Ανεπάρκεια αορτικής βαλβίδας: εκτίμηση της βαρύτητας και ενδείξεις χειρουργικής αντιμετώπισης

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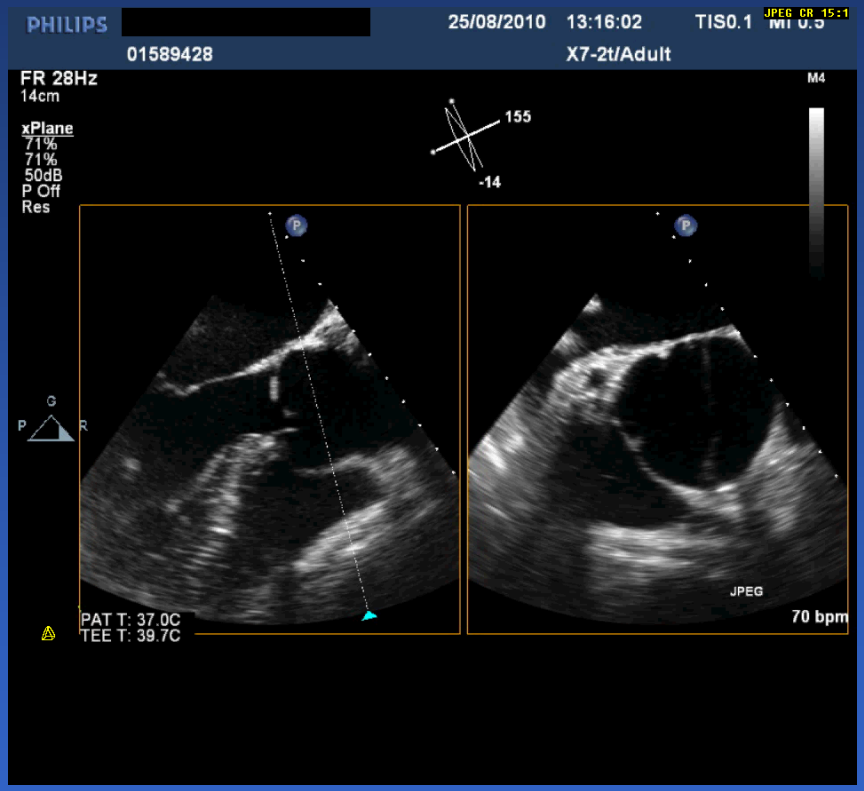
Prevalence of AR

	Age, y				
	26–39	40–49	50–59	60–69	70–83
Men	(n=91)	(n-352)	(n=433)	(n=359)	(n=91)
None	96.7%	95.4%	91.1%	74.3%	75.6%
Trace	3.3%	2.9%	4.7%	13.0%	10.0%
Mild	0%	1.4%	3.7%	12.1%	12.2%
≥Moderate	0%	0.3%	0.5%	0.6%	2.2%
Women	(n=93)	(n=451)	(n=515)	(n=390)	(n=90)
None	98.9%	96.6%	92.4%	86.9%	73.0%
Trace	1.1%	2.7%	5.5%	6.3%	10.1%
Mild	0%	0.7%	1.9%	6.0%	14.6%
≥Moderate	0%	0%	0.2%	0.8%	2.3%

Singh et al. Am J Cardiol 1999

Aetiology of AR



Major causes of aortic regurgitation

Leaflet abnormalities	Aortic root or ascending aorta
Rheumatic fever	Systemic hypertension
Endocarditis (bacterial or marantic)	Aortitis (eg, syphilis)
Trauma	Reactive arthritis
Bicuspid aortic valve	Ankylosing spondylitis
Rheumatoid arthritis	Trauma
Myxomatous degeneration	Dissecting aneurysm
Ankylosing spondylitis	Marfan syndrome
Acromegaly	Ehlers-Danlos syndrome
Fenfluramine-phentermine	Pseudoxanthoma elasticum
	Inflammatory bowel disease
	Osteogenesis imperfecta
	Annuloaortic ectasia

Aetiology of AR

Cause of AR	Total	Ages at Operation, Range (Mean), y	M	F	Acute	Chronic	SH	Coronary Artery Bypass Grafting	Portions of Ascending Aorta		
									Excised	Examined Histologically	CMN (3+, 4+)
Valve (122 [46%])											
Congenital malformation without Infective endocarditis											
Bicuspid	59 (22%)	22–77 (55)	49	10	0	59	39 (66%)	18 (31%)	22	22	11
Quadricuspid	2 (1%)	53–79 (66)	0	2	0	2	0	1 (50%)	0	0	0
Tricuspid	5 (2%)	33–48 (40)	3	2	0	5	2 (40%)	0	1	1	0
Infective endocarditis	46 (17%)	21–82 (45)	31	15	27	19	29 (63%)	7 (15%)	6	4	0
Rheumatic?	8 (3%)	25–63 (47)	6	2	0	8	6 (75%)	2 (25%)	0	0	0
Miscellaneous	2 (1%)	24–42 (33)	1	1	0	2	2 (100%)	1 (50%)	0	0	0
Nonvalve (146 [54%])											
Aortic dissection	28 (10%)	25–78 (58)	20	8	21	7	22 (79%)	5* (17%)	28	20	5
Marfan or forme fruste	15 (6%)	21–71 (47)	9	6	0	15	10 (67%)	1† (7%)	15	13	13
Aortitis	12 (4%)	35–82 (66)	5	7	0	12	10 (83%)	5 (42%)	12	12	12
Cause unclear	91 (34%)	50–84 (66)	58	33	0	91	83 (91%)	46 (51%)	7	7	0
Total	268 (100%)	21–84 (57)	182 (68%)	86 (32%)	48 (18%)	220 (82%)	203 (76%)	86 (32%)	91 (34%)	76	41

