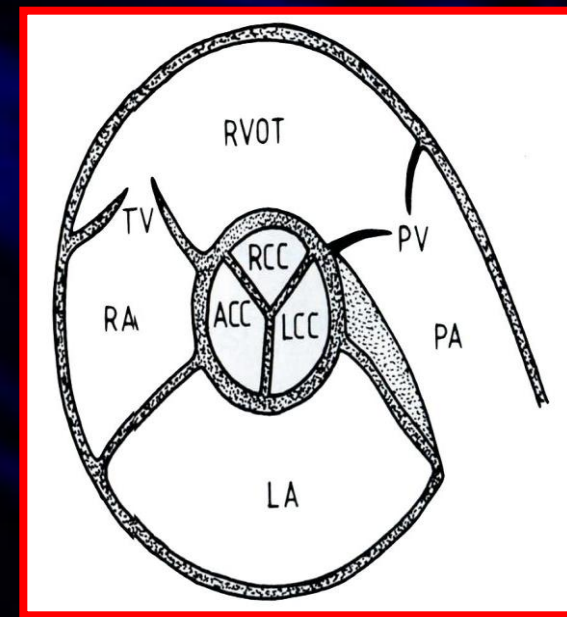
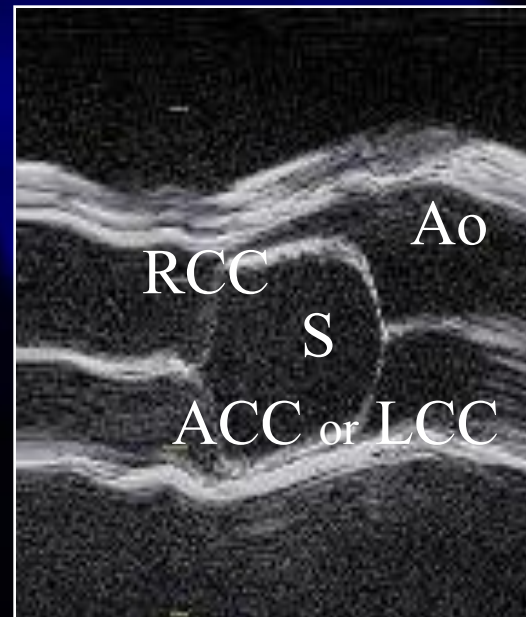
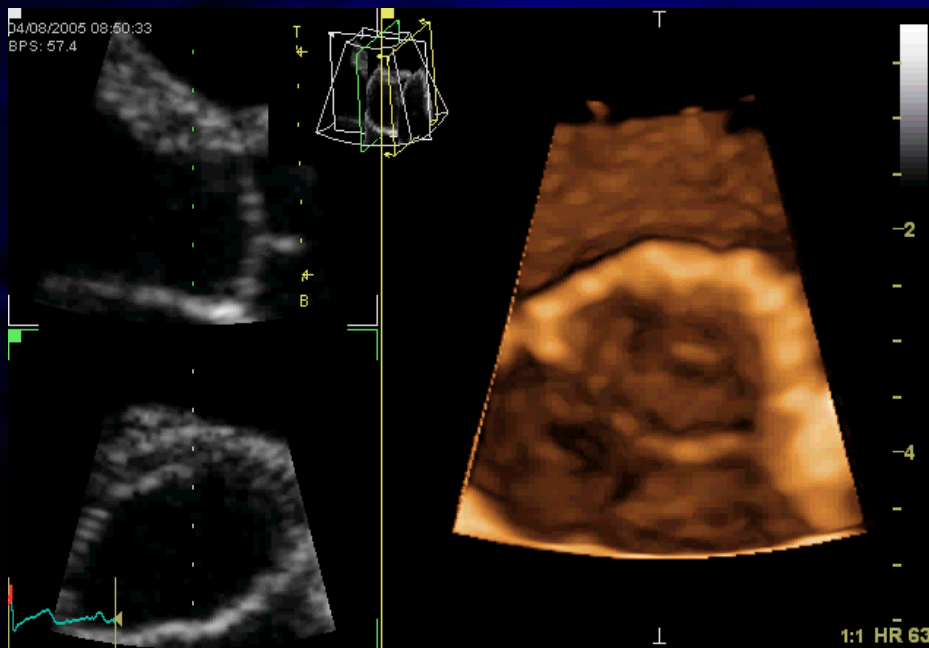
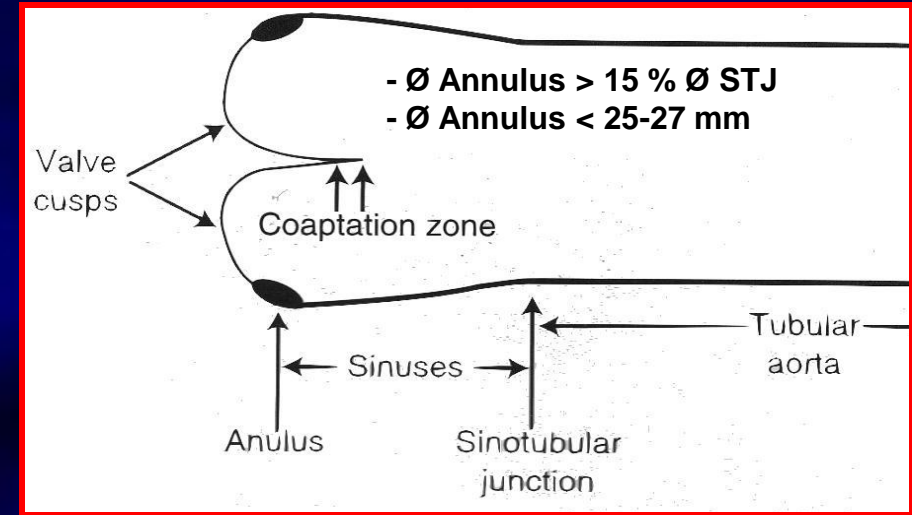
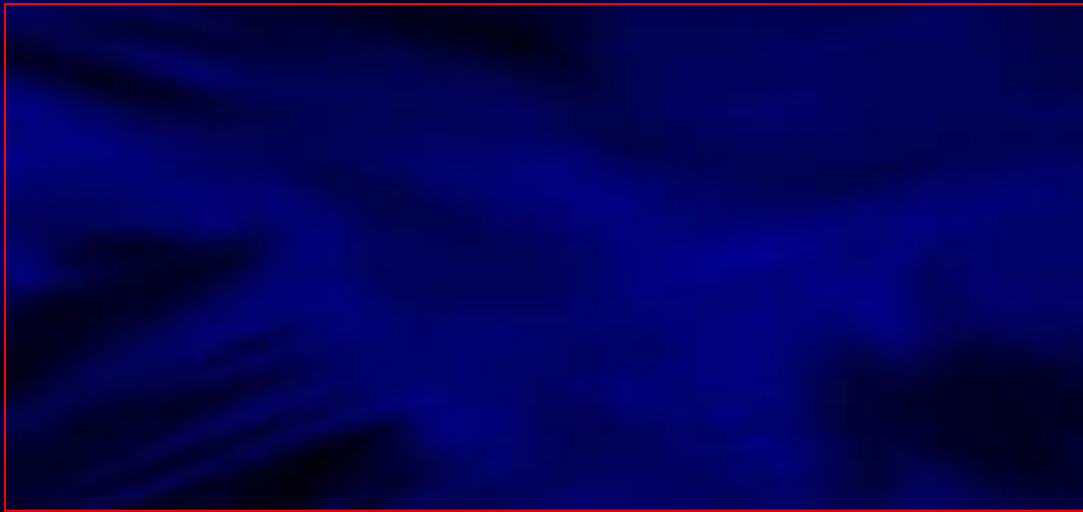


Aortic Regurgitation

Prof. Patrizio LANCELLOTTI , MD, PhD
University of Liège, CHU Sart Tilman, BELGIUM

Anatomical structure of the aortic valve



Etiology

ACUTE AR

1. Infective endocarditis
2. Acute rheumatic fever
3. Prosthetic valve dysfunction
4. Aortic dissection
5. Trauma
6. Systemic hypertension

CHRONIC AR

1. Idiopathic aortic root dilation
2. Infective endocarditis
3. Bicuspid aortic valve
4. Rheumatic
5. **Congenital lesions:** AS, VSD
6. Connective tissue disease :
Marfan's syndrome, Ehlers-Danlos, Osteogenesis imperfecta
7. **Autoimmune diseases:** SLE, Ankylosing spondylitis, Reiter's syndrome
8. **Aortitis :** Takayasu's aortitis, Syphilitic

