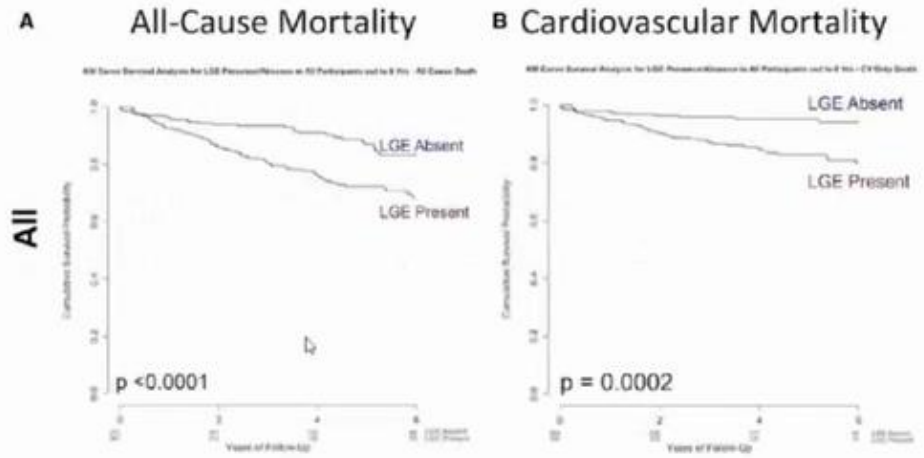
Dweck et al, *J Am Coll Cardiol* 2011; 58:1271–9



Mortality in 674 patients undergoing AVR or TAVI – AS700 consortium

Musa et al, *Circulation* 2018; 138:1935–47



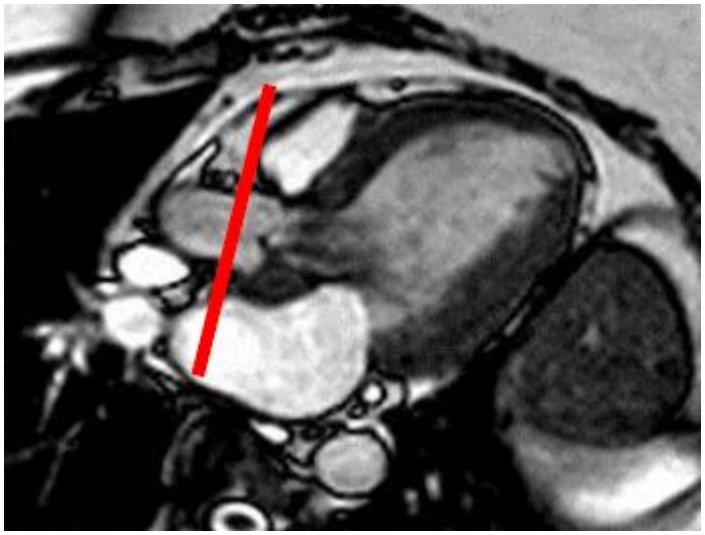


Aortic regurgitation

Causes of AR

- Degenerative (most common with age)
- Dilated aortic root (e.g. hypertension, Marfan syndrome, inflammatory/aortitis)
- BAV (bicuspid aortic valve)
- Infective endocarditis or rheumatic fever

Aortic dissection



Severely dilated, eccentric hypertrophied LV

Jet direction

- **Central** (aortic sinus dilatation, symmetric tethering)
- **Eccentric** (prolapse, flail, asymmetric tethering, BAV)

Aortic assessment is likely to identify the cause of AR

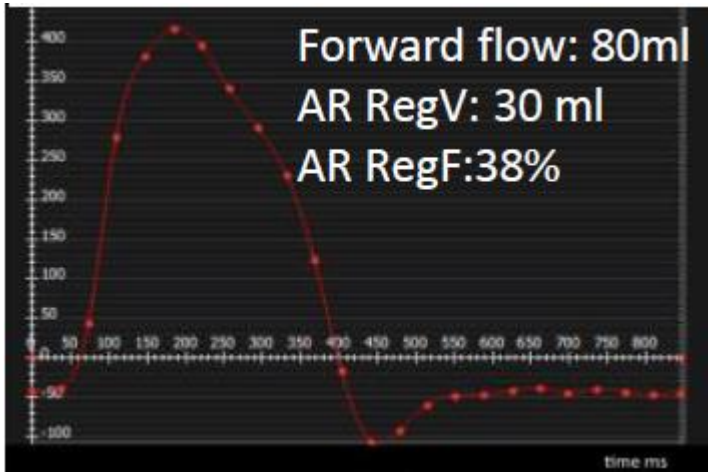
RegF of >33% predict symptom progression and indications for corrective surgery*