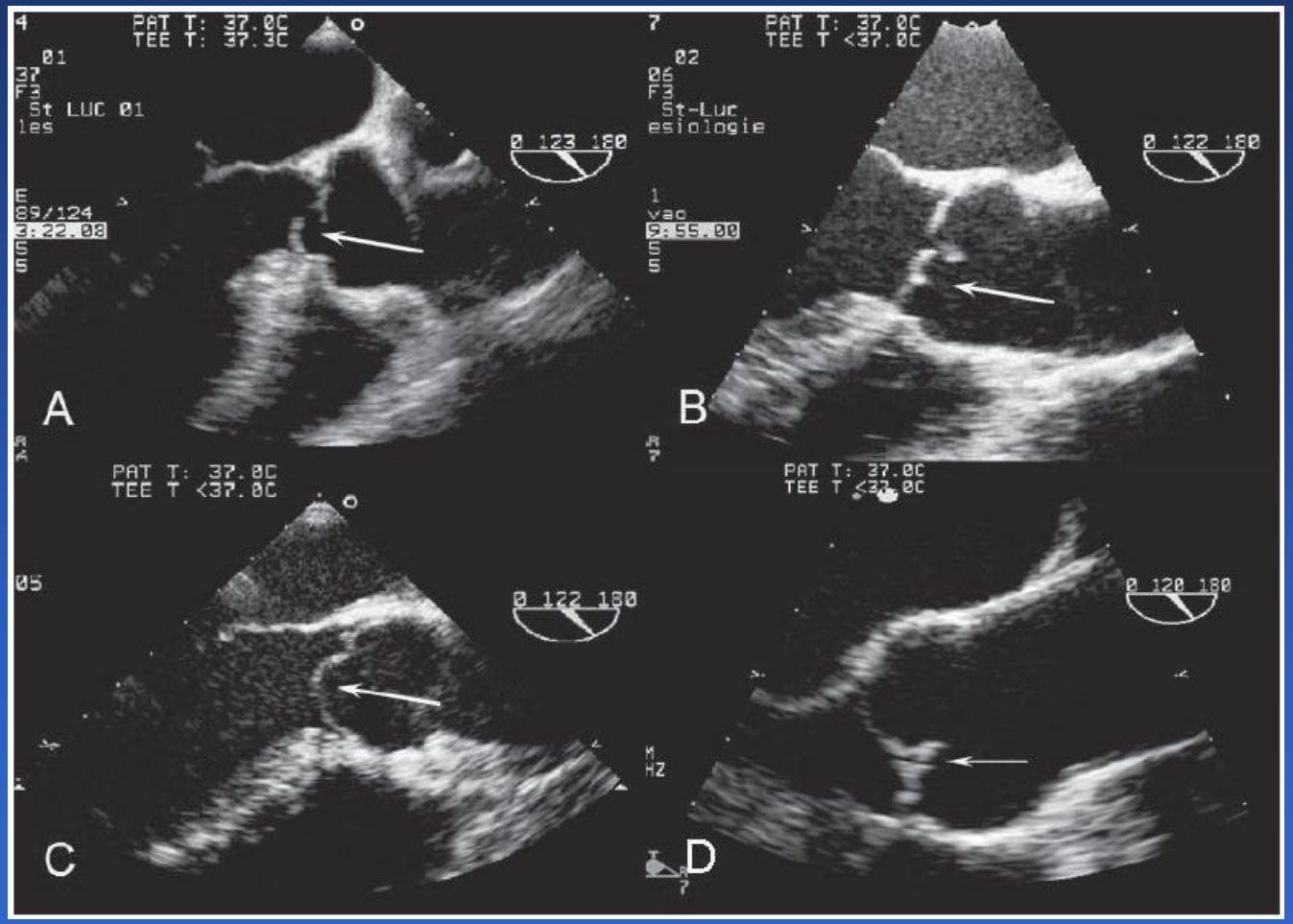
Roberts WC et al. Circulation 2006

Mechanisms of AR

Dysfunction	Echo findings
I: enlargement of the aortic root with normal cusps	Dilatation of any components of the aortic root (aortic annulus, sinuses of Valsalva, sinotubular junction)
IIa: cusp prolapse with eccentric AR jet	
Cusp flail	Complete eversion of a cusp into the LVOT in long-axis views
Partial cusp prolapse	Distal part of a cusp prolapsing into the LVOT (clear bending of the cusp body on long-axis views and presence of a small circular structure near the cusp free edge on short-axis views)
Whole cusp prolapse	Free edge of a cusp overriding the plane of aortic annulus with billowing of the entire cusp body into the LVOT (presence of a large circular or oval structure immediately beneath the valve on short-axis views)
IIb: free edge fenestration with eccentric AR jet	Presence of an eccentric AR jet without definite evidence of cusp prolapse
III: poor cusp quality or quantity	Thickened and rigid valves with reduced motion
	Tissue destruction (endocarditis)
	Large calcification spots/extensive calcifications of all cusps interfering with cusp motion

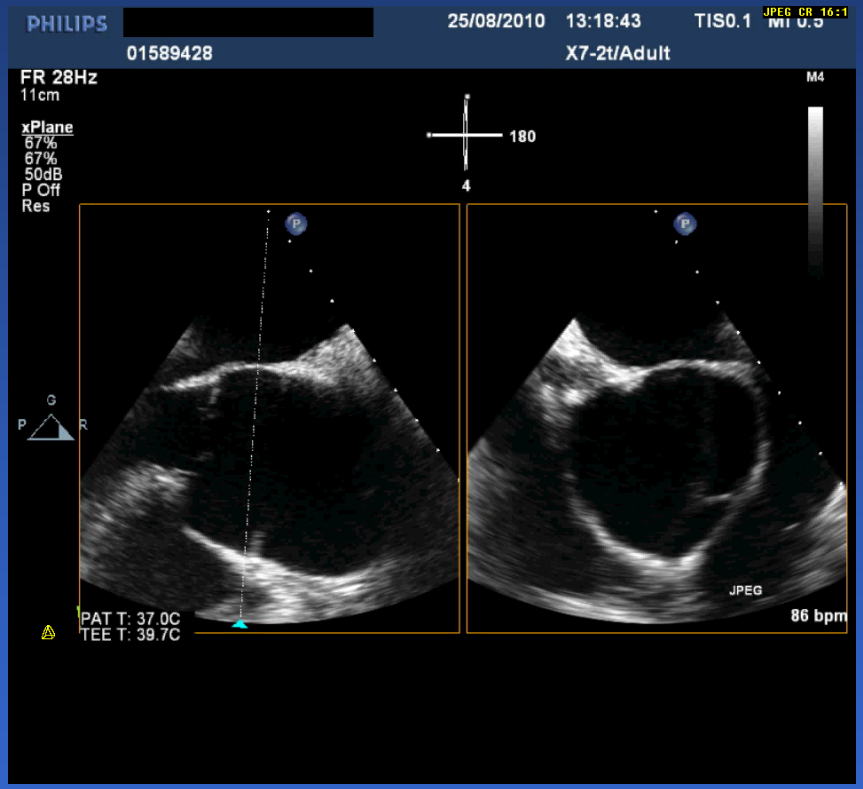
EAE recommendations. Eur J Echocardiogr 2010