VALVE ANALYSIS

Etiology



Lesions



Dysfunction

The Cause of
Valve Disease

The Result of the
Disease Process

The Result of
the Lesions

Dysfunction

Type I

Normal leaflet motion

Type II

Excessive leaflet motion

Type III

Reduced leaflet motion

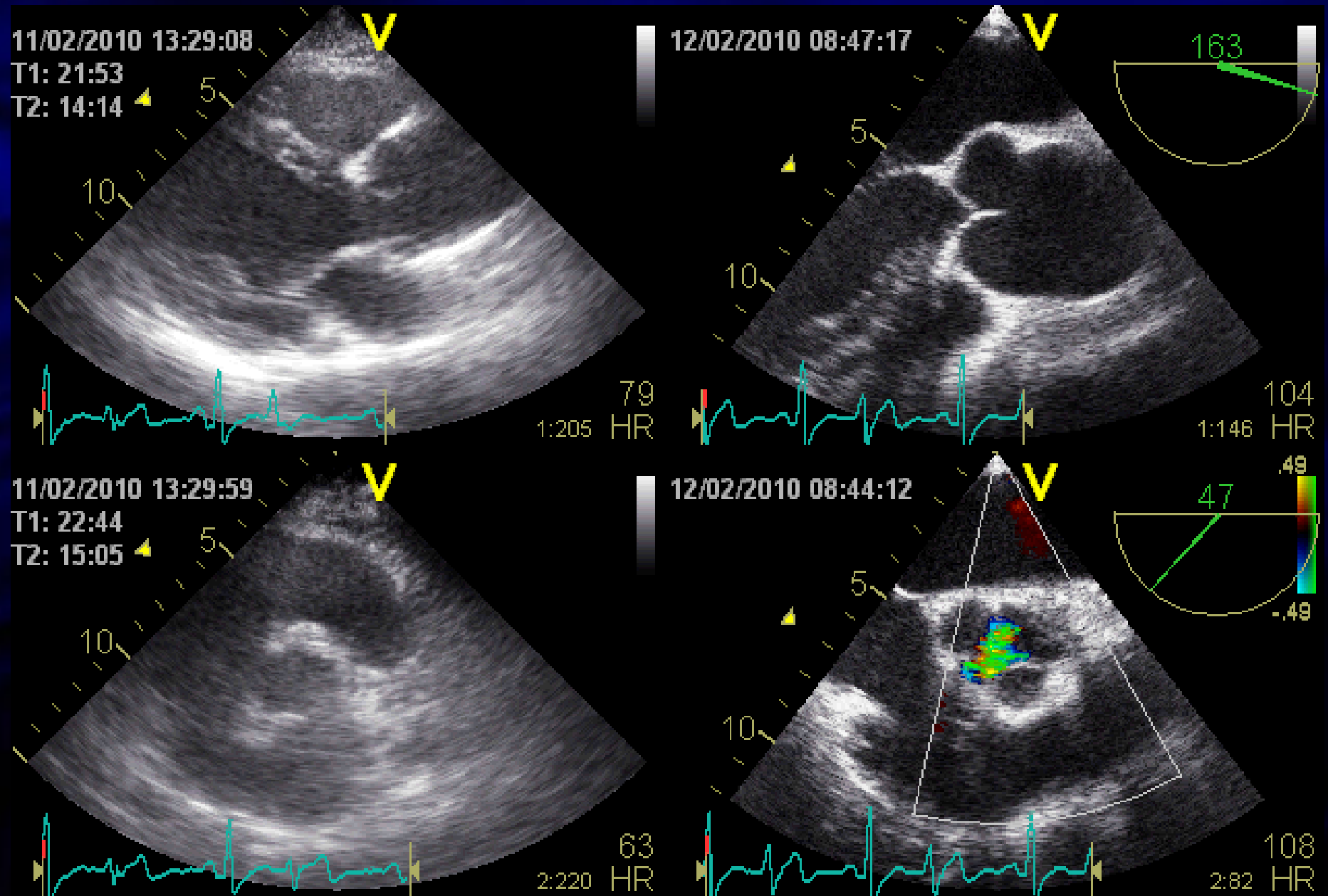
Lesions

- Enlargement of the aortic root
- Leaflet perforation

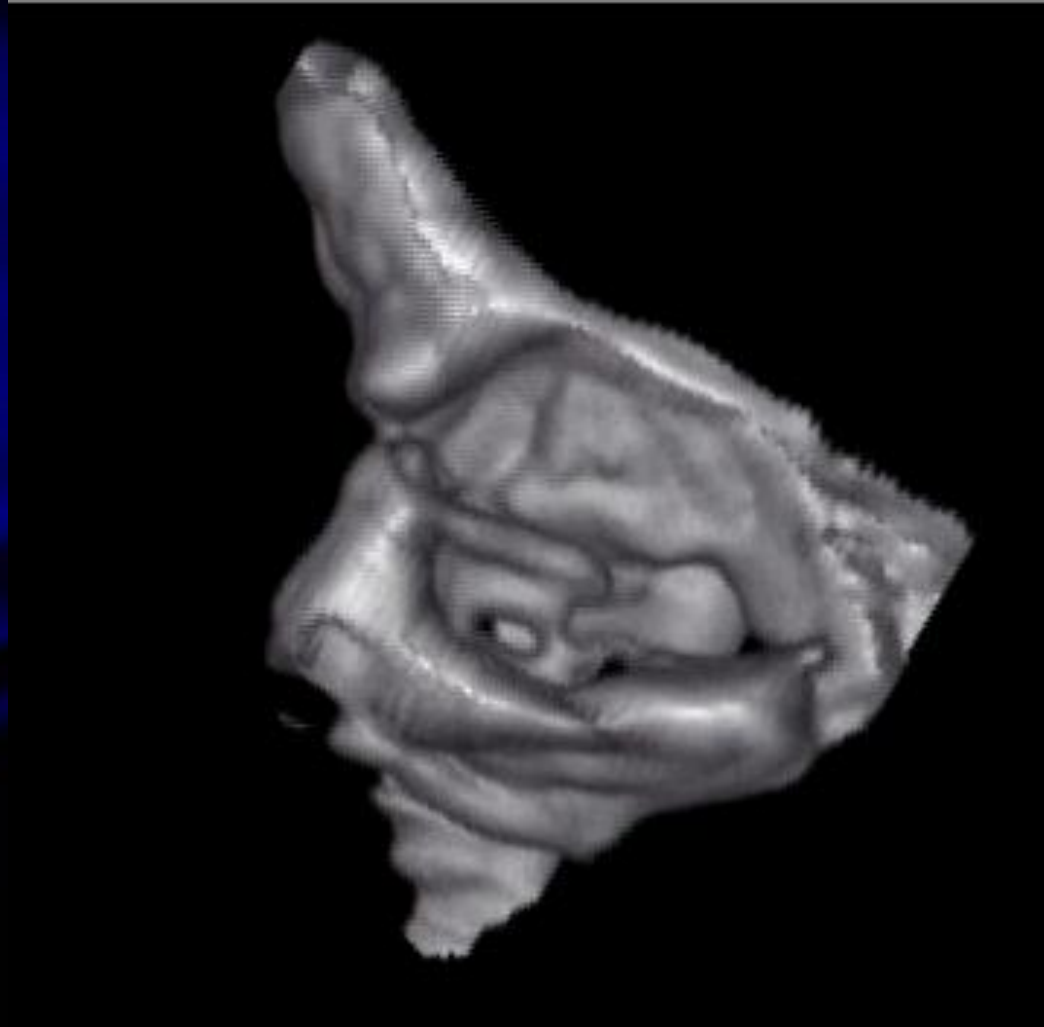
- Cusp prolapse or fenestration

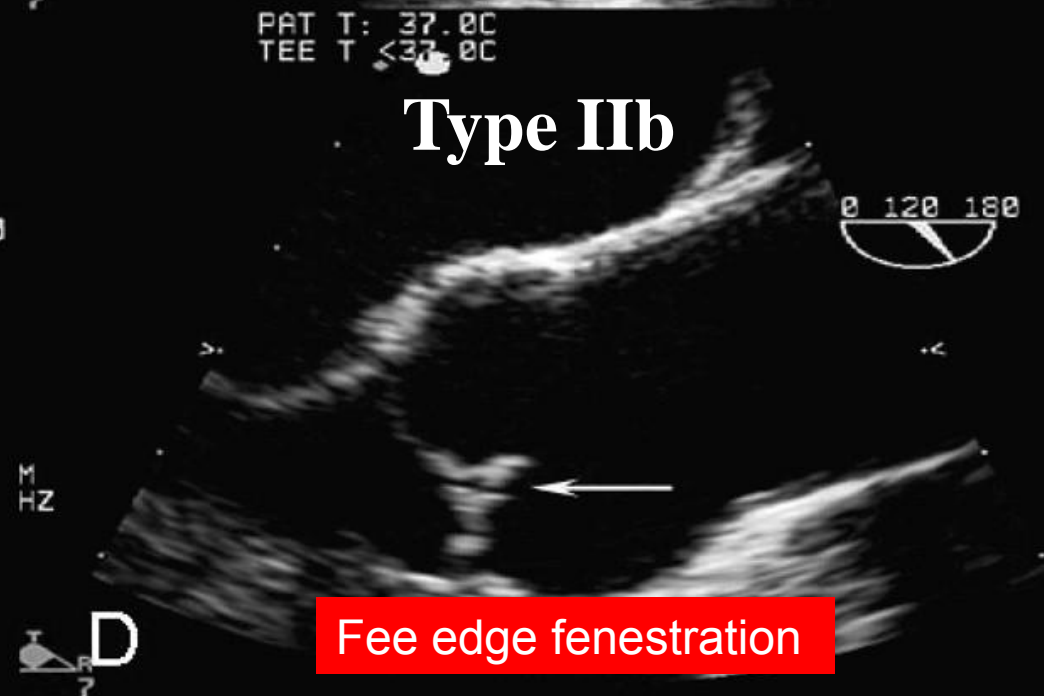
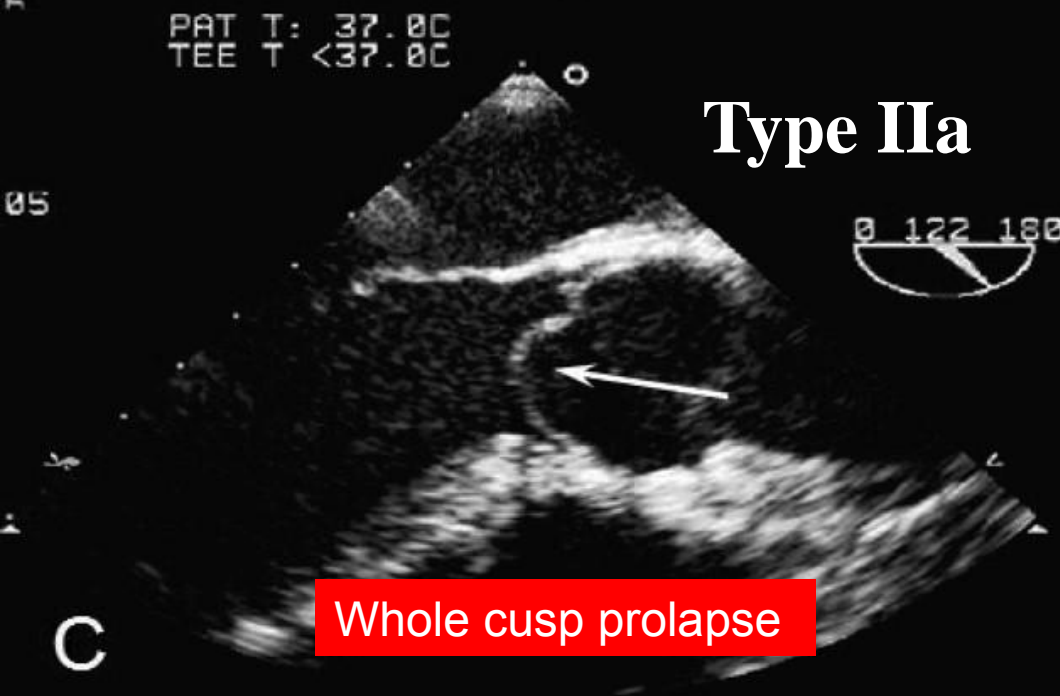
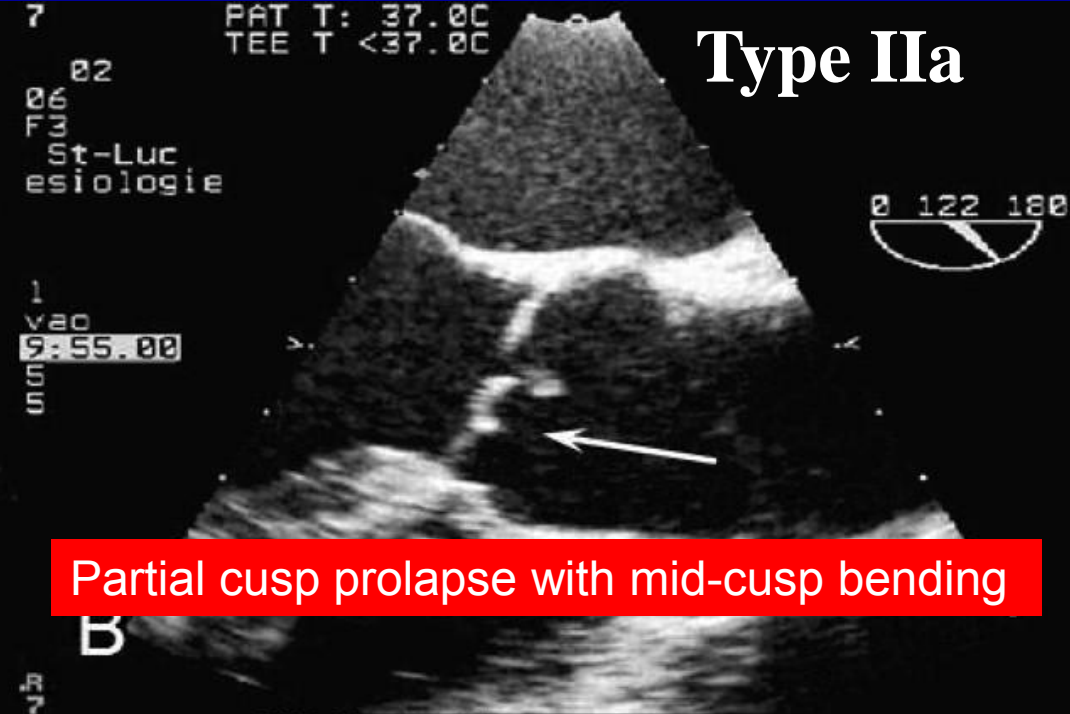
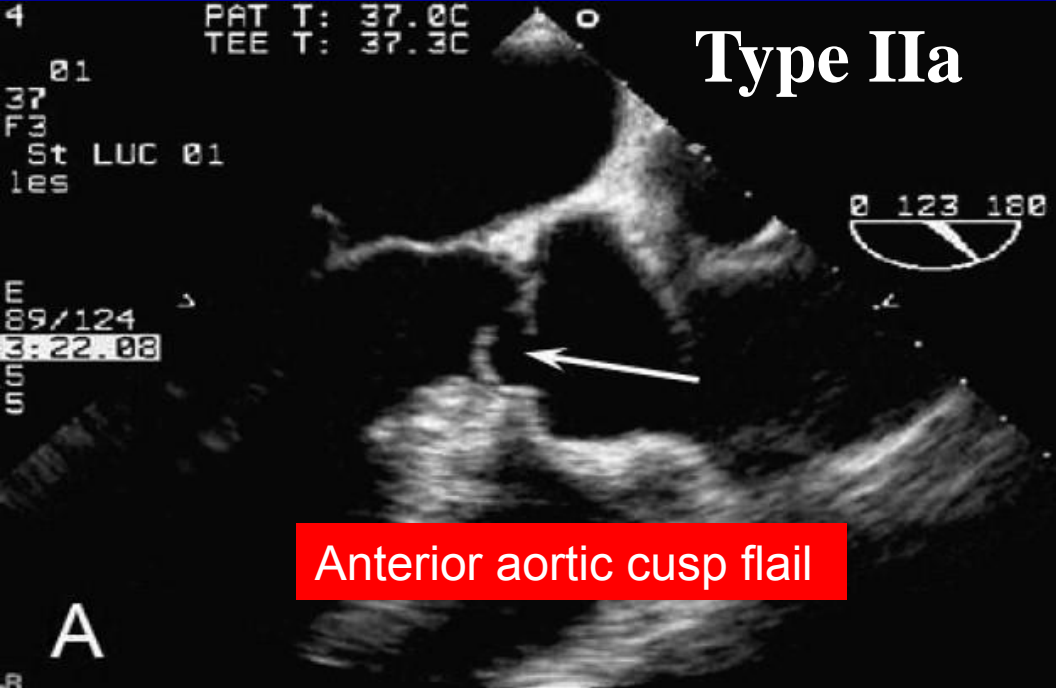
- Poor cusp tissue quality or quantity
(Thickening, Calcifications, Fusion,...)