Consider flow measurement in descending aorta to detect (holo)diastolic backflow

Aortic regurgitation

Quantification

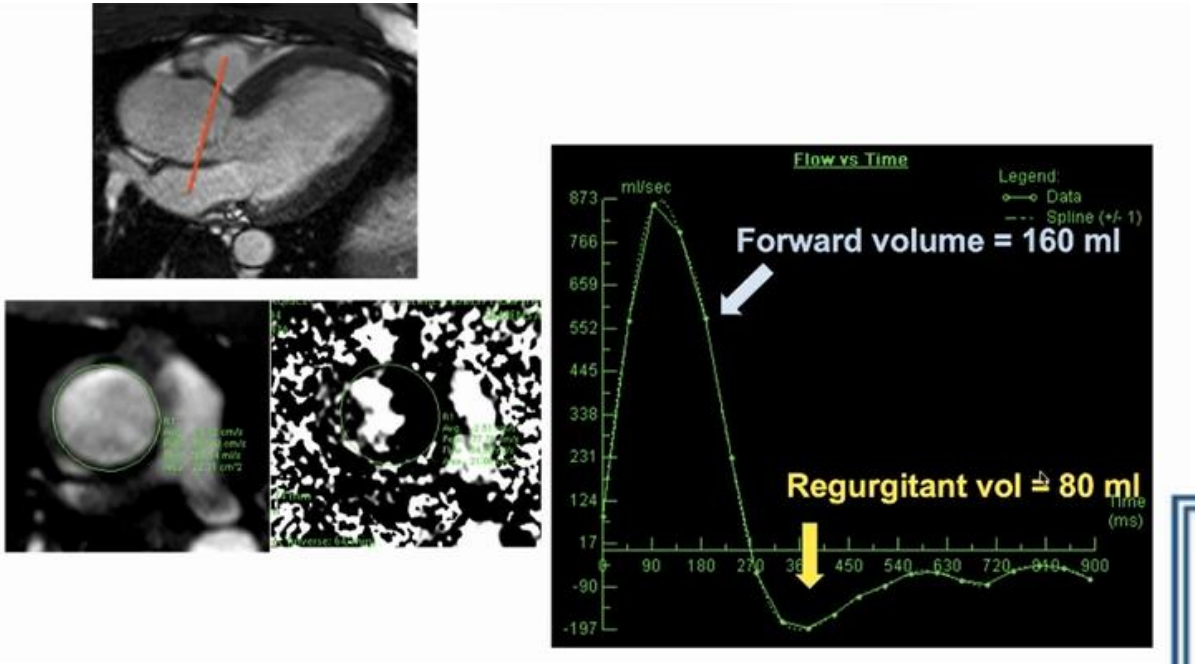
- **Regurgitant Volume (ml):** Through-plane phase contrast (ml)
- **Regurgitant Volume (ml) alternatively = cine LV SV– cine RV SV** (only valid in single valve disease)
- **Regurgitant Fraction (%)** = RegV / Forward flow x 100