Mechanisms of AR



Le Polain de Waroux JB et al. Circulation 2007

Type I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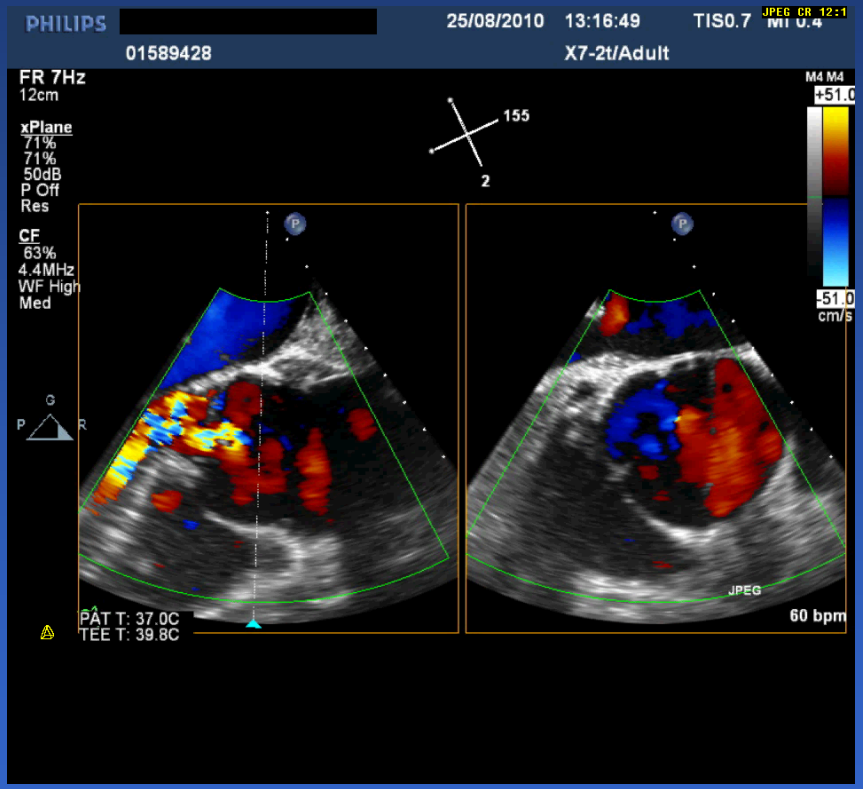
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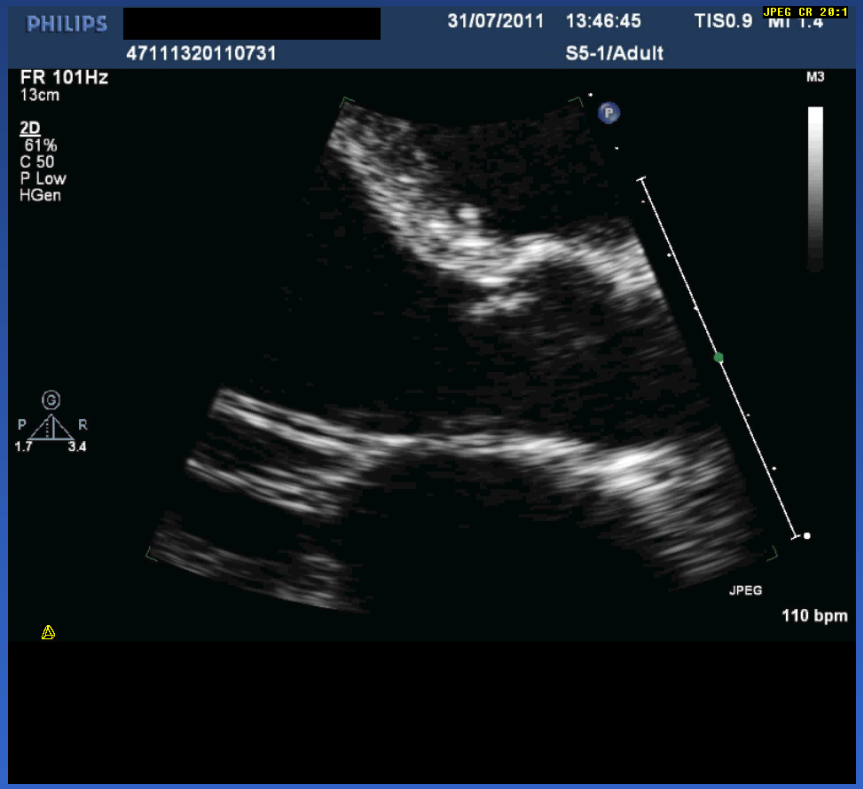
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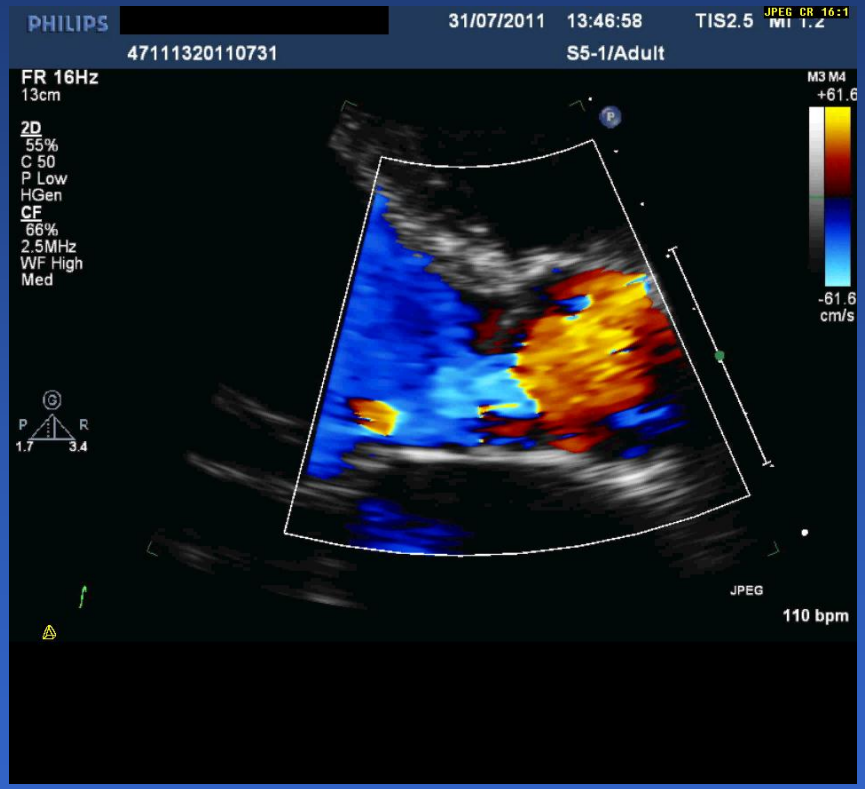