Type I

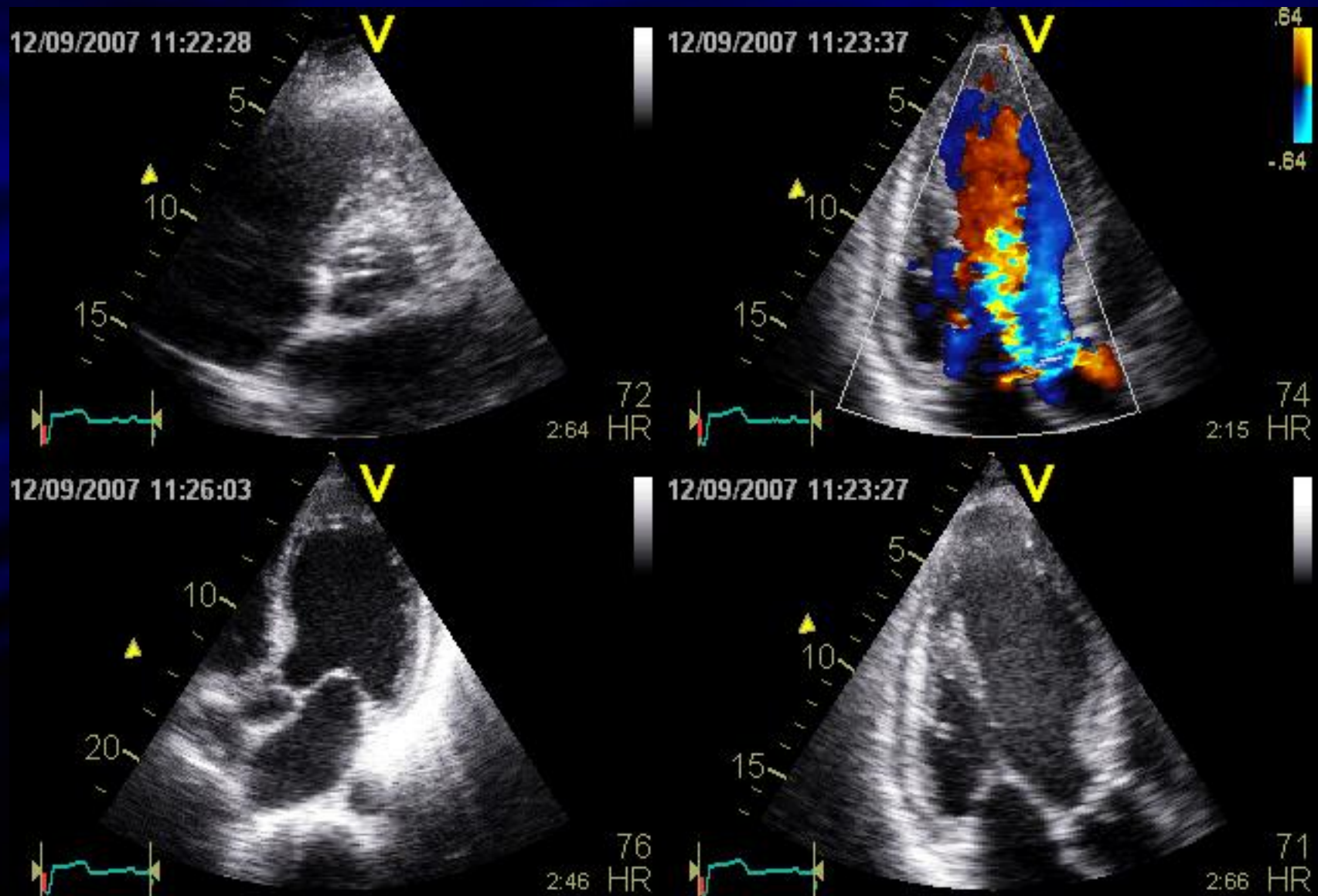


Type I

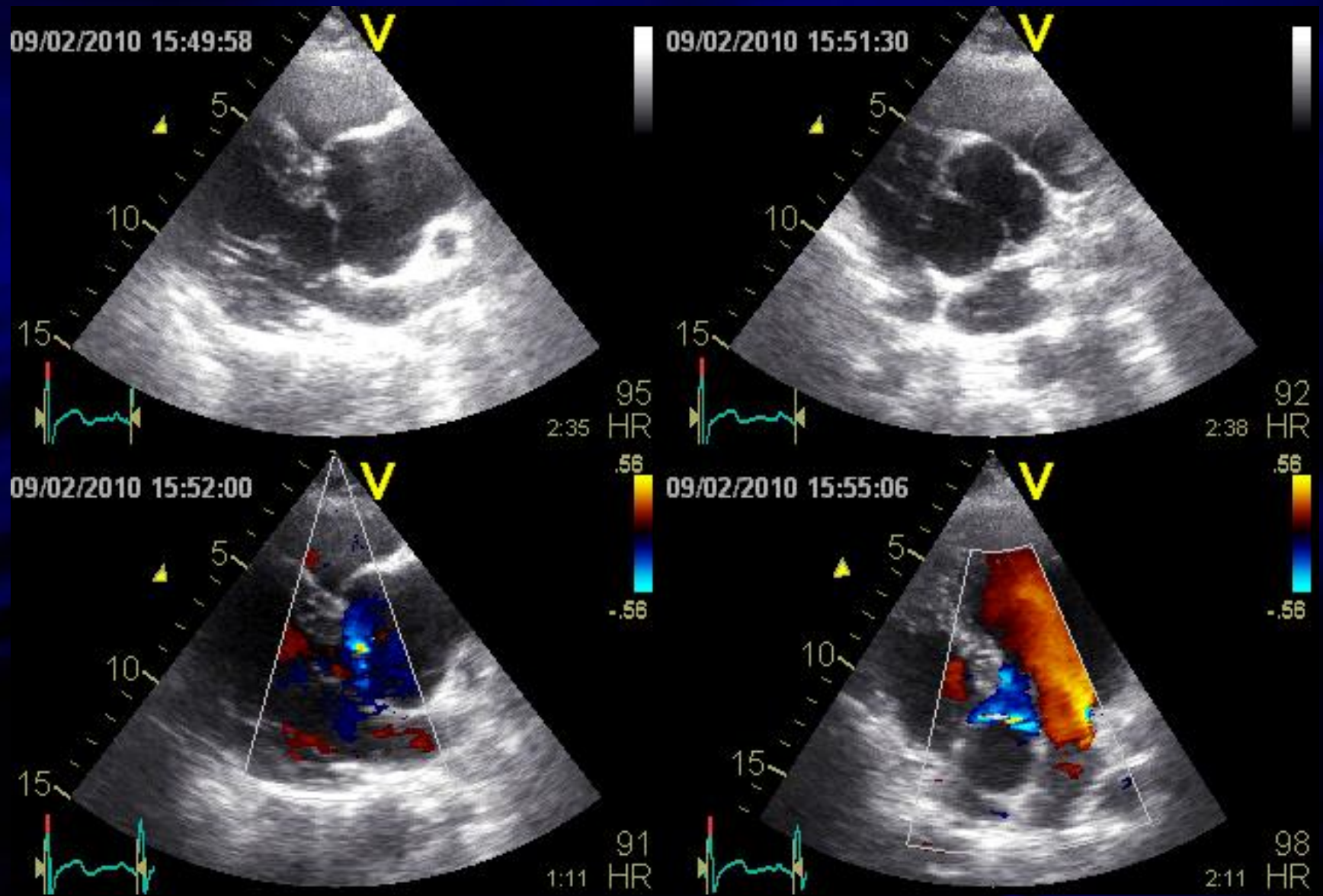


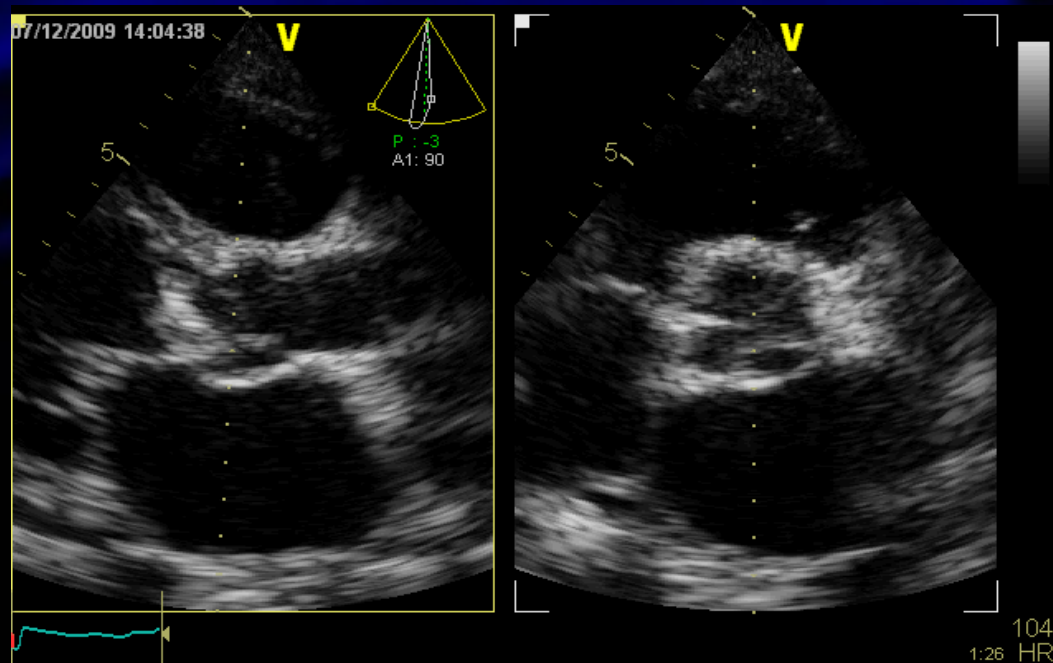
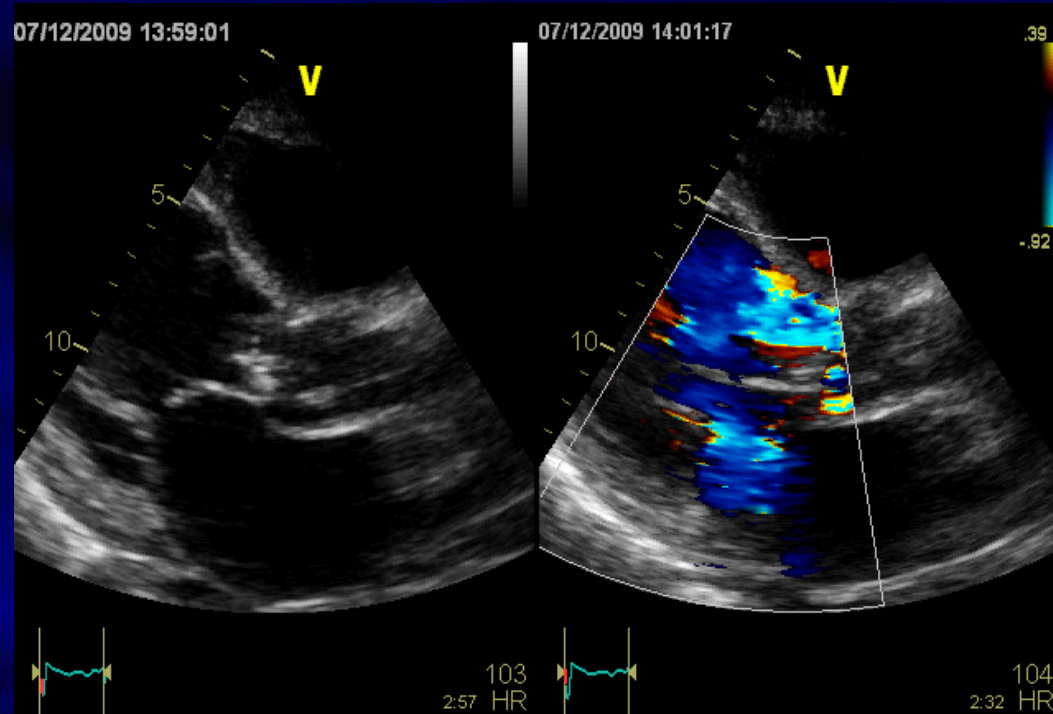


Type IIa: Tricuspid leaflet + Normal Aorta



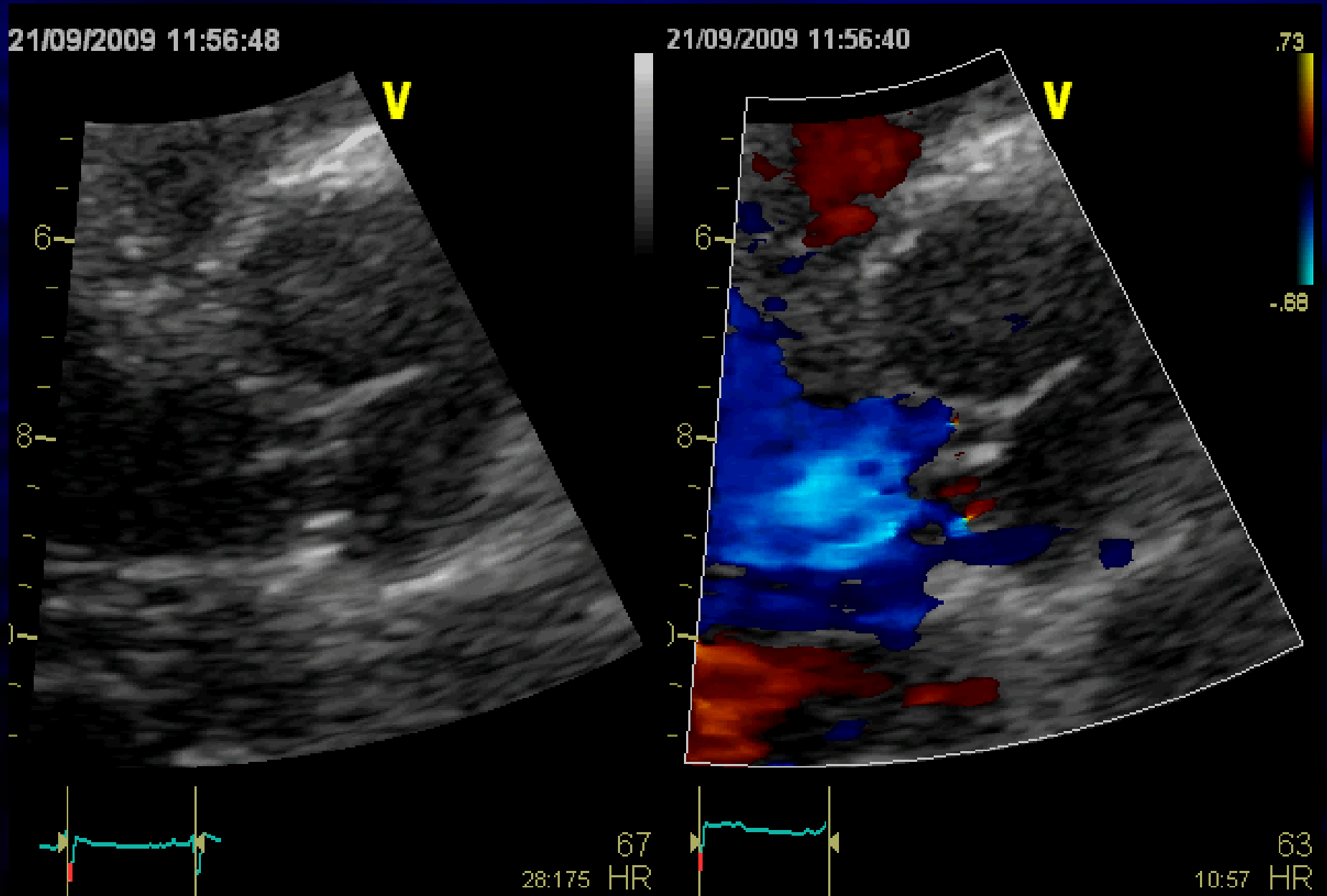
Type IIa: Bicuspid leaflet + Aortic Dilatation