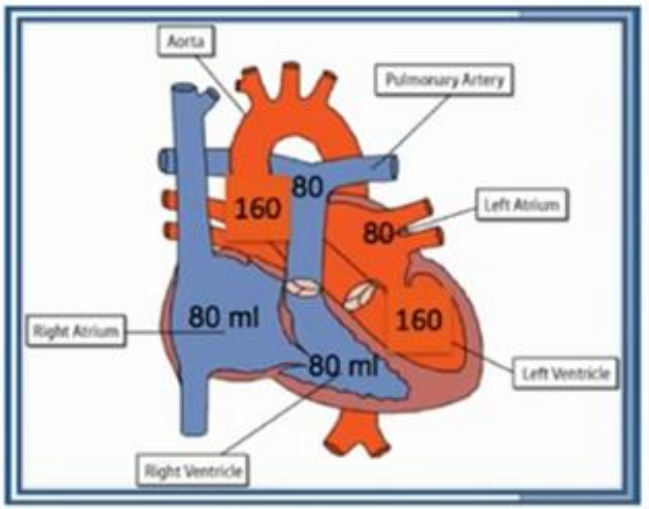


Aortic Regurgitant Volume =

1. Direct measurement of regurgitant flow
2. Indirect methods:
   - LVOT FF - Pulmonic Net
   - LVSV - RVSV

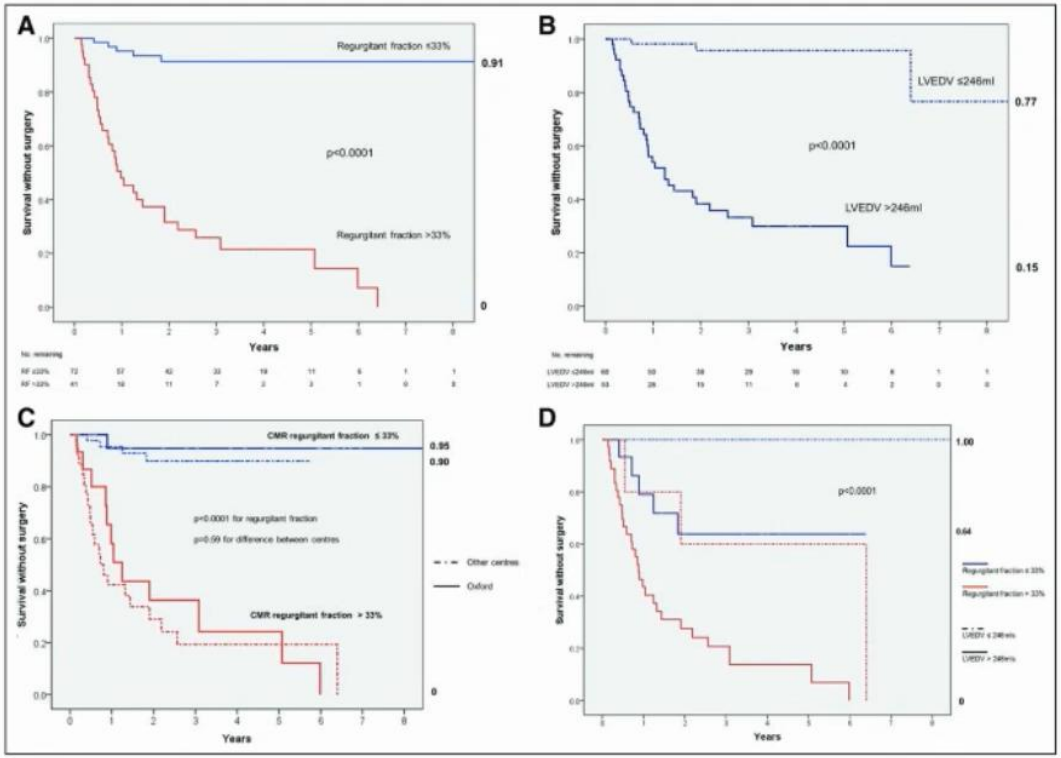
Aortic regurgitation



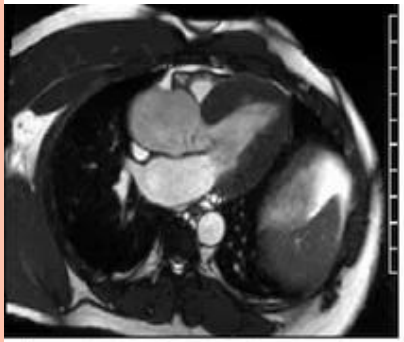


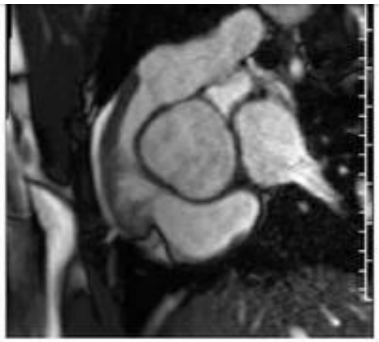
Aortic regurgitation

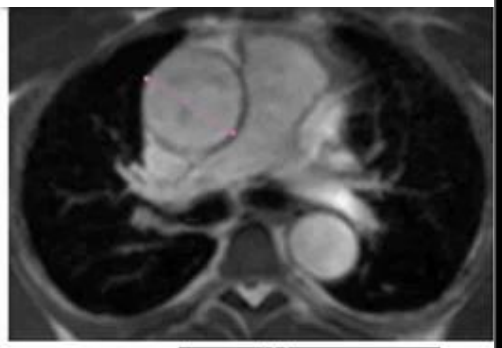
CMR-derived left ventricular end-diastolic volume >246 mL predicted survival without surgery but the combination of this measure with regurgitant fraction provided the best discriminatory power