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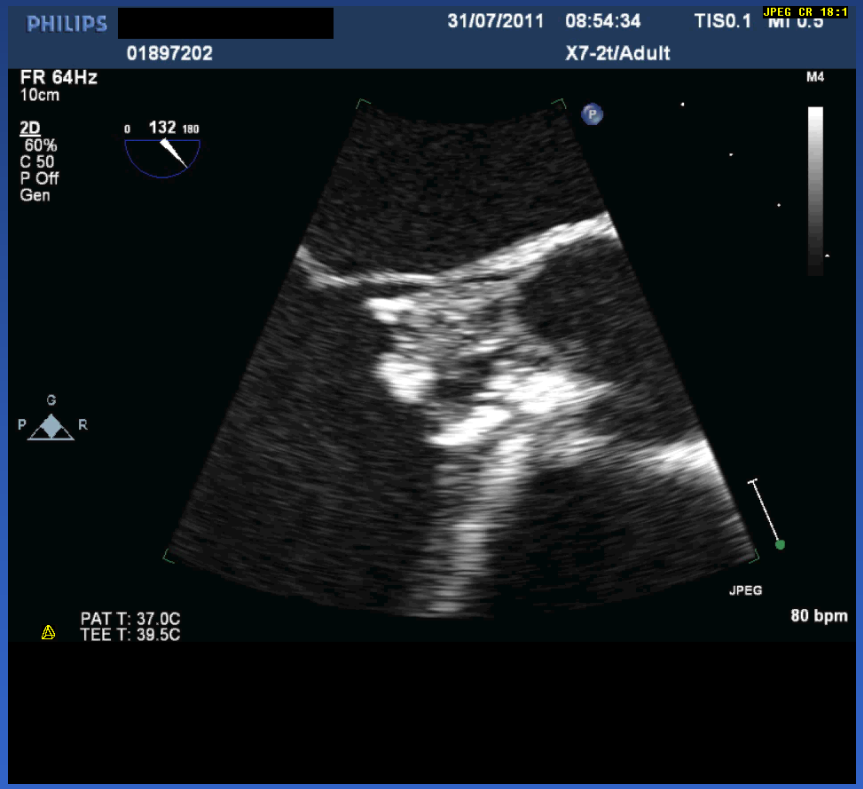
Type III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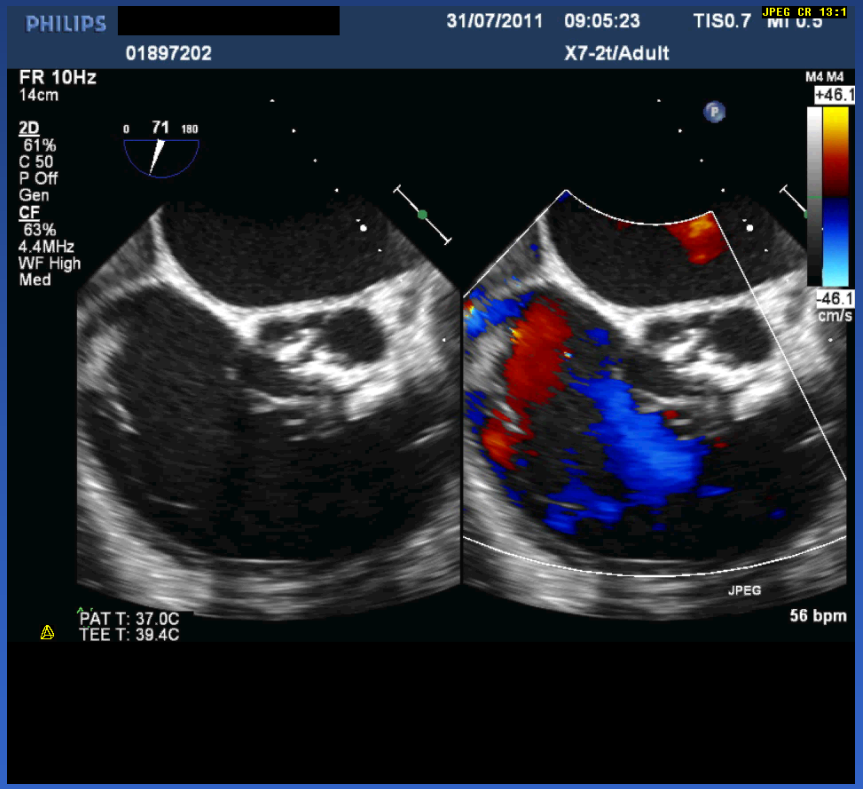
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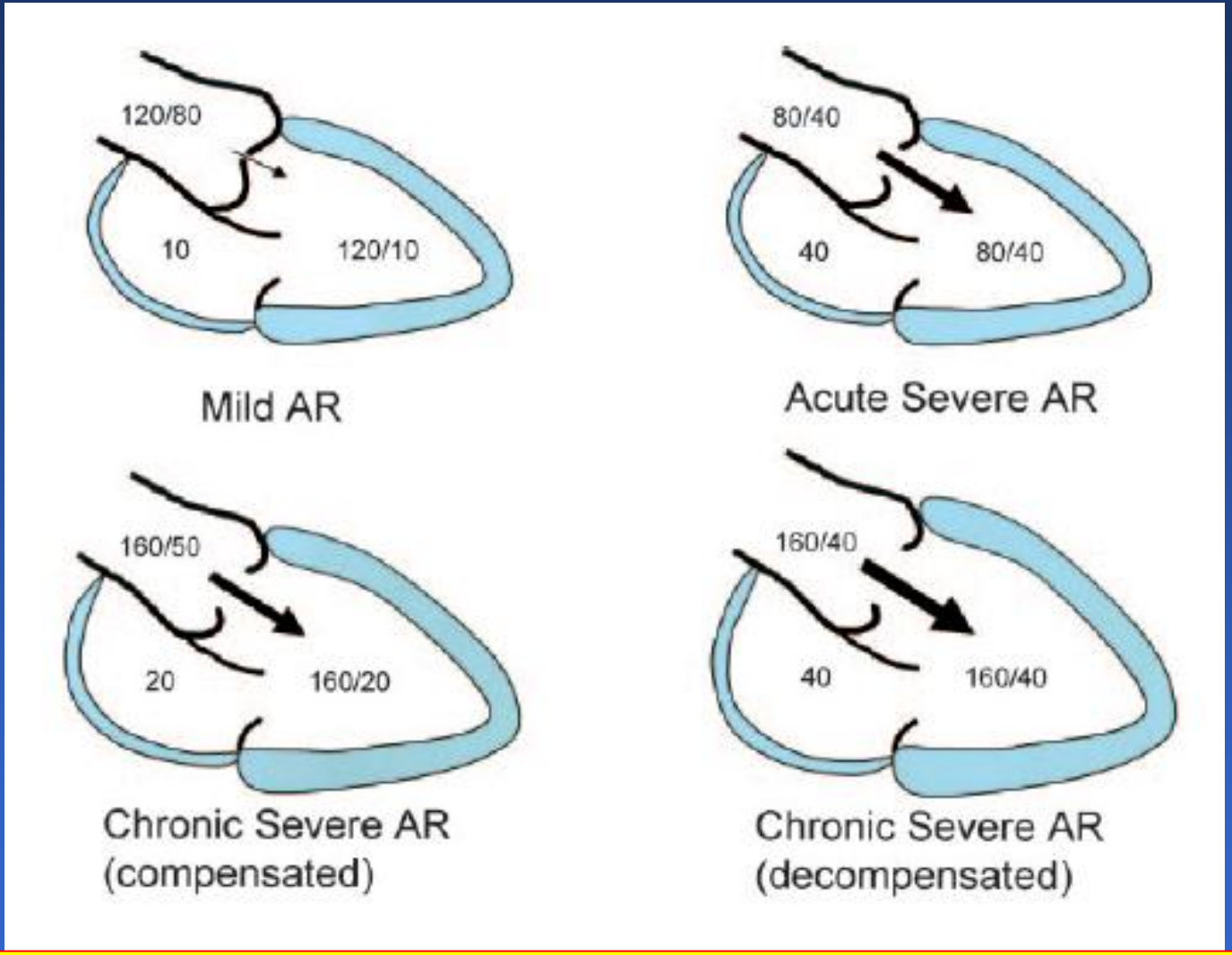
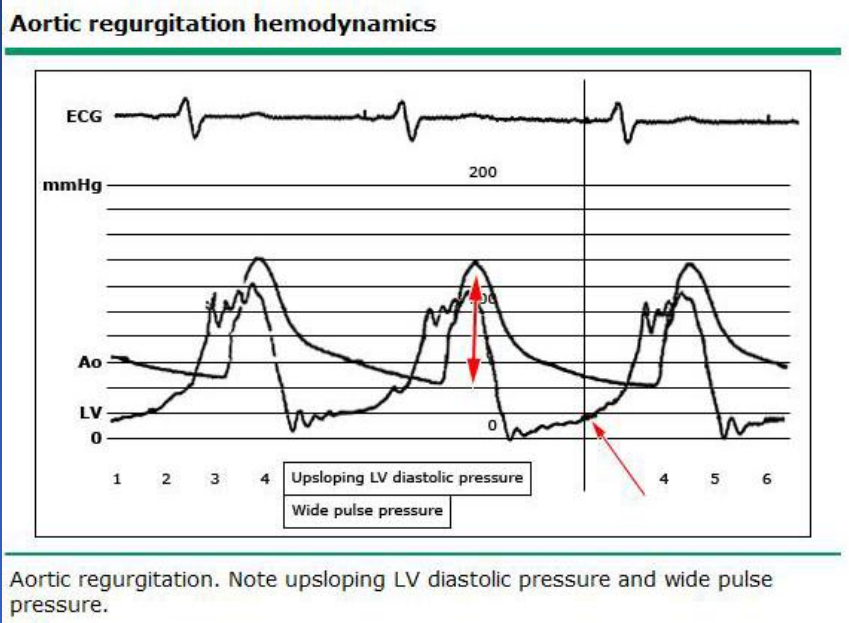
TEE T: 39.4C