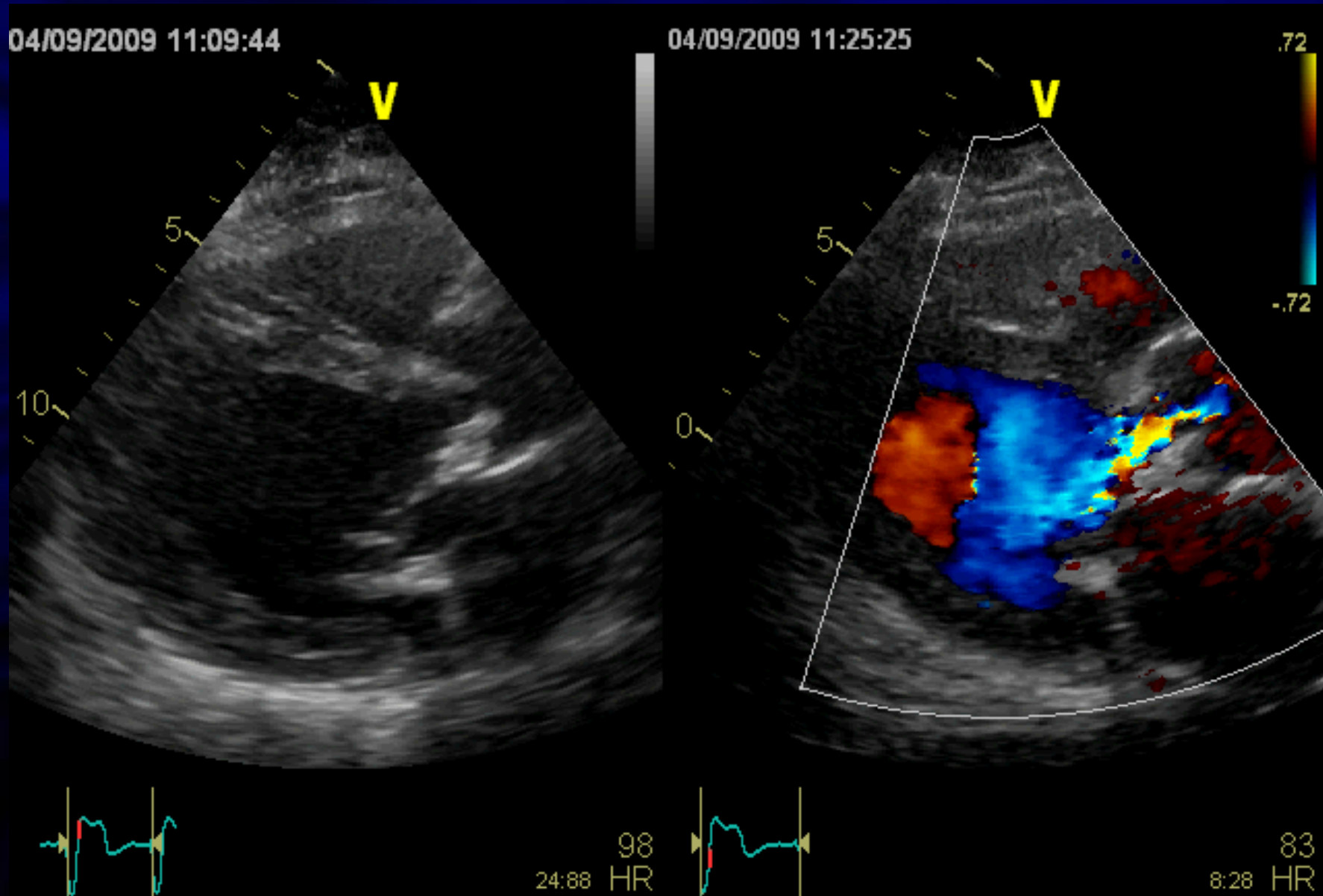


**Type IIa:
Endocarditis**

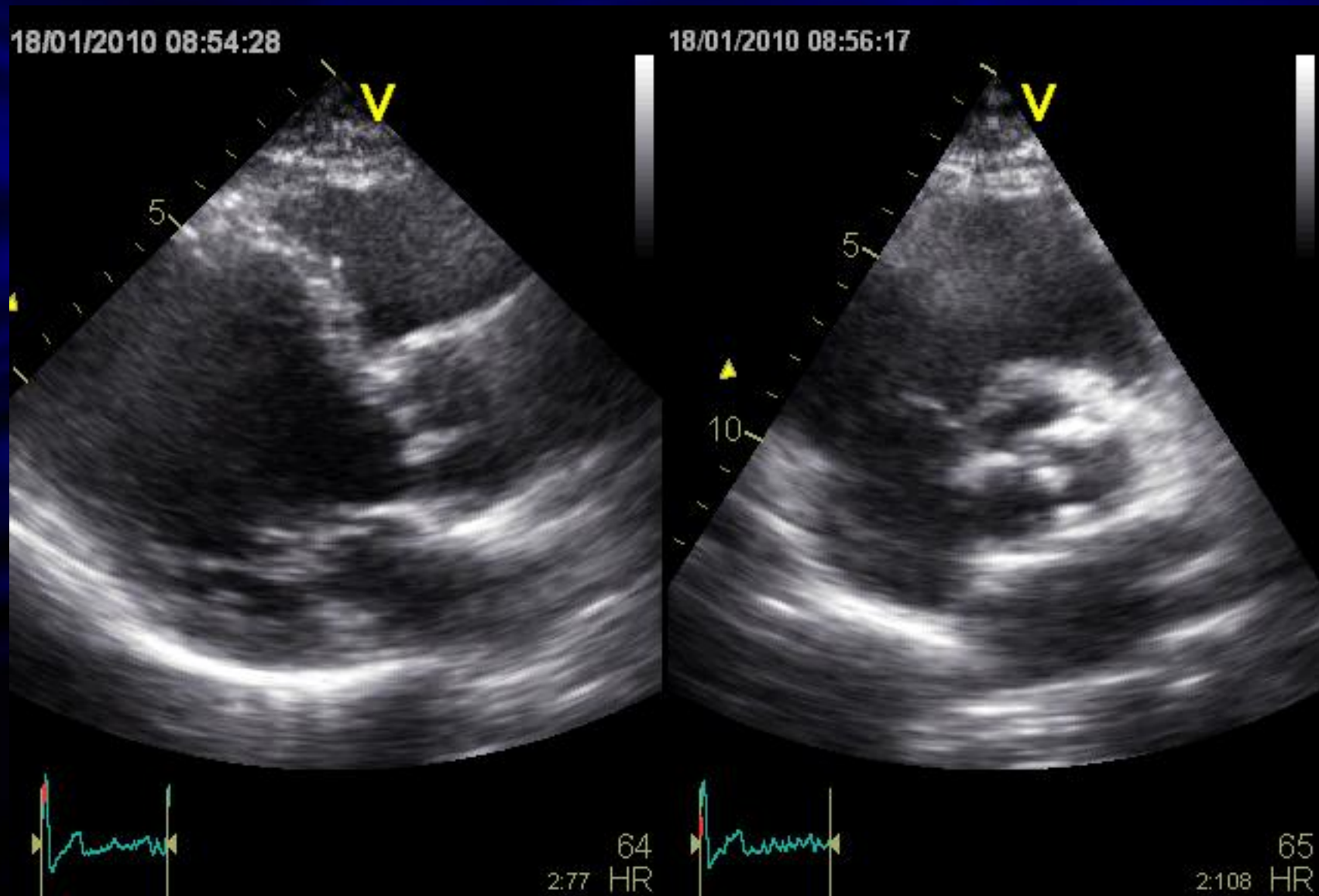
Type IIb: Fenestration



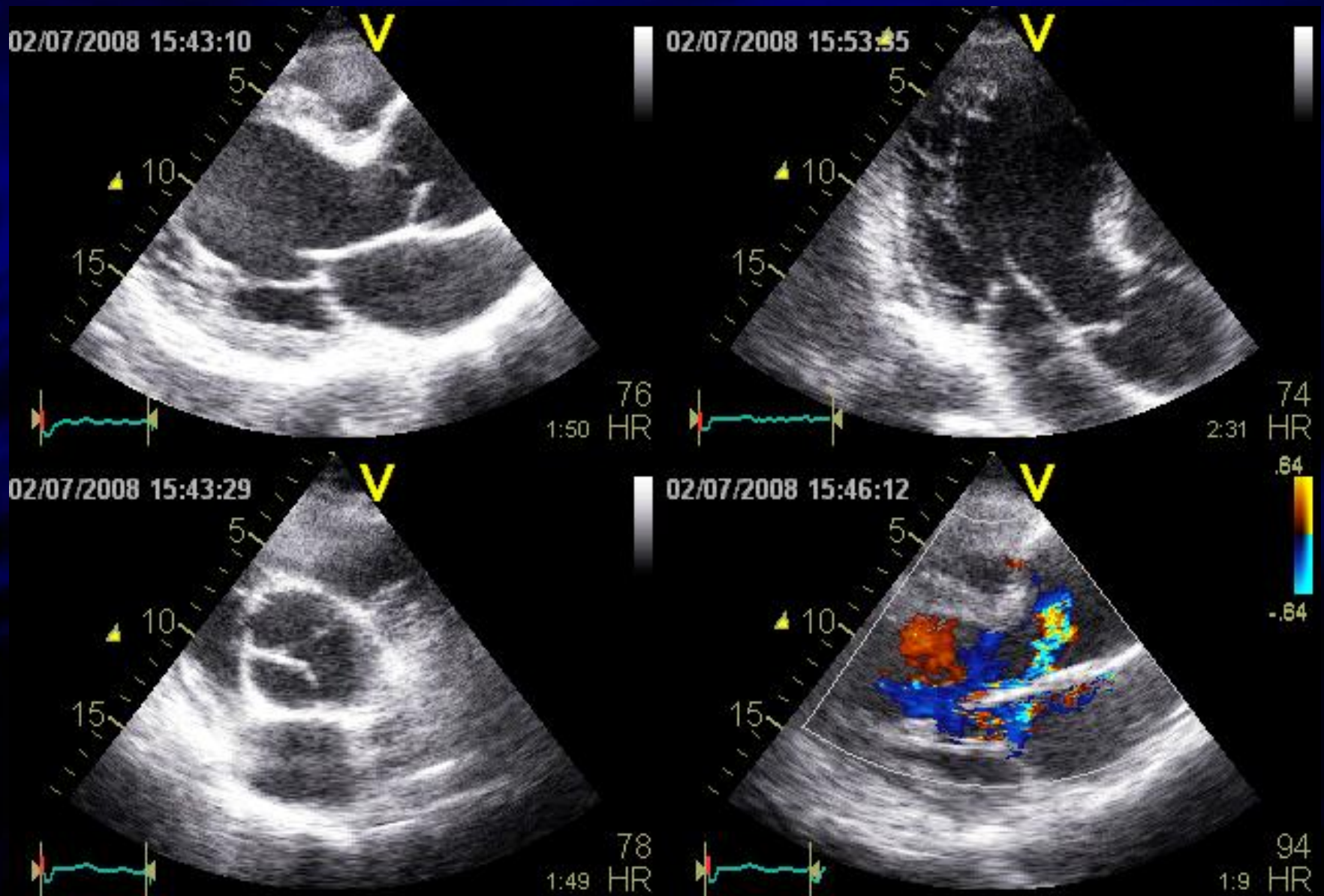
Type III: Leaflet retraction: Tricuspid valve



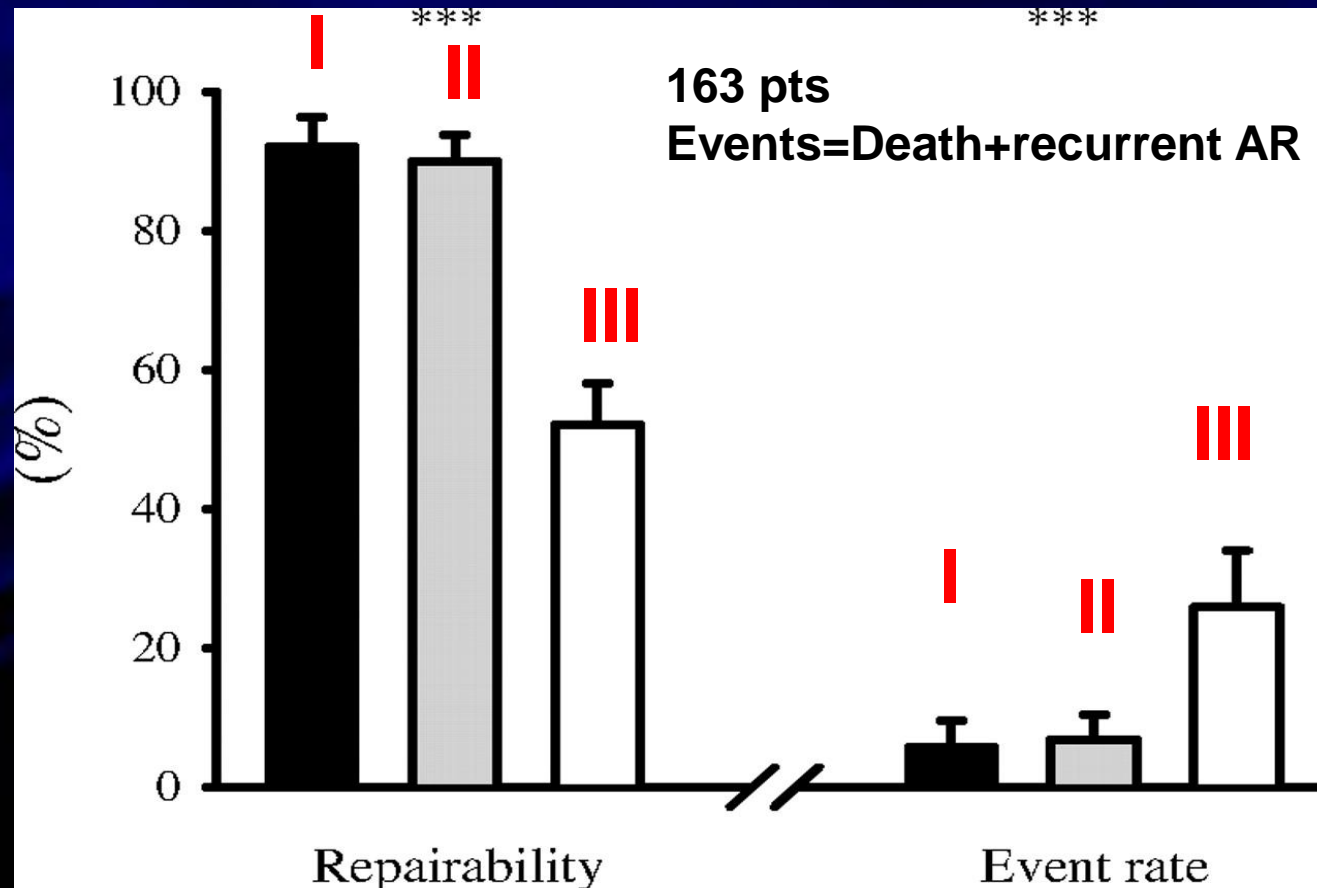
Type III: Leaflet retraction: Bicuspid valve

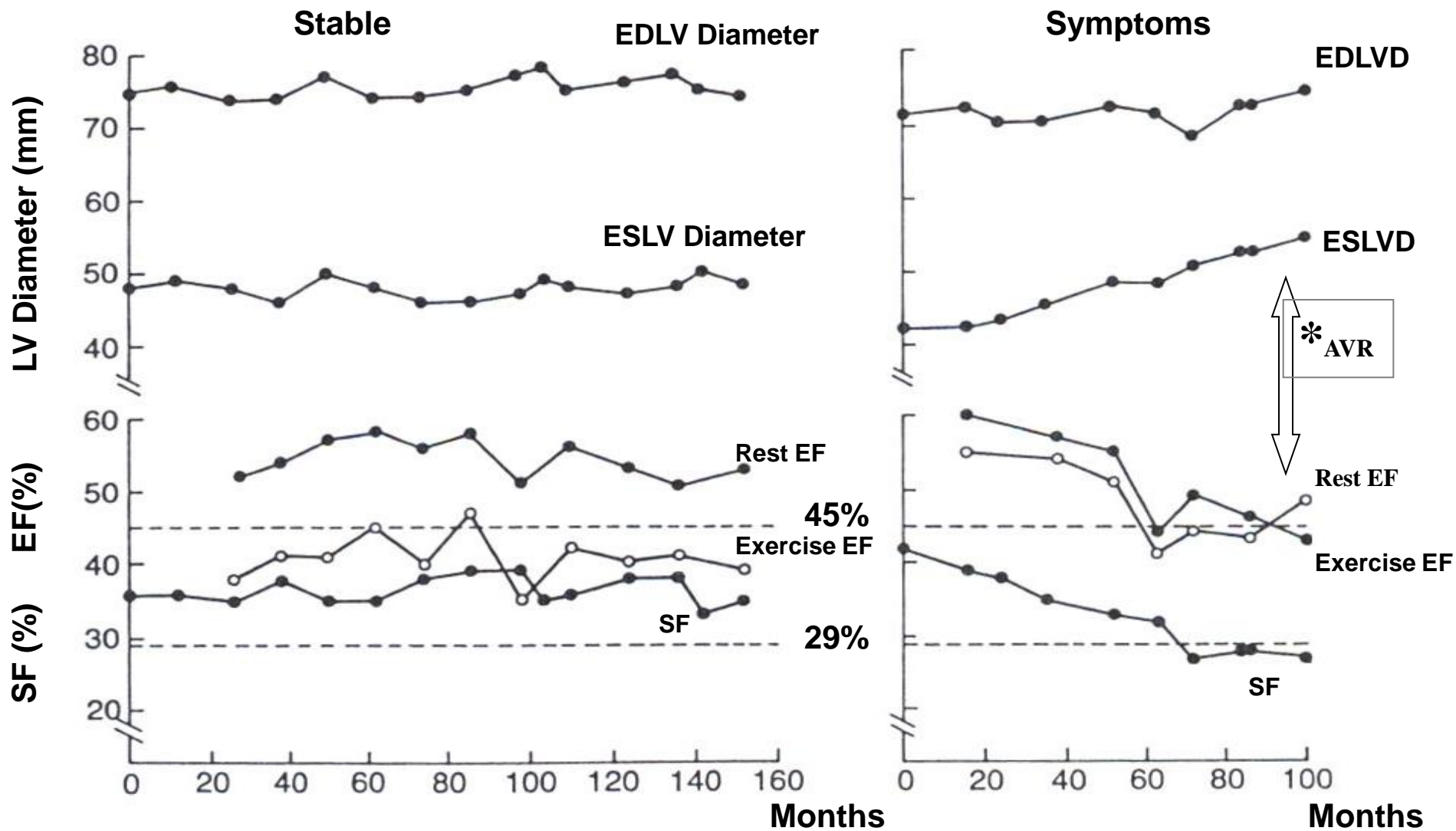


Type ???