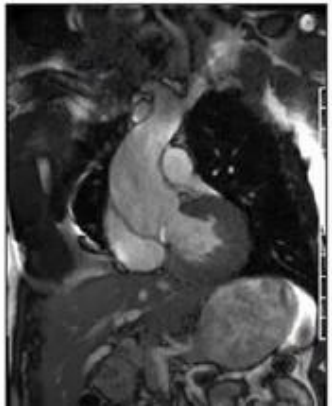


Bicuspid Aortic Valve



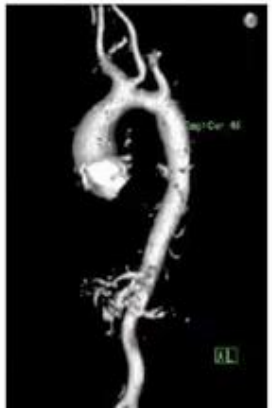


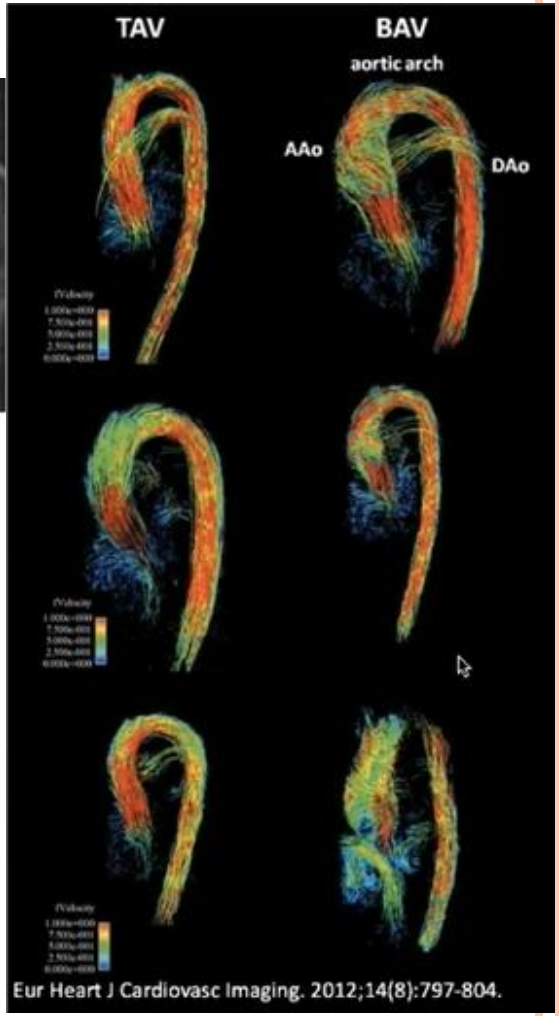




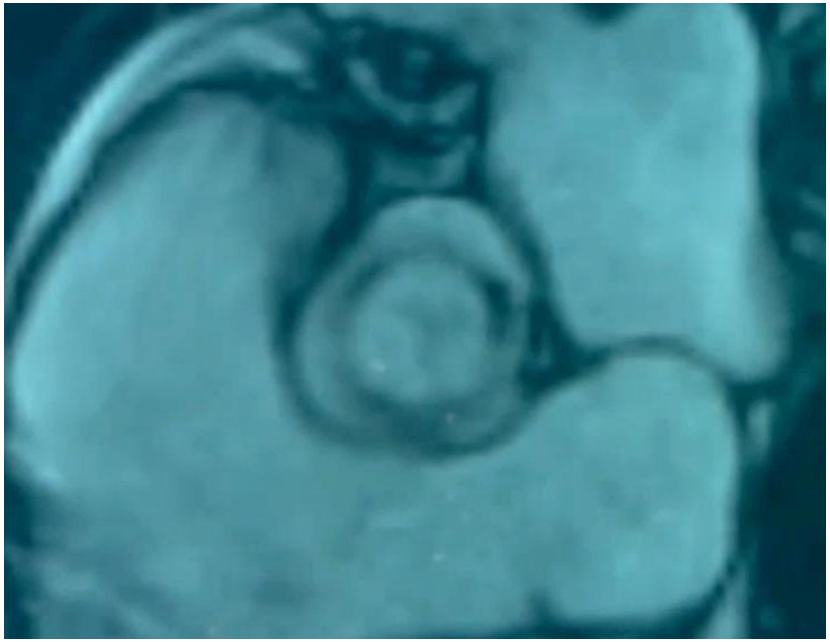
39 yr old with SOB:

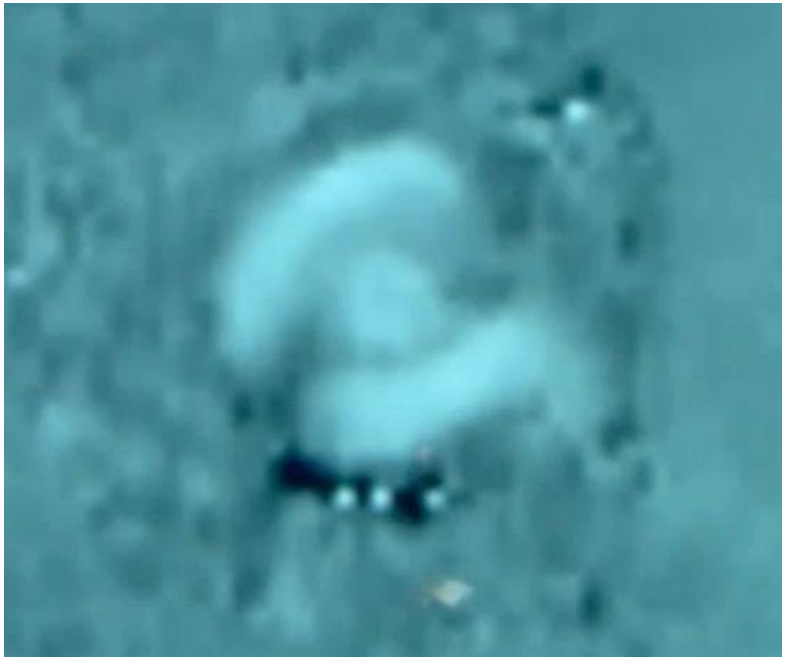
-Moderately enlarged LA and LV
-Normal LV and RV systolic fxn
-Bicuspid aortic valve with severe AI(RV 80 ml, RF 50%)
-Severely dilated aortic root (5.3 cm at the sinus)
- Moderately dilated ascending aorta (5.2 cm)



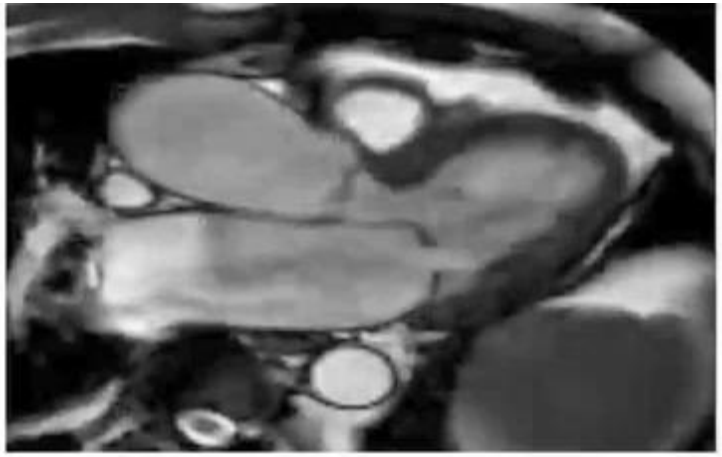


Prosthetic AV

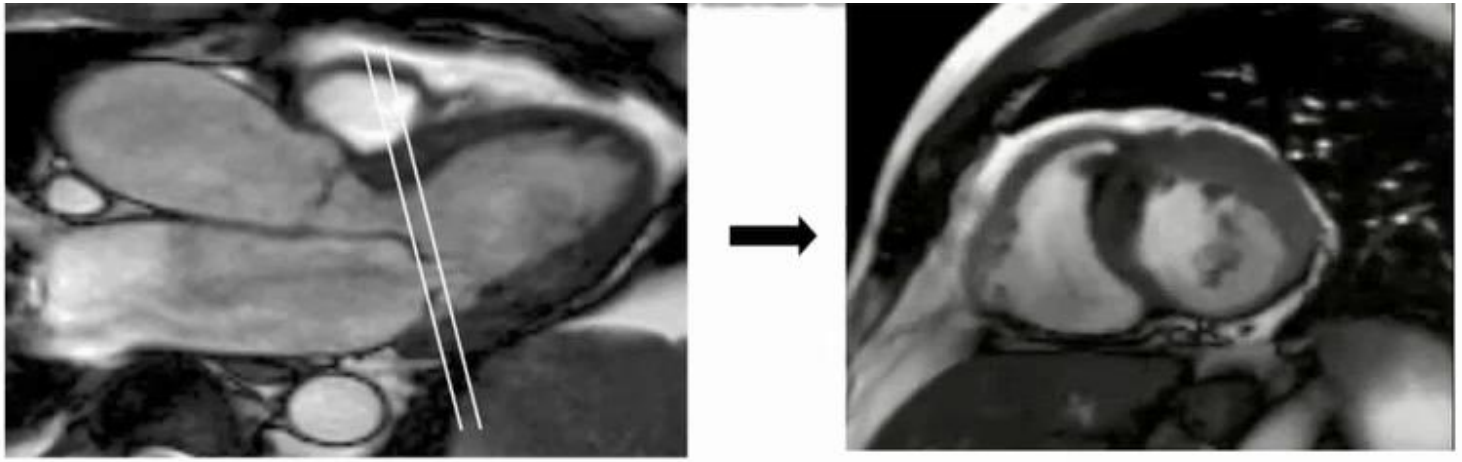




Mitral stenosis



Valve morphology/ aetiology
Valve area (planimetry)
LV volume/function
LA volume



Mitral stenosis

Causes

- Rheumatic valve disease
- Systemic lupus erythematous
- Degenerative
- Congenital