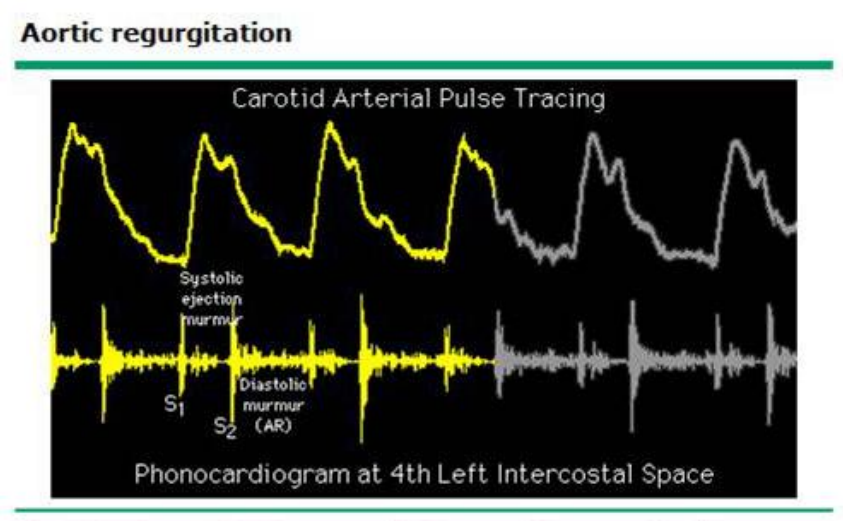
Pathophysiology of AR



Chronic AR is a condition of both volume and pressure overload

Clinical presentation





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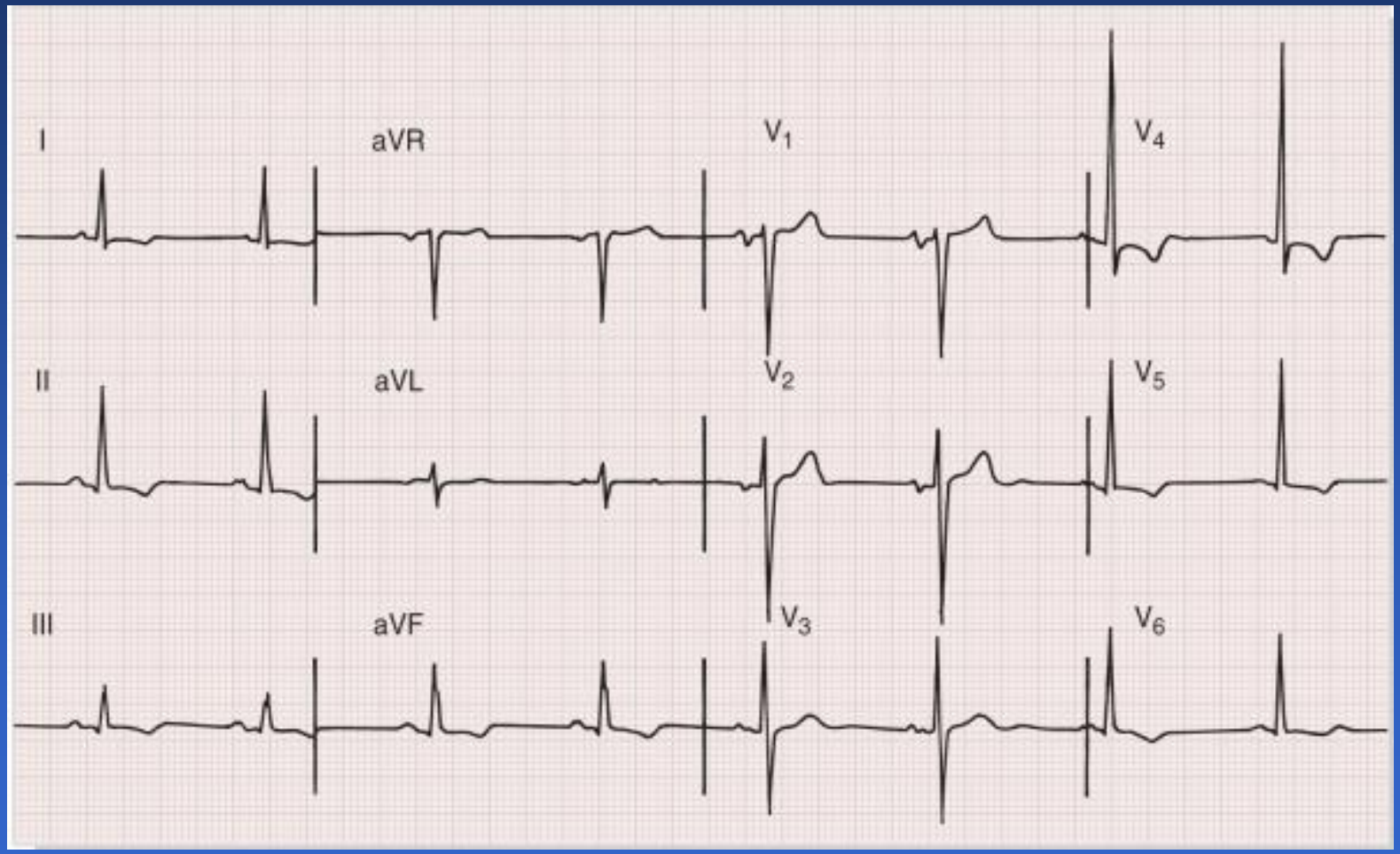


Chen J et al. Am Heart J 1996

Echo assessment of AR severity

- **Aortic valve and root structure**
- **Colour flow Doppler**
- Proximal jet methods
- Vena Contracta
- PISA
- **Pulsed wave Doppler**
- Volumetric methods
- Flow reversal in aorta
- **Continuous wave Doppler**
- Signal density
- Aortic deceleration time (pressure half time)

And always check the consequences of AR on LV size and function

Proximal jet methods

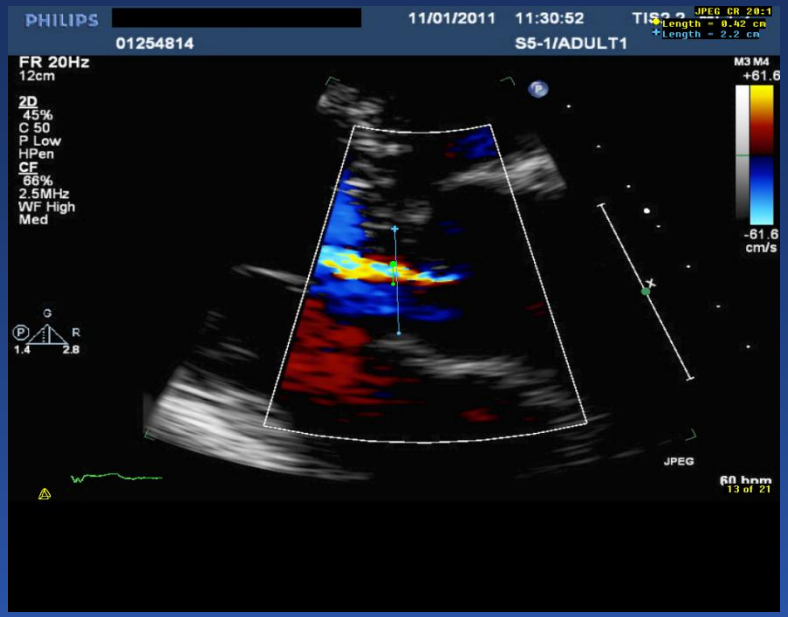
- Semi quantitative approach
- Parasternal views are preferred

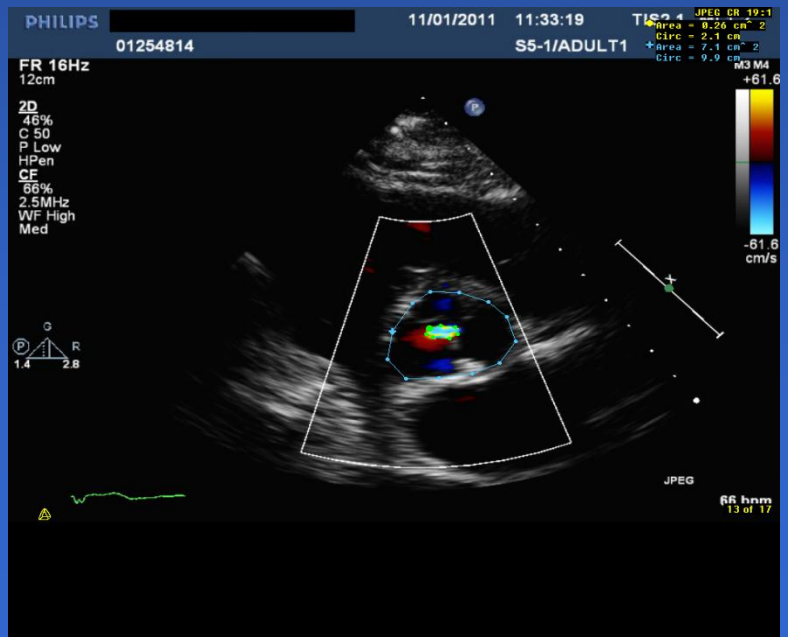
Jet width/LVOT diameter

- Maximum jet diameter is measured at end-diastole immediately below the AV

Jet area/LVOT area

- Measured in the SAX view





Proximal jet methods

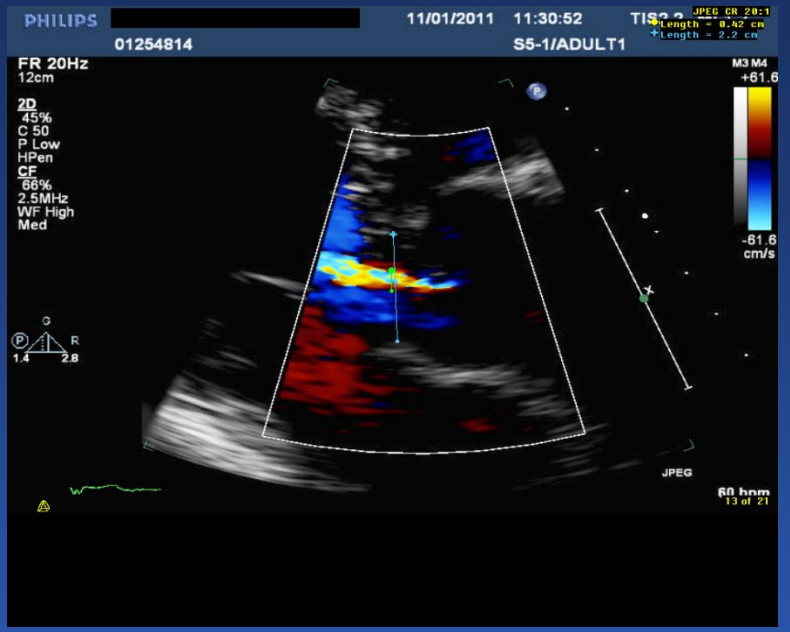
- **Jet width/LVOT diameter values:**
  - < 25% mild AR
  - > 65% severe AR