Incidence of valve sparing or repair surgery according to anatomic classification by TEE





Natural History of AR

☺ Asymptomatic patients with normal left ventricular (LV) systolic function

Progression to symptoms and/or LV dysfunction < 6%/year

Progression to asymptomatic LV dysfunction <3.5%/year

Sudden death <0.2%/year

☺ Asymptomatic patients with LV systolic dysfunction

Progression to cardiac symptoms >25%/year

☺ Symptomatic patients

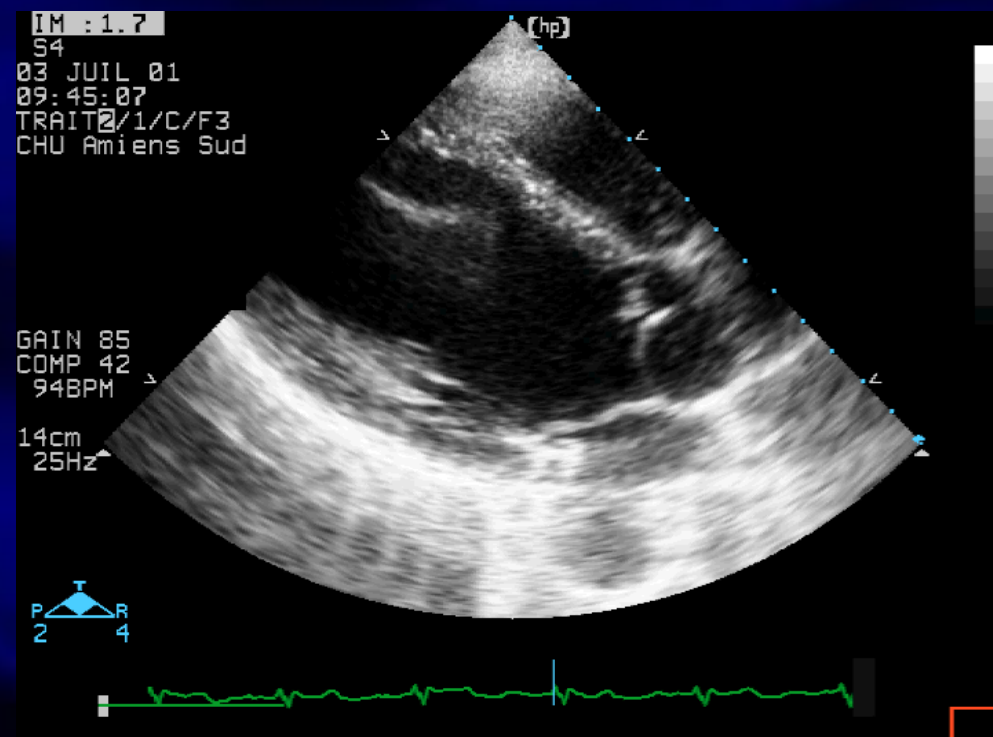
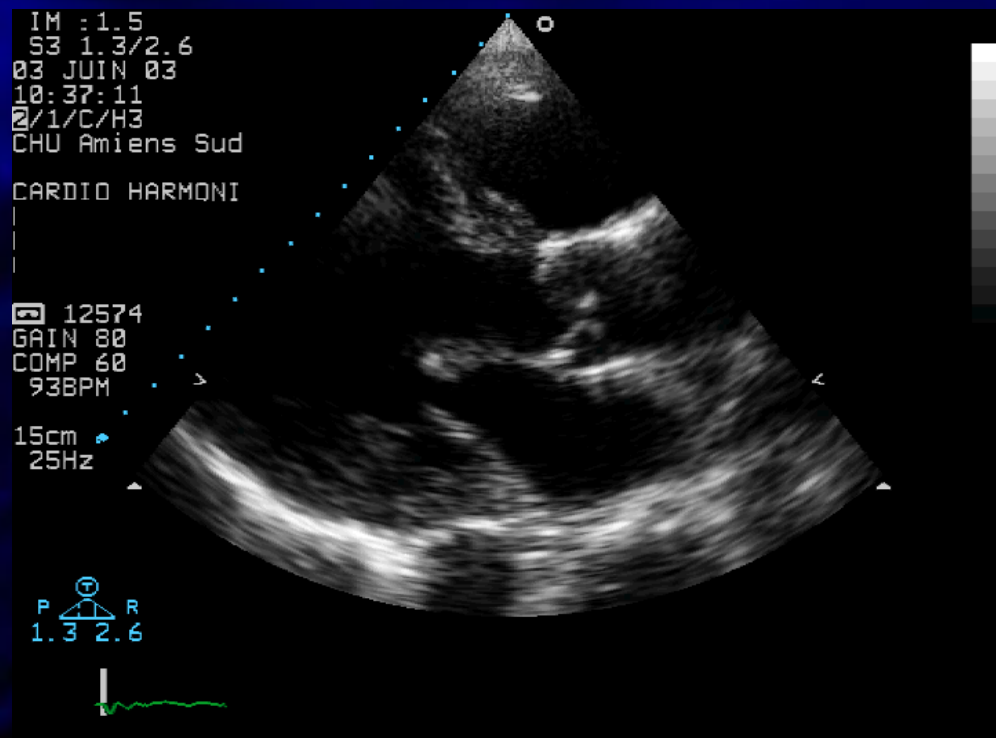
Mortality rate >10%/year

AORTIC REGURGITATION

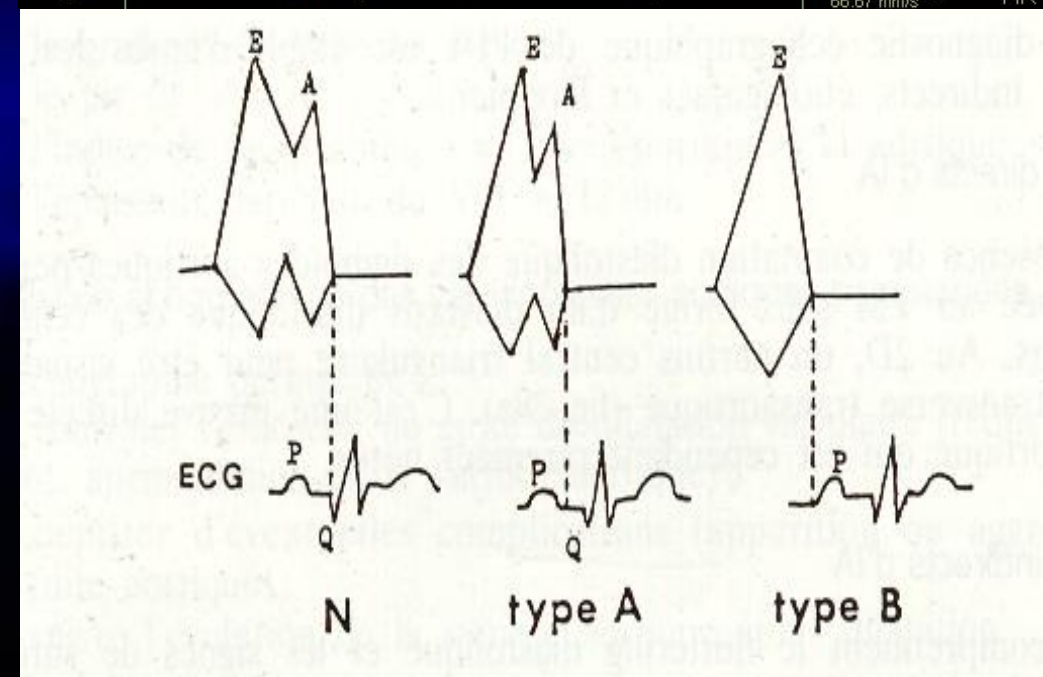
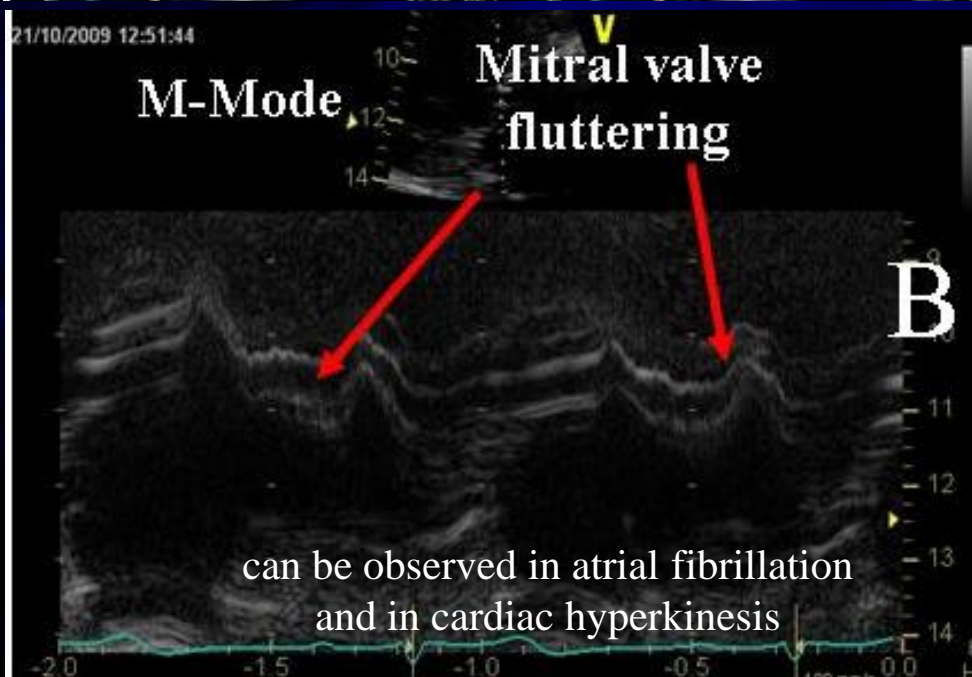
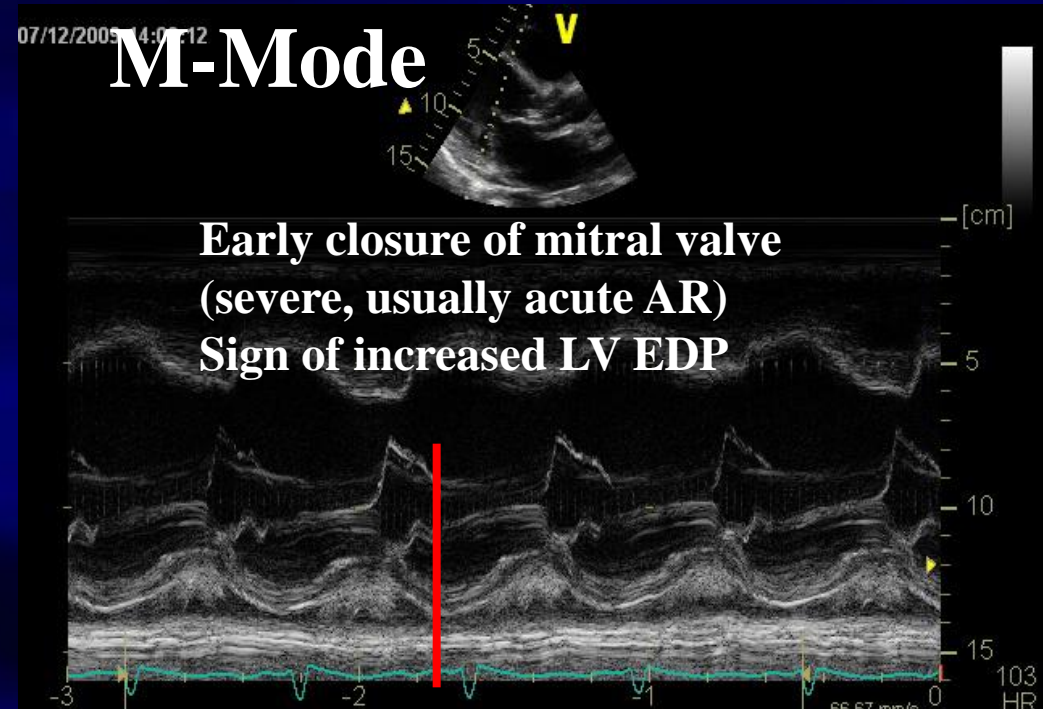
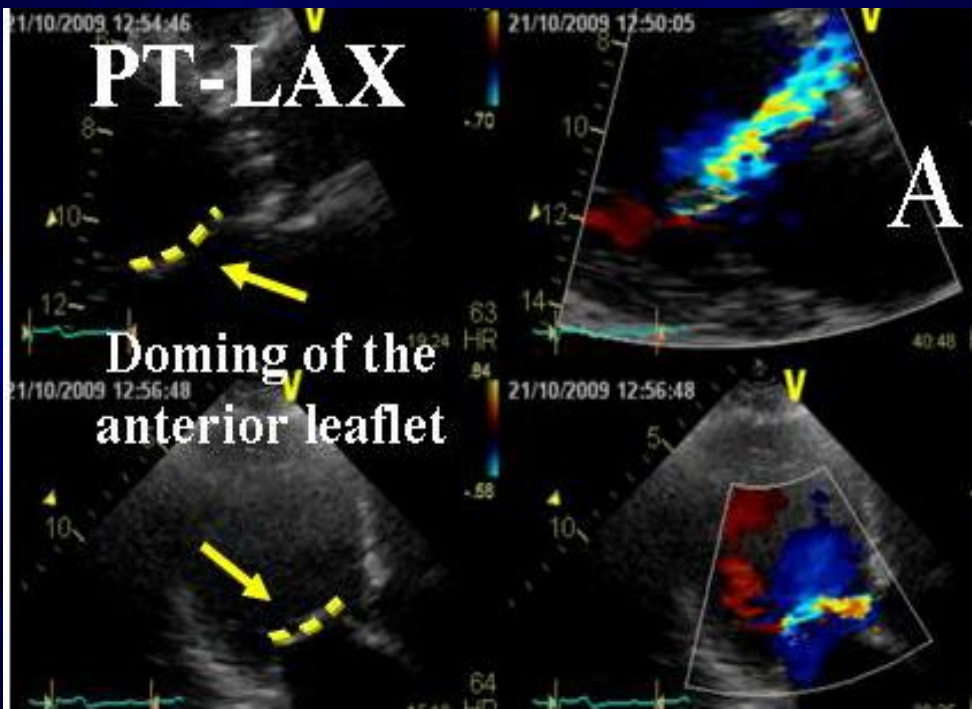
6 Key Questions

1. Is the patient asymptomatic?
2. Is the AR severe?
3. What is the impact on the LV?
4. What is the degree of the dilatation of ascending aorta?
5. What is the mechanism and etiology of AR?
6. What is the operative risk?