Quantification

- MV orifice area planimetry (cm^2)
- MV pressure gradients unreliable because of motion of MV apparatus

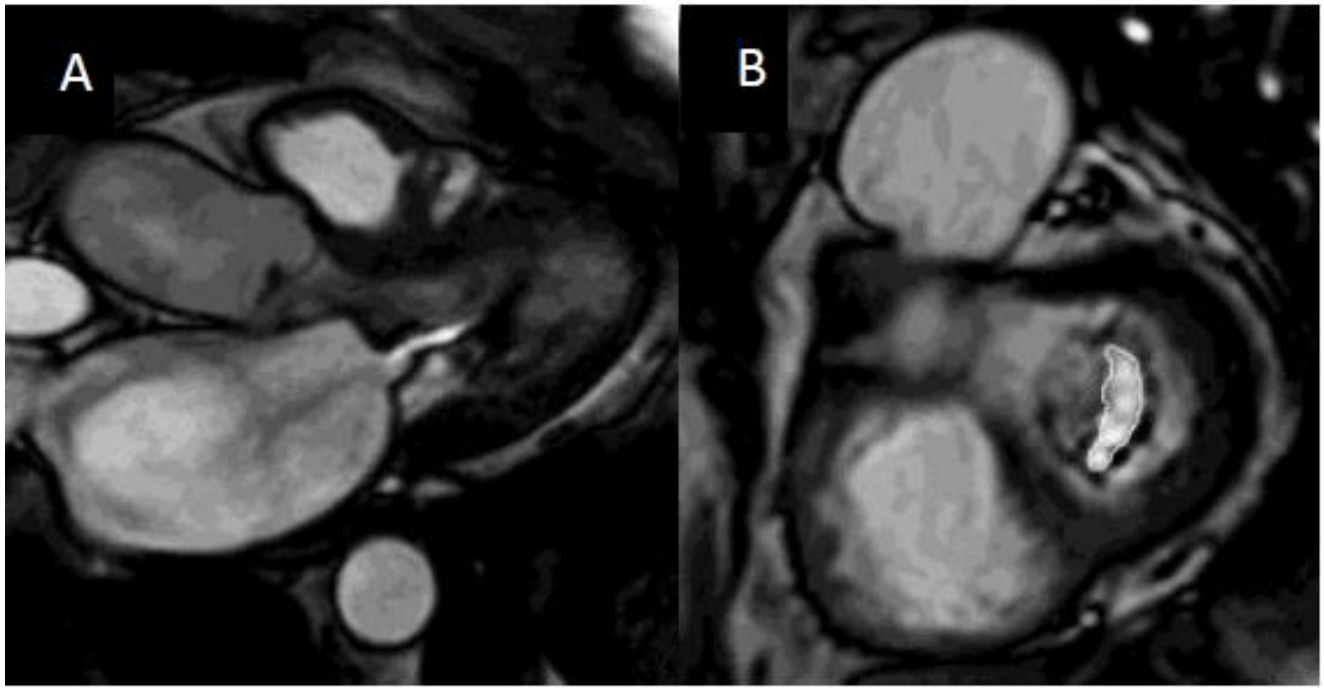
Rheumatic MS: fusion and thickening at the tips of the leaflets results in 'doming' of the MV leaflets ('hockey-stick').

Degenerative MS: Annular thickening and calcification affect the base and body of MV leaflet while sparing the tips

Progressive LA dilatation is marker of increased LV filling pressure and worsening MS

Advanced MS has adverse impact on RV systolic function (secondary to pulmonary hypertension