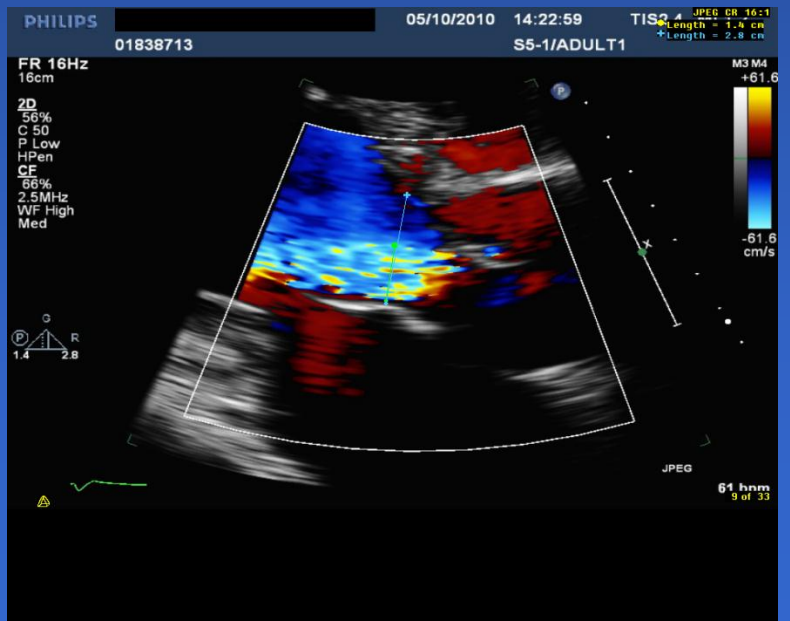
Limitations

- Inaccurate for eccentric jets
- Influenced by technical and haemodynamic factors

In practice visual estimation

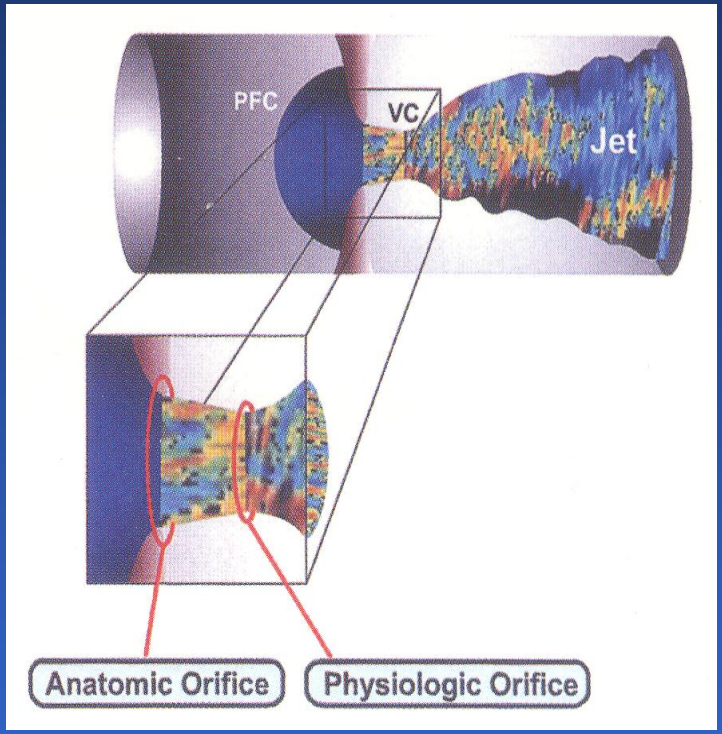
Perry GJ et al. JACC 1987





Vena Contracta

- Narrowest portion of the AR jet, just before the proximal jet
- All three components of the regurgitant jet must be seen
- Best measured in PLAX
- Nyquist limit = 50 – 60 cm/s
- Optimize your image (high FR)



Vena Contracta

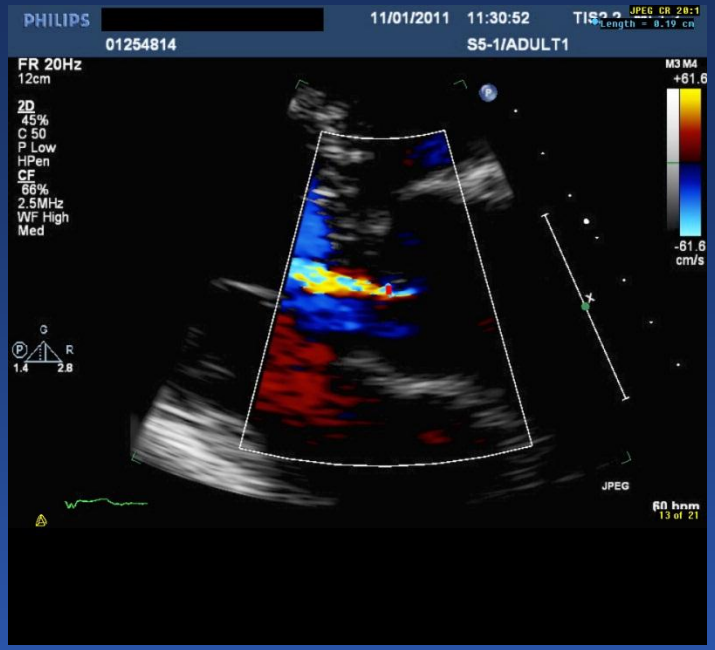
- **Values:**
  < 3 mm mild
  > 6 mm severe

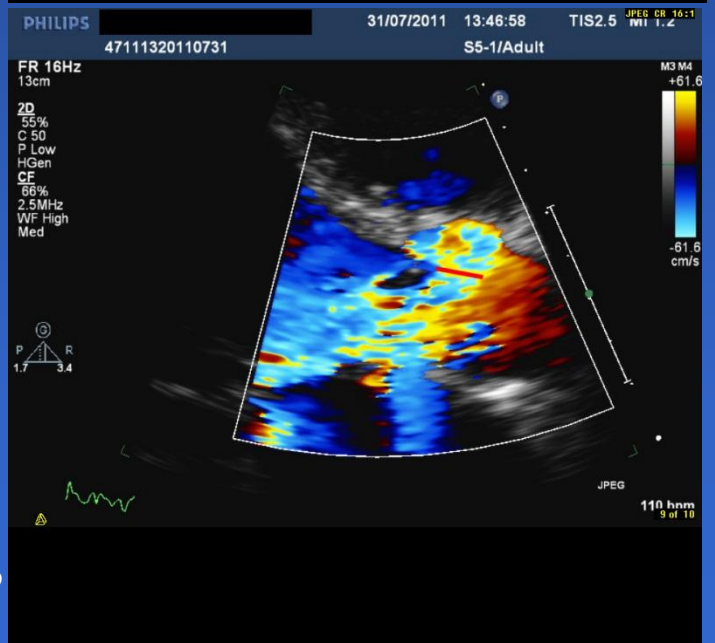
Limitations

- It is often obscured by the leaflets
- Multiple jets
- Small errors → large mistakes