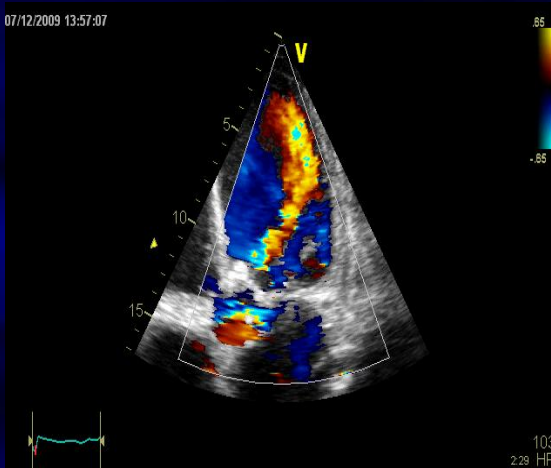
Is the AR severe?



Yes, if there is a defect of coaptation on 2D echo



Color Flow Imaging

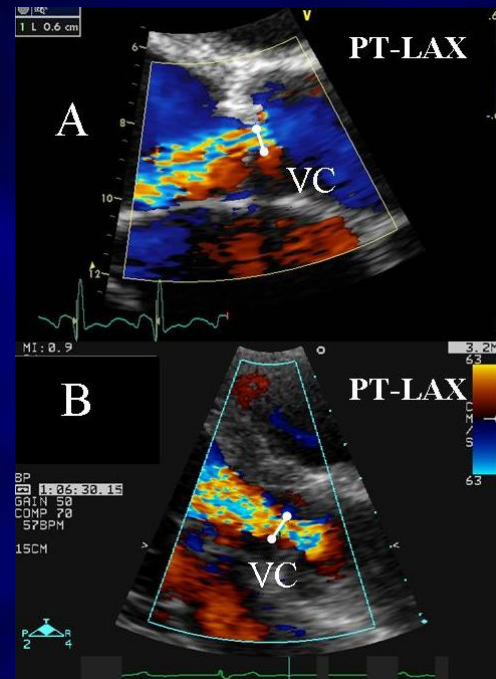
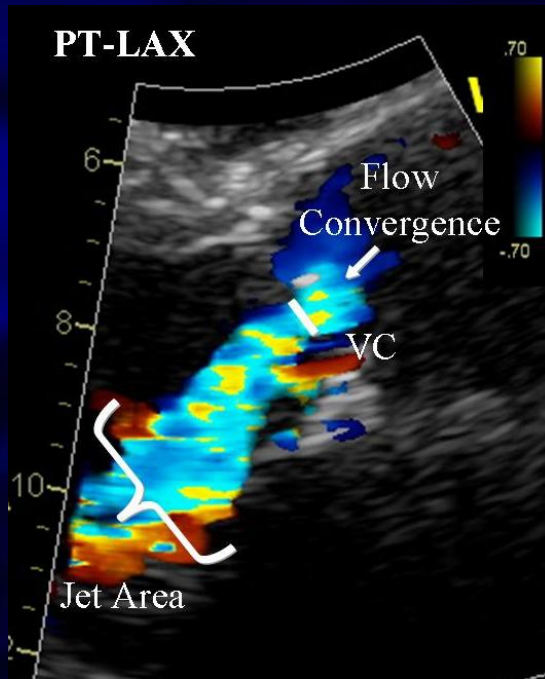


- Ease of use
- Evaluates the speed of the AR jet
- Quick set up

NOT VALID FOR AR QUANTIFICATION

- Technical factors (Gain settings, PRF)
- Inaccurate for eccentric jet
- Loading conditions
- Expands unpredictably below the orifice

Vena Contracta Width



Limitations

- Not valid for multiple jets
- Small values (large % error)
- Intermediate values need confirmation
- Affected by changes in regurgitant flow
- Orifice often non-circular

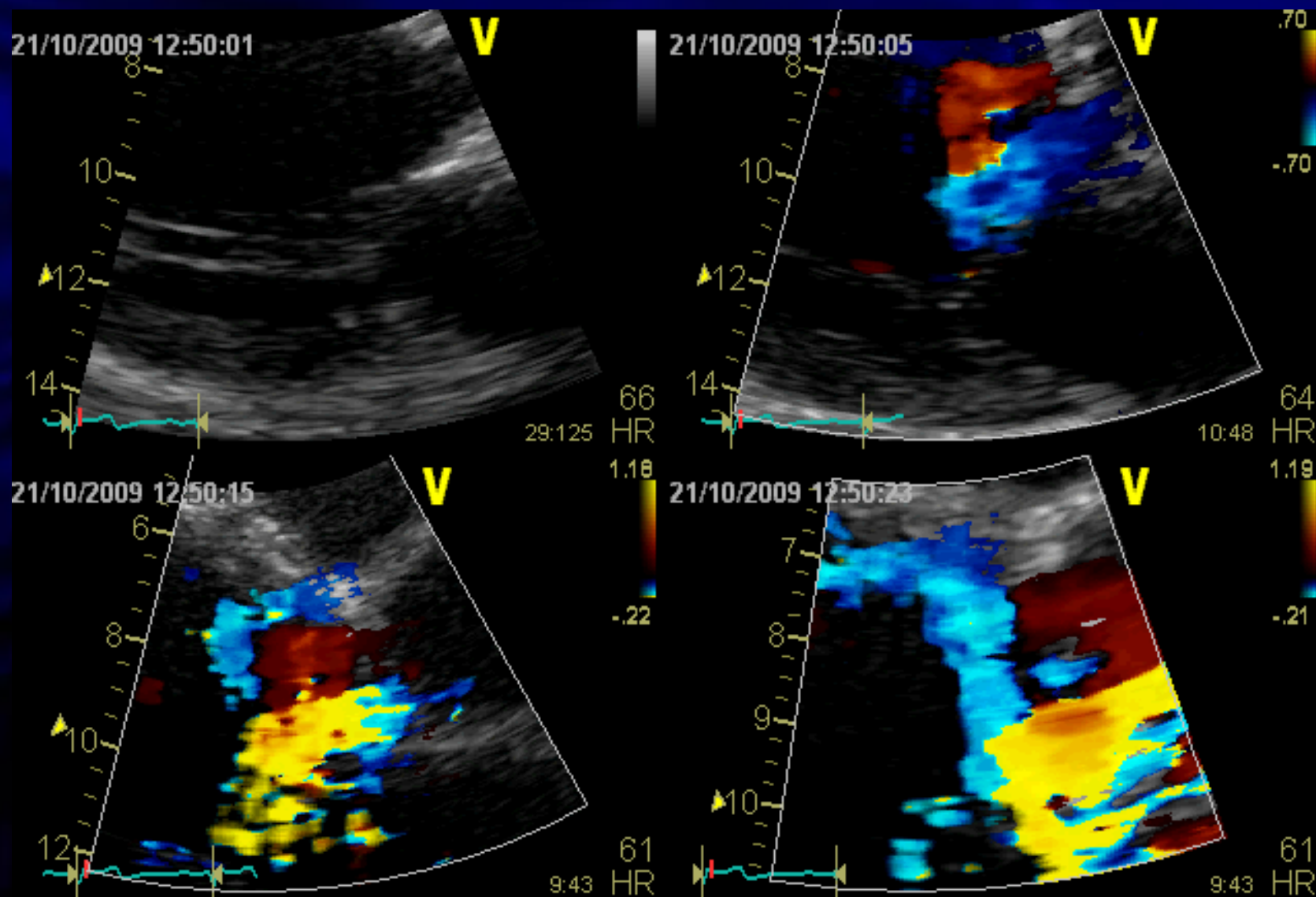
- PT-LAX
- Zoom to optimize visualization
- Color sector as narrow as possible
- Small influence of haemodynamics
- Not affected by other valve leak

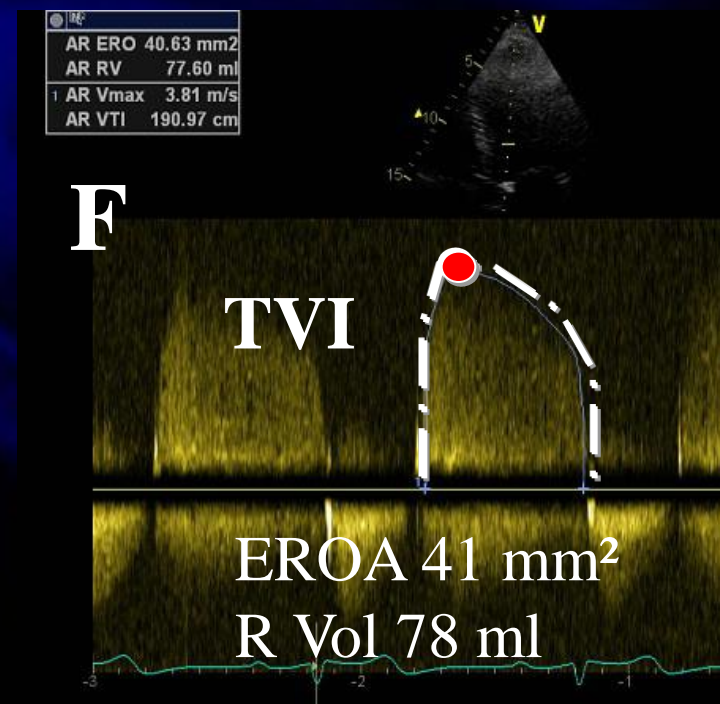
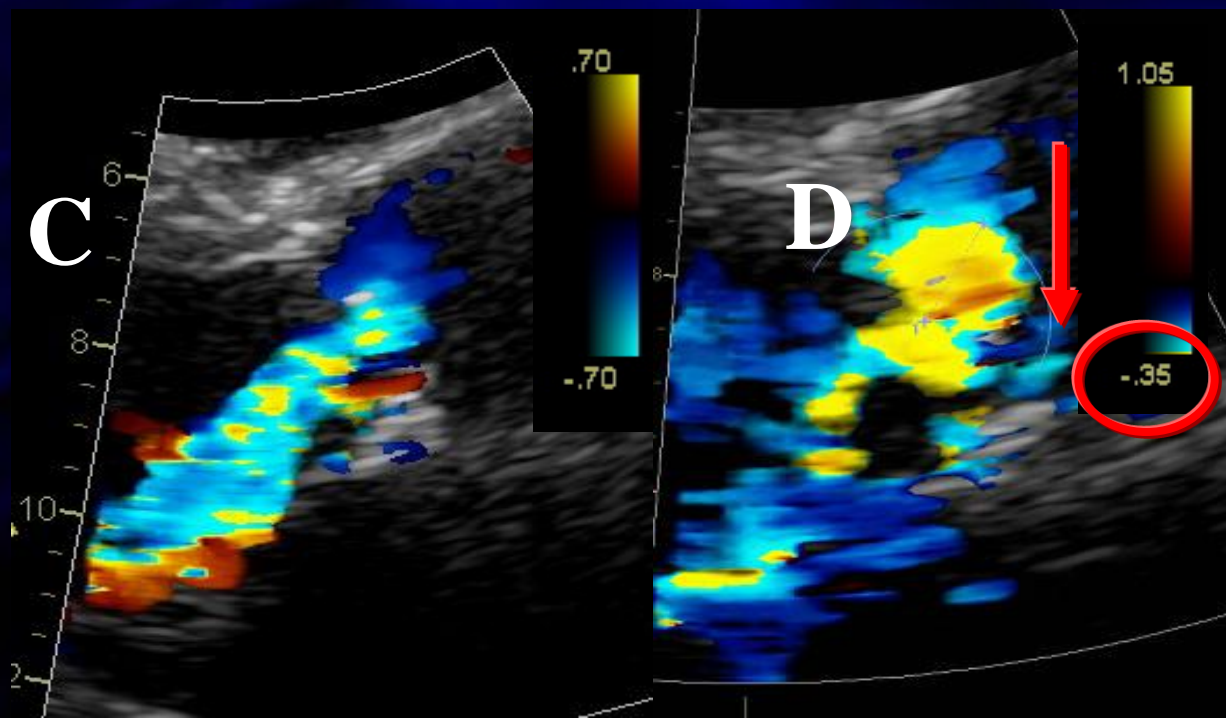
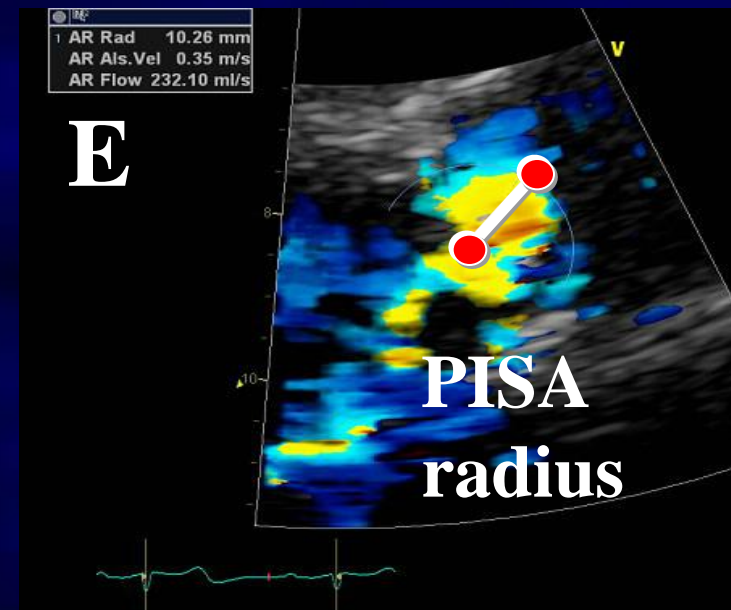
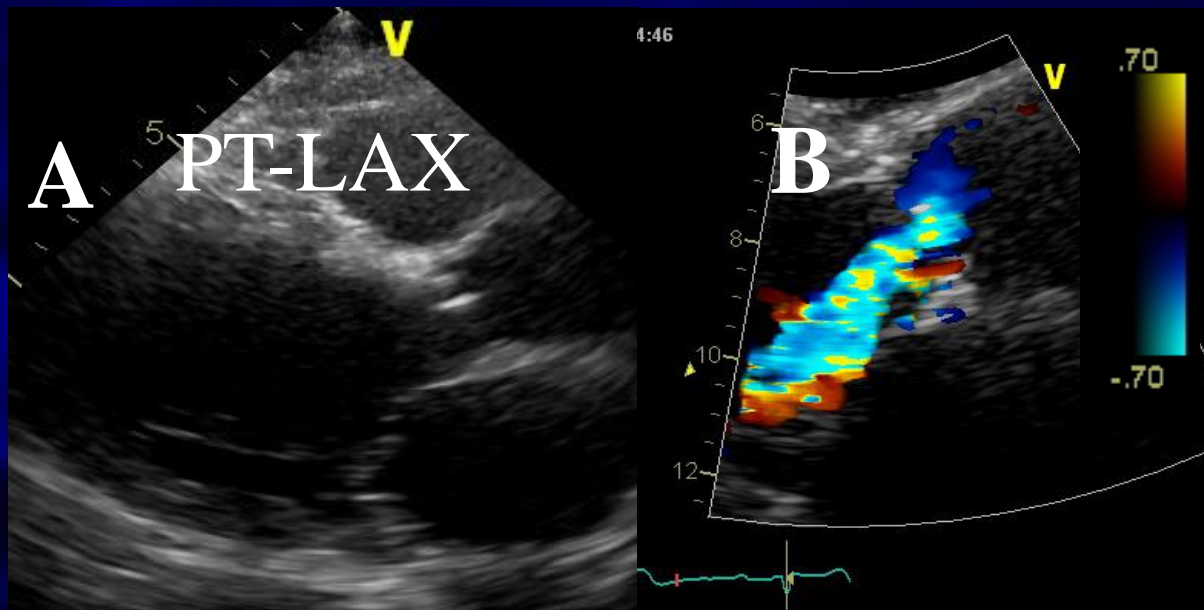
Mild < 3 mm