Rheumatic MS



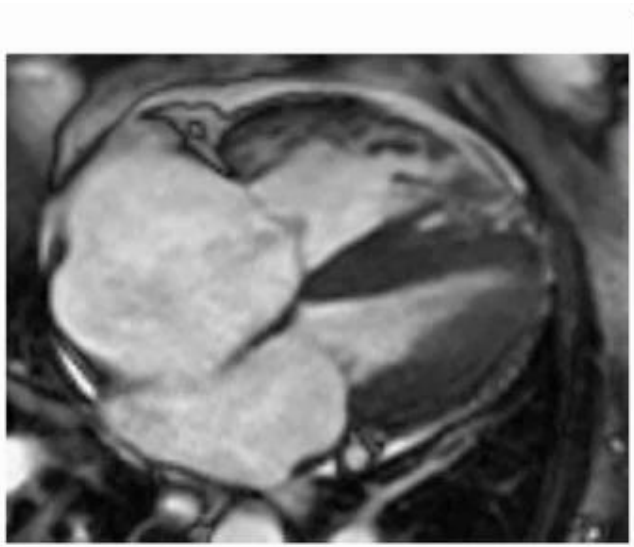
Cine 3CH (A): Restrictive opening of the thickened MV leaflets particular the posterior one. The classical sign of 'hockey stick' can be seen for the anterior MV leaflet.

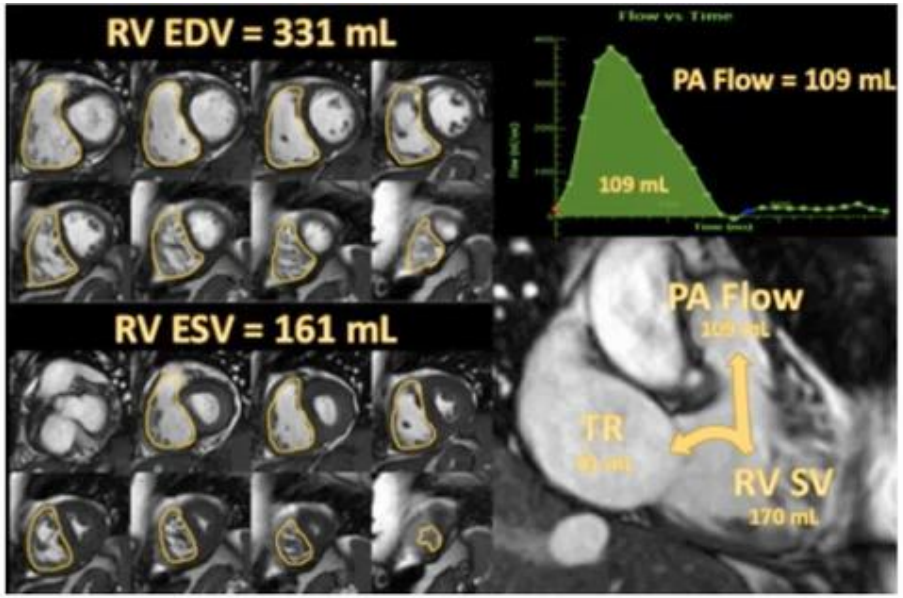
Cine SA (B): MVA planimetry

Tricuspid regurgitation

- Potential reasons for TR neglect (by echo):
  - Long indolent period, even for severe TR
  - Symptoms are non-specific and confused
  - Reliance on TR jet area by color Doppler
  - Consistent under-coding of TR severity and often of TV annular size, RV size and function
  - Variability of TR severity with diuresis / preload status

Tricuspid regurgitation



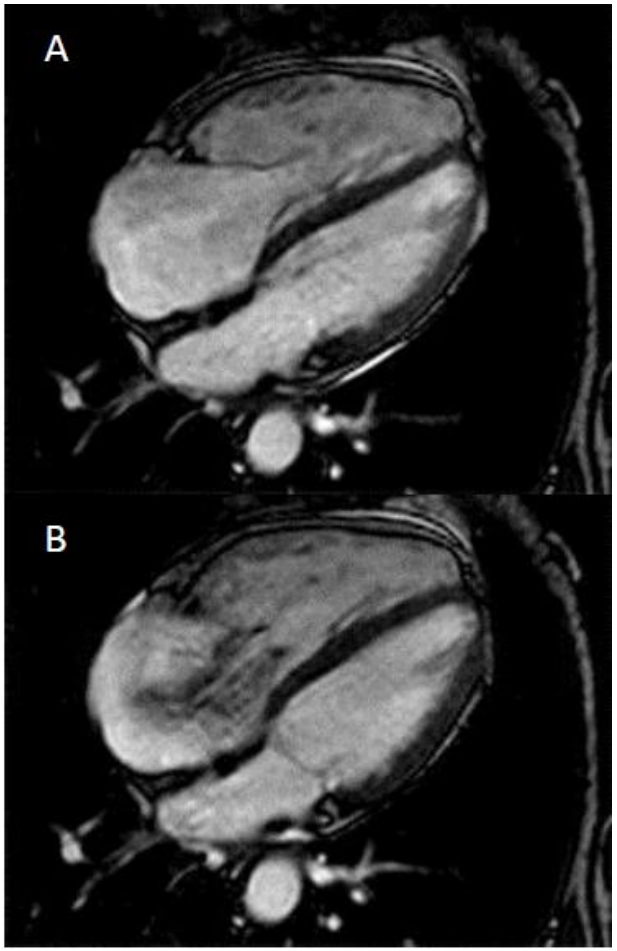


Through-plane TV cine can show the leaflets, and identify coaptation defects, regurgitant orifices or valve stenosis.