In practice if you don't clearly see the VC don't measure it

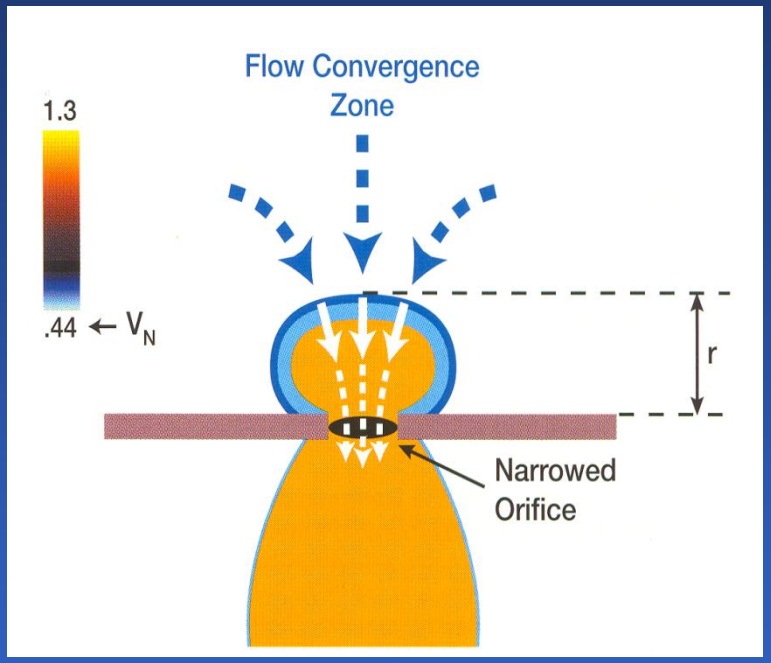
Tribouilloy CM et al. Circulation 2002





PISA or Proximal Flow Convergence

- Less experience than MR
- PISA radius can be obtained from PLAX (eccentric jets better) or apical views
- Optimize your image (high FR – baseline adjustment)
- Peak a frame at early diastole



PISA or Proximal Flow Convergence

- Values

ERO < 0.1 cm² or RV < 30 ml: mild

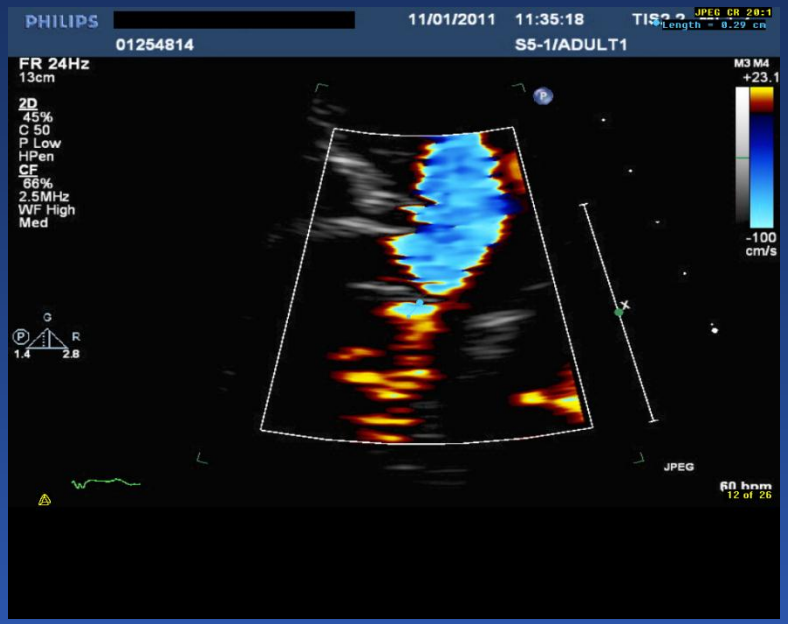
ERO > 0.3 cm² or RV > 60 ml: severe

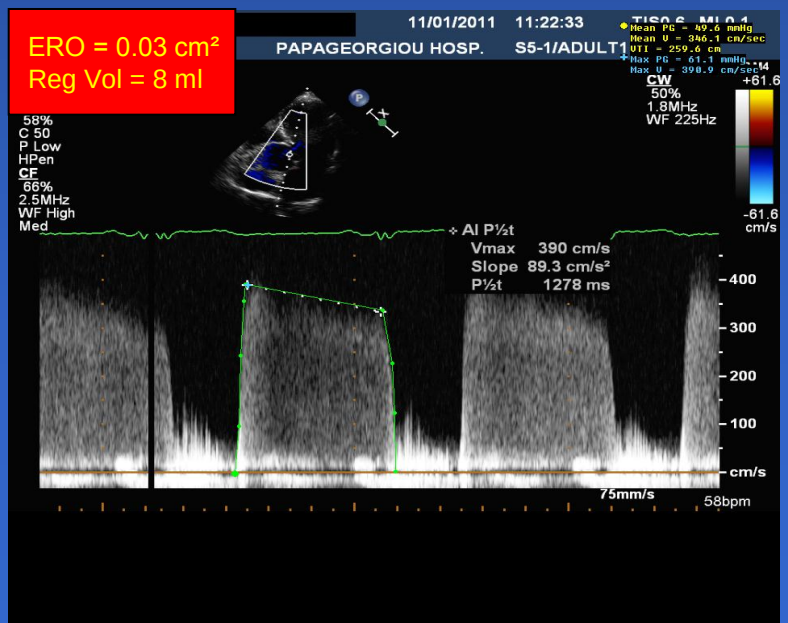
Limitations

- Very often interposition of valve tissue
- Error in PISA radius is squared

In practice it is used when the severity of AR is not determined by other simpler echo data

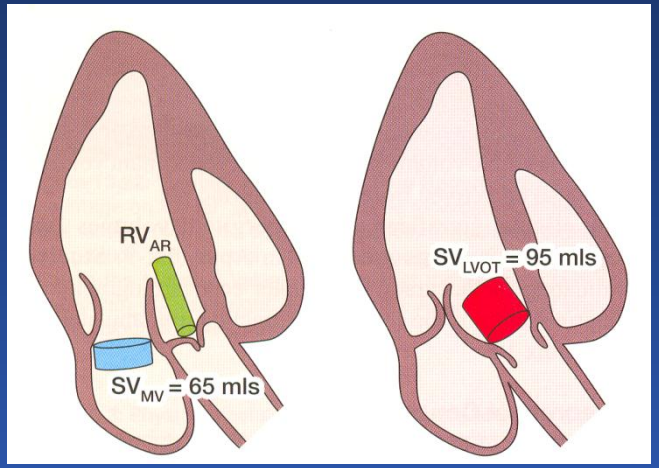
Tribouilloy CM et al. JACC 1998





Volumetric method

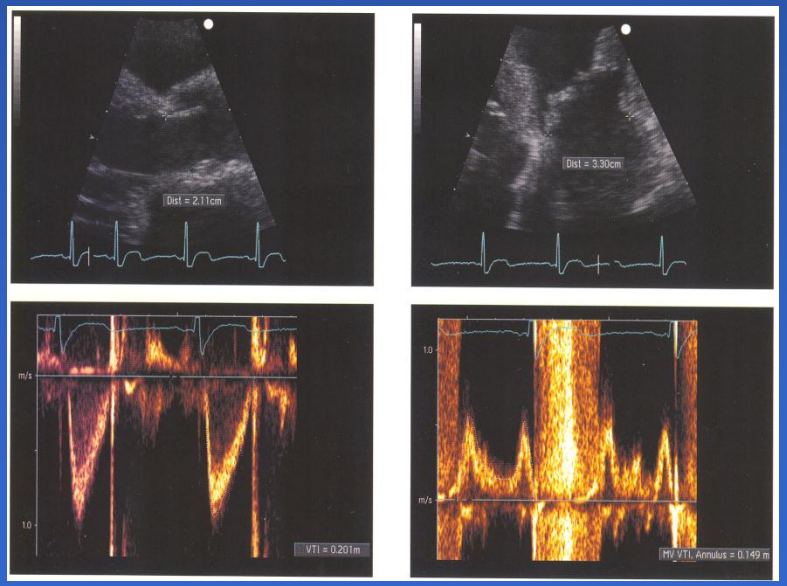
- Based on the calculation of the stroke volume through the AV and another competent valve (usually MV)



Limitations

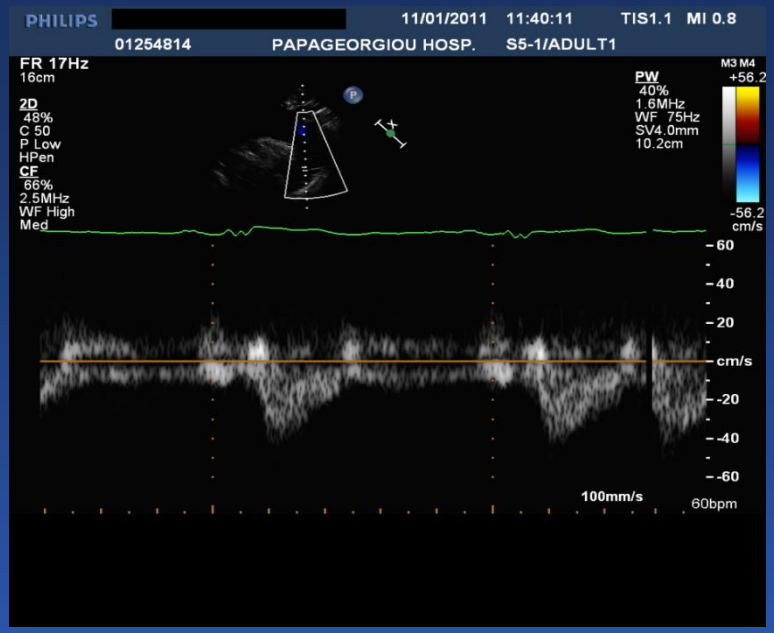
- Lots of theoretical **assumptions**
- Error in diameter is **squared**
- Invalid when there is more than mild MR
- Time consuming
- **Significant learning curve**

Requires significant expertise



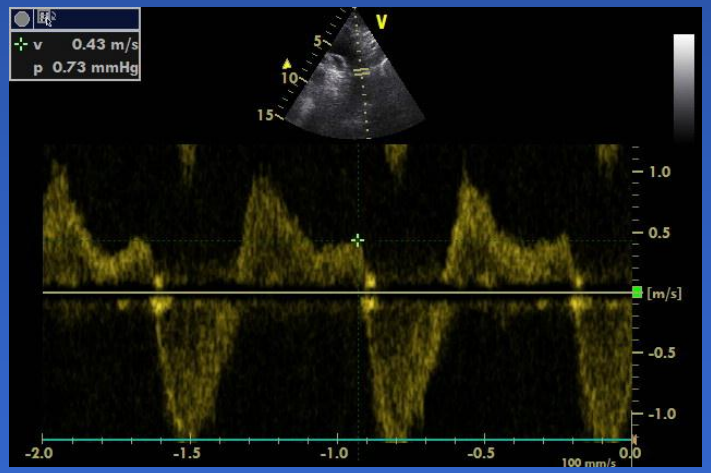
Holodiastolic flow reversal in aorta

- Very sensitive and specific sign of significant AR
- ED velocity > 20 cm/s
- Simple



Limitations

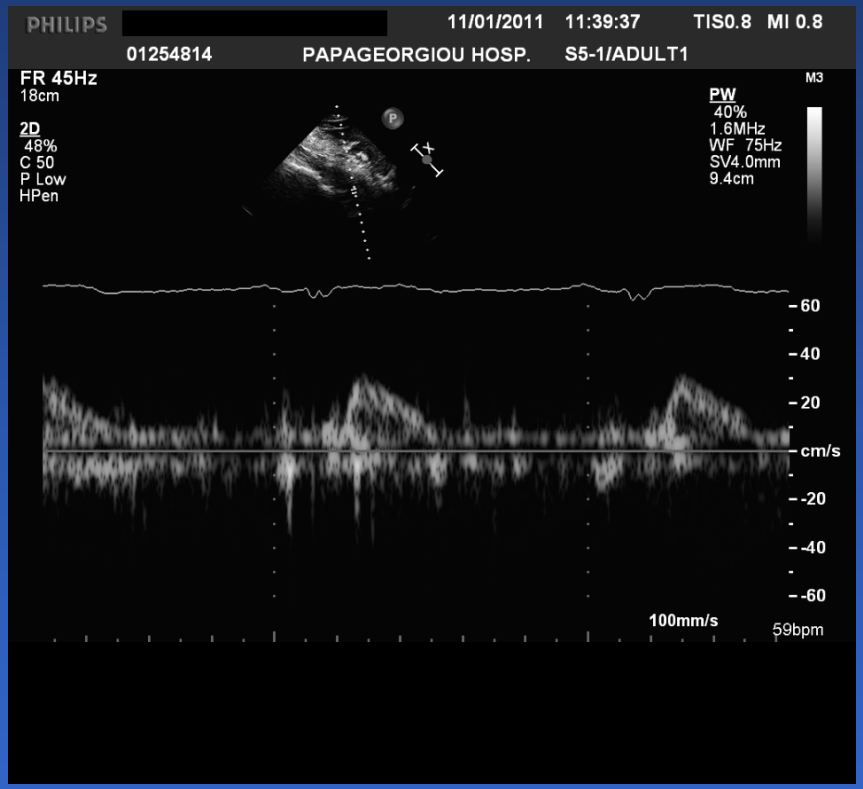
- This sign can also be seen in aorta aneurysm or dissection
- Difficult to assess with ↑ HR

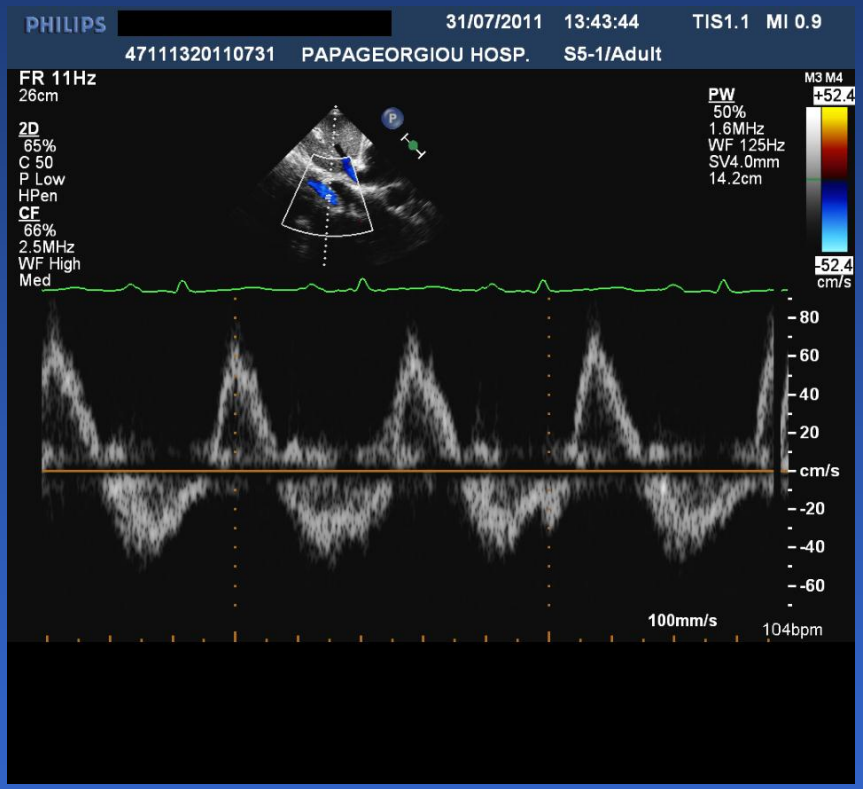


In practice one of the most useful parameters in the assessment of AR severity

Tribouilloy CM et al. Br Heart J 1991

Flow reversal in the abdominal aorta