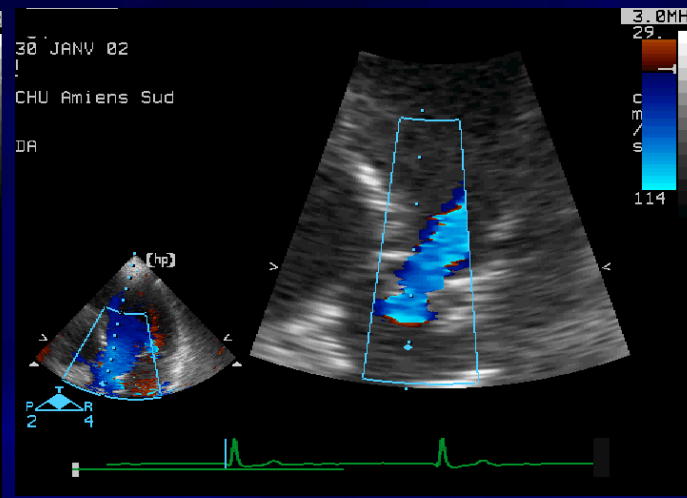
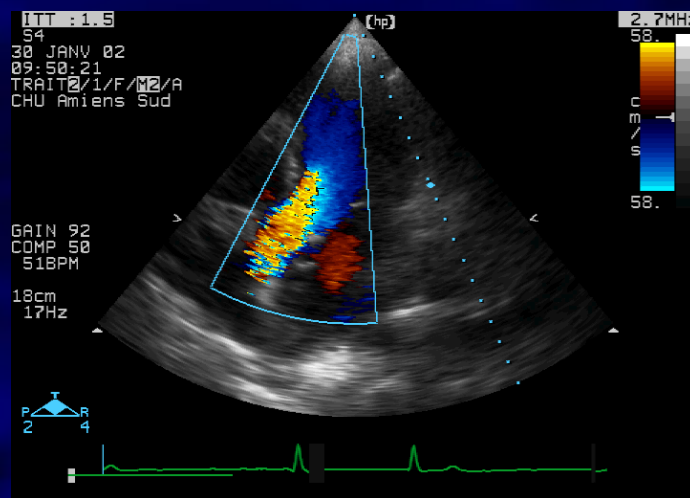
Moderate 3-6 mm

Severe > 6 mm

PISA METHOD







Mild AR ERO $< 10 \text{ mm}^2$; Severe AR ERO $> 30 \text{ mm}^2$

Advantages (ERO)

Independent of HR

Small influence of haemodynamics

Can be used in eccentric jet

Pitfalls (ERO)

Underestimation of ERO

if obtuse angle (Ao aneurysm)

PISA shape affected

- by the aliasing velocity
- in case of non-circular orifice
- by systolic changes in regurgitant flow
- by adjacent structures (flow constraintment)

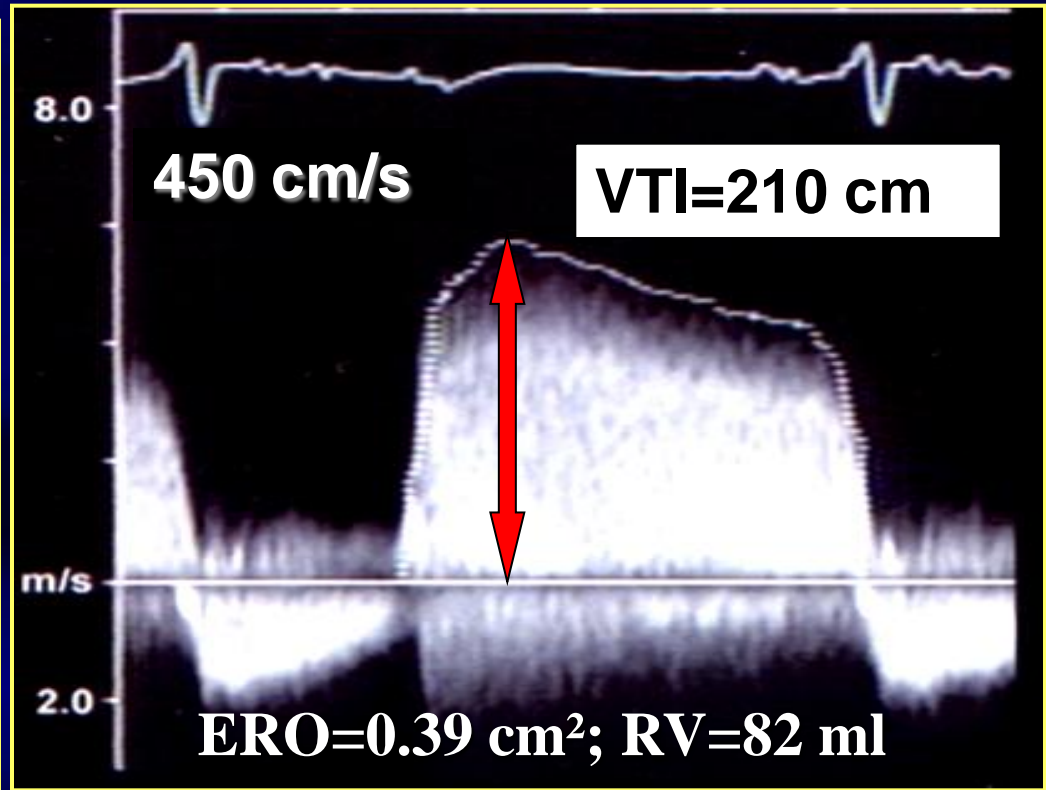
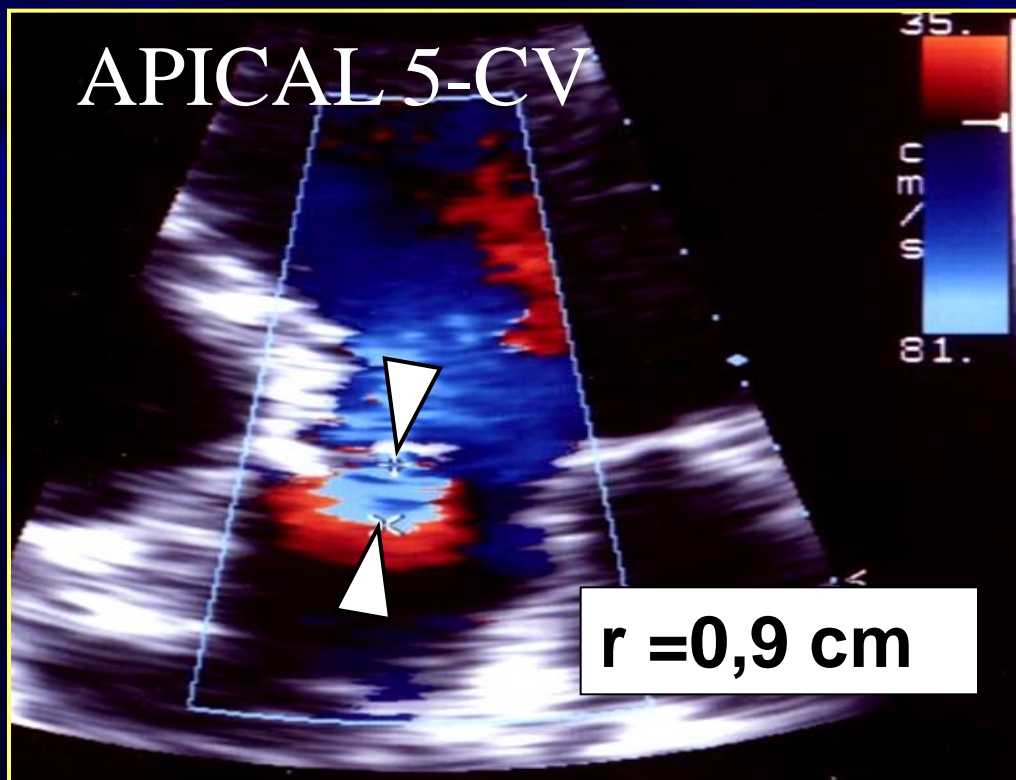
PISA radius is more a hemi-ellipse

Errors in PISA measurement are squared

Inter-observer variability

Not valid for multiple jets

Feasibility limited by aortic valve calcifications

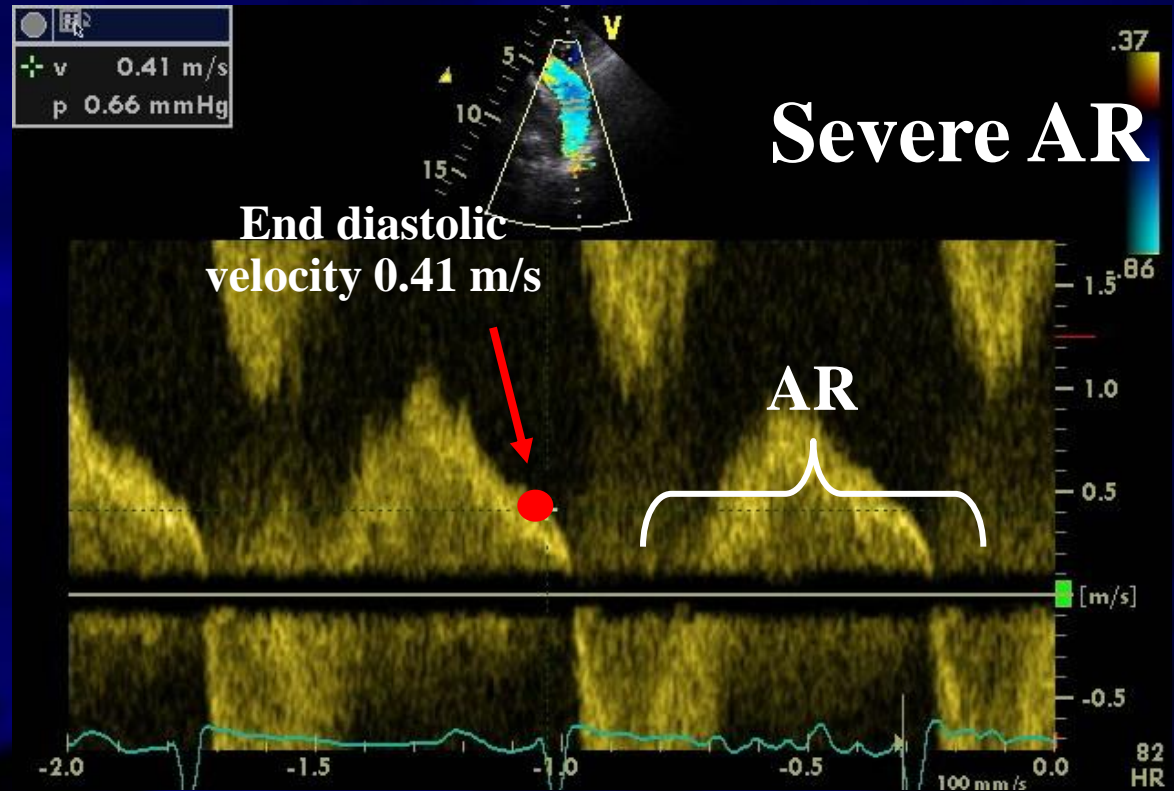
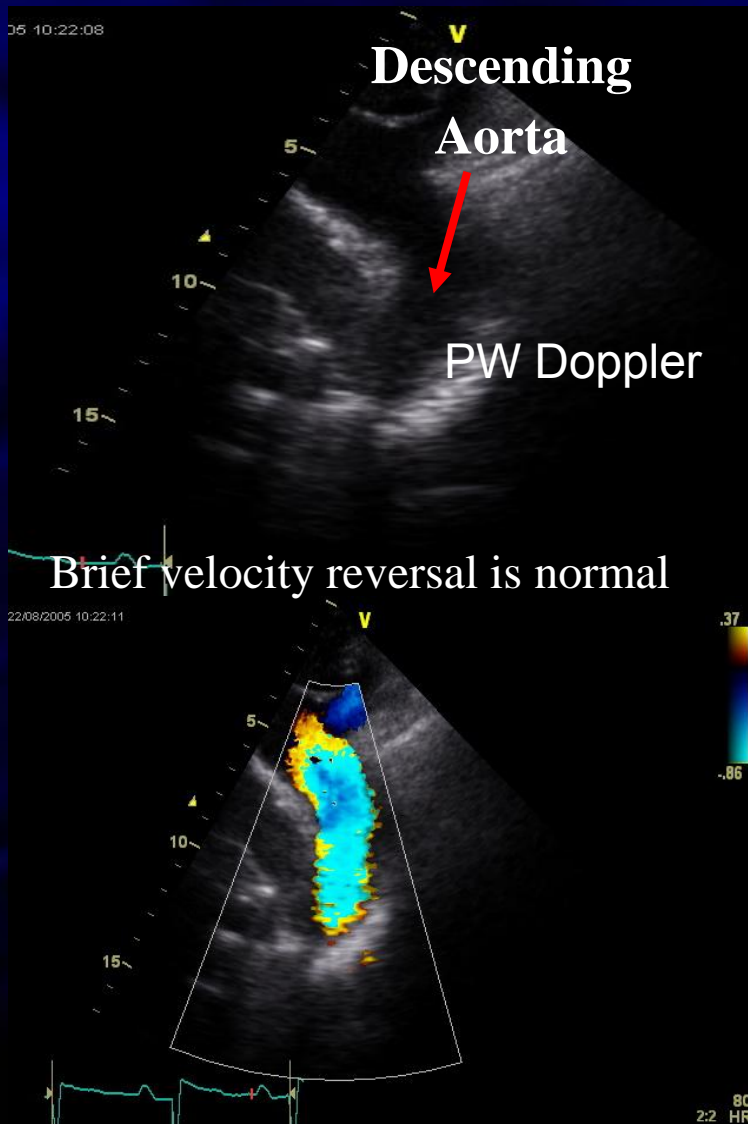


A

$$\text{ERO} = \text{Flow} / \text{Peak velocity} = 178/450 = 0.39 \text{ cm}^2$$

$$\text{R Vol.} = \text{EROA} \times \text{TVI} = 0.39 \text{ cm}^2 \times 210 \text{ cm} = 82 \text{ ml}$$

Diastolic flow reversal in the descending aorta



Cut-off validated for distal aortic arch

In severe AR: holodiastolic flow reversal/EDV > 20 cm/s

Affected by sample volume location and acuity of AR

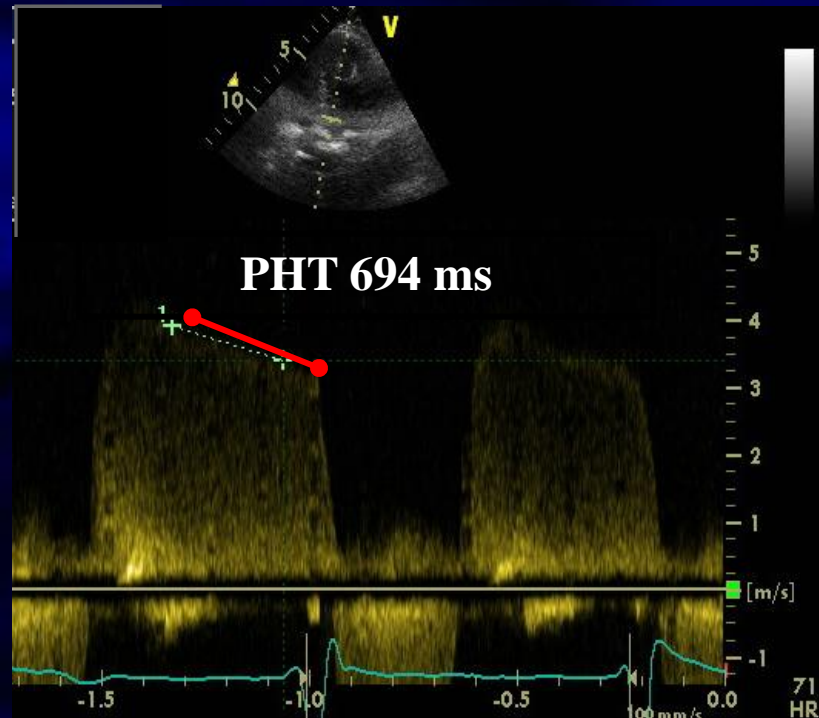
Affected by aortic compliance

Pressure half-time

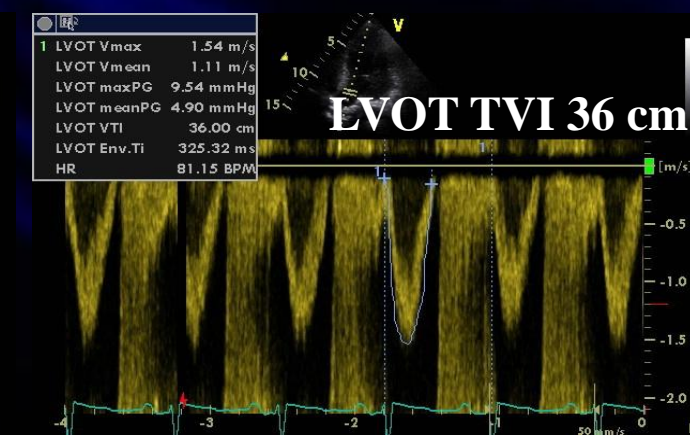
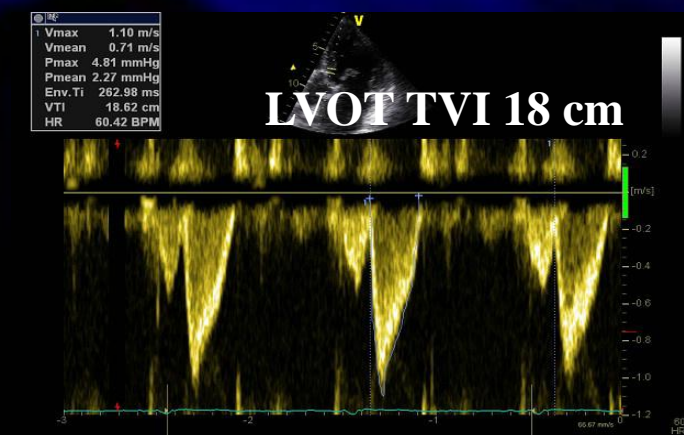
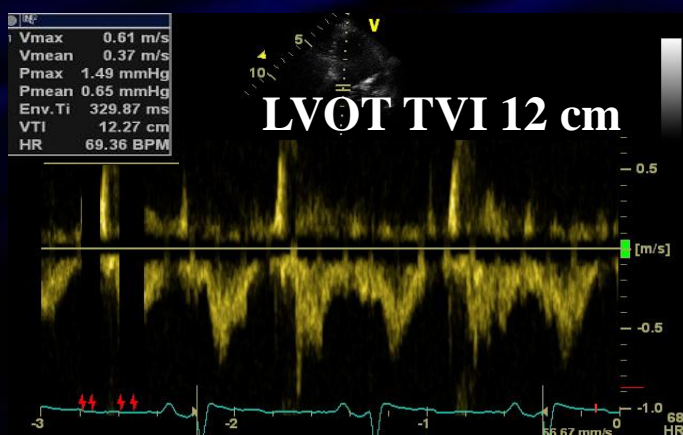
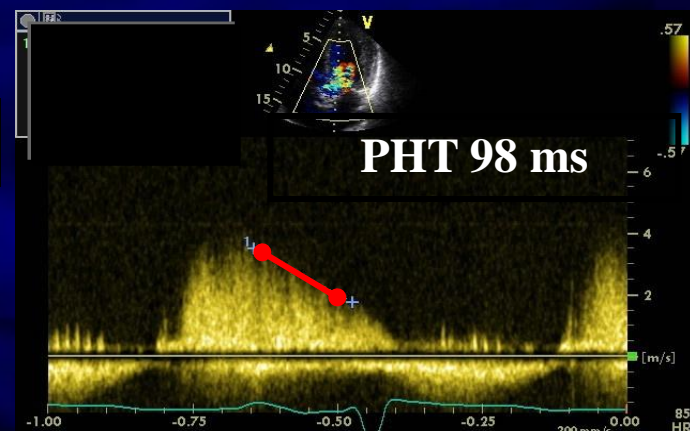
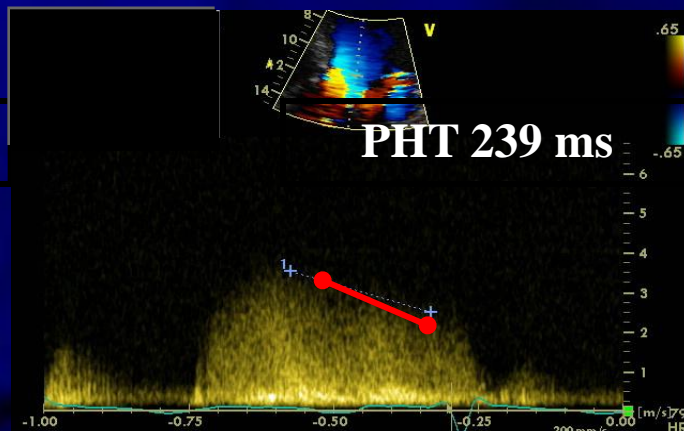
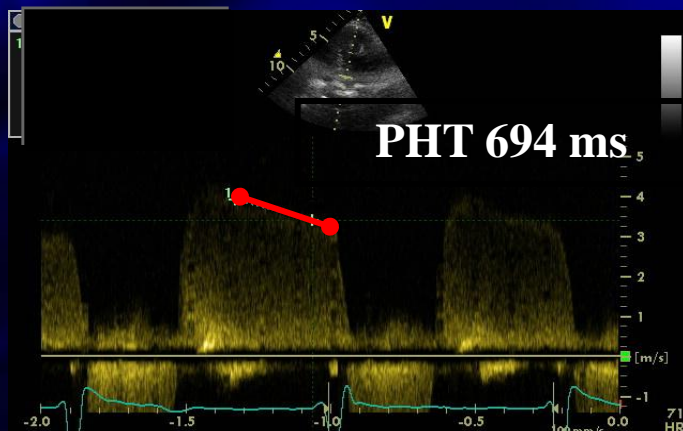
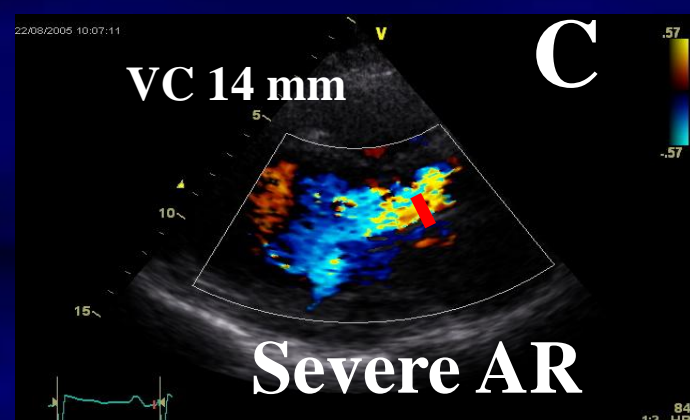
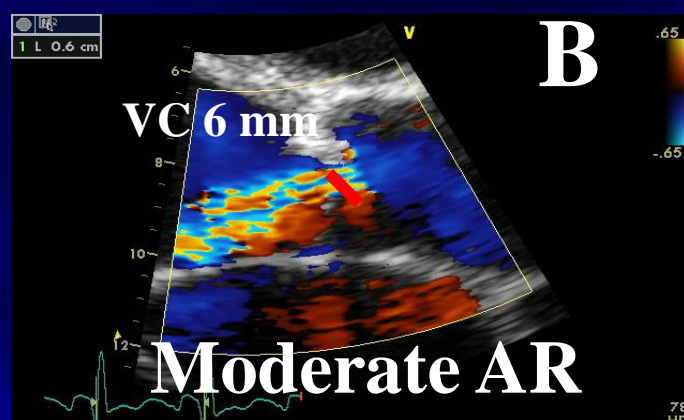
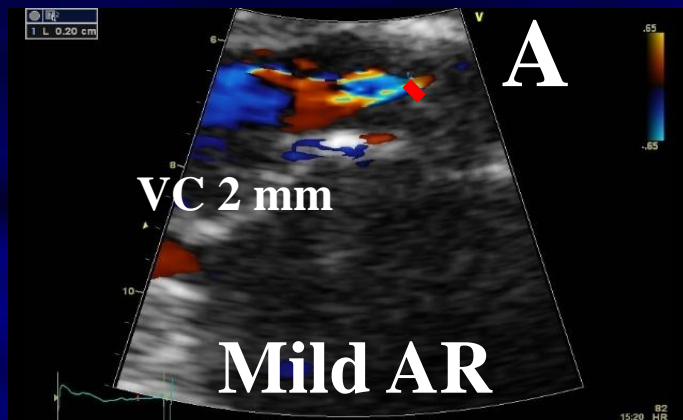
Mild AR > 500 ms, Severe AR < 200 ms

Pitfalls

- \downarrow PHT when
 - \uparrow SVR
 - \uparrow LVED
 - \uparrow Aortic compliance
 - \downarrow LV relaxation
- Adequate spectrum definition



- Apical 5-chamber
- Simple, easily available
- Qualitative, Complementary finding
- Complete signal difficult to obtain in eccentric jet



Integrating indices of AR severity

Parameters	Mild	Moderate	Severe
Qualitative Aortic valve morphology Colour flow AR jet width \$ CW signal of AR jet Diastolic flow reversal in descending aorta	Normal/Abnormal Small in central jets Incomplete/Faint Brief, protodiastolic flow reversal	Normal/Abnormal Intermediate Dense Intermediate	Abnormal/ Flail/ Large coaptation defect Large in central jet, variable in eccentric jets Dense Holodiastolic flow reversal (end-diastolic velocity > 20 cm/s)
Semi-quantitative VC width (mm) Pressure half-time (ms) £	< 3 > 500	Intermediate Intermediate	≥ 6 < 200
Quantitative EROA (mm ²) R Vol (ml)	< 10 < 30	10-19 ; 20-29! 30-44 ; 45-59!	≥ 30 ≥ 60
+ LV size §			

European Association of Echocardiography recommendations for the assessment of valvular regurgitation. Part 1: aortic and pulmonary regurgitation (native valve disease)

Patrizio Lancellotti(Chair)^{1*}, Christophe Tribouilloy², Andreas Hagendorff³,
Luis Moura⁴, Bogdan A. Popescu⁵, Eustachio Agricola⁶, Jean-Luc Monin⁷,
Luc A. Pierard¹, Luigi Badano⁸, and Jose L. Zamorano⁹ on behalf of the European
Association of Echocardiography