SV from phase contrast VENC above pulmonary valve – LV SV
Alternatively RV SV – LV SV

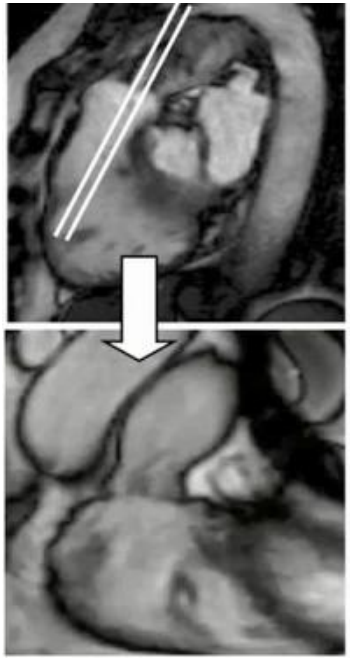
TR usually secondary to RV dilatation. Accurate quantification of RV volumes (SAX and axial cine stacks).

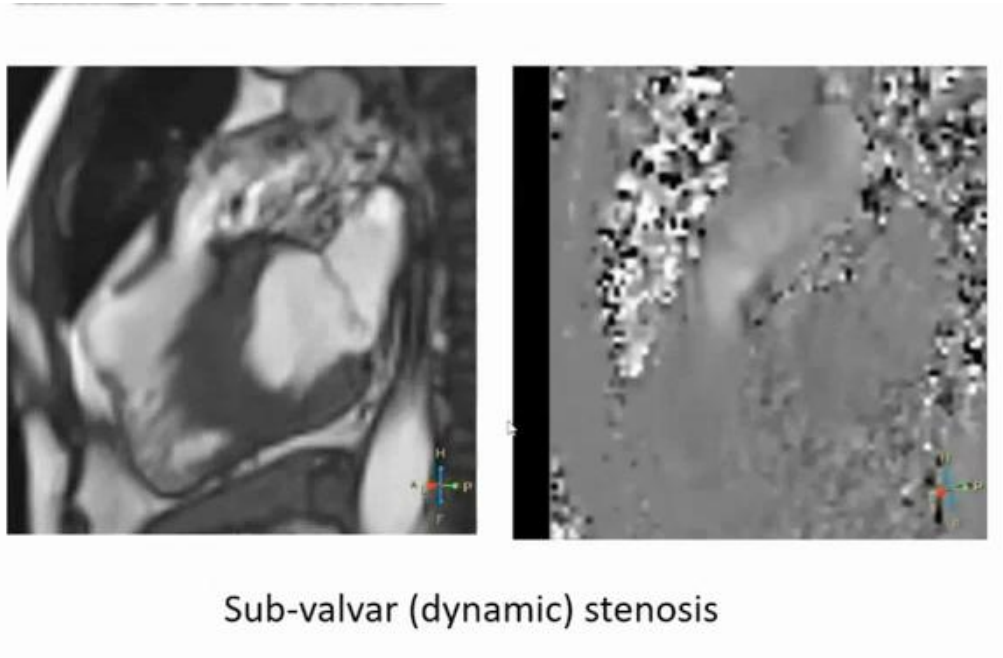
Carcinoid heart disease



Cine 4CH in diastole **(A)** and systole **(B)**
The tricuspid leaflets are thickened and severely reduced in their mobility leading to free tricuspid regurgitation

Pulmonary stenosis





Sub-valvar (dynamic) stenosis

Pulmonary regurgitation	• Regurgitation volume/fraction from phase contrast VENC above pulmonary valve • RVSV–LVSV

Conclusions

- CMR is a complimentary imaging tool in VHD
- Allows comprehensive assessment of the valve, myocardium and surrounding structures, with no ionizing radiation
  - Consider if TTE is suboptimal
  - Borderline cases
  - When have complex patients / need multiple answers
  - When the echo and clinical data do not fit!

Future role as an imaging biomarker for assessing therapeutic interventions and timing of intervention

Thank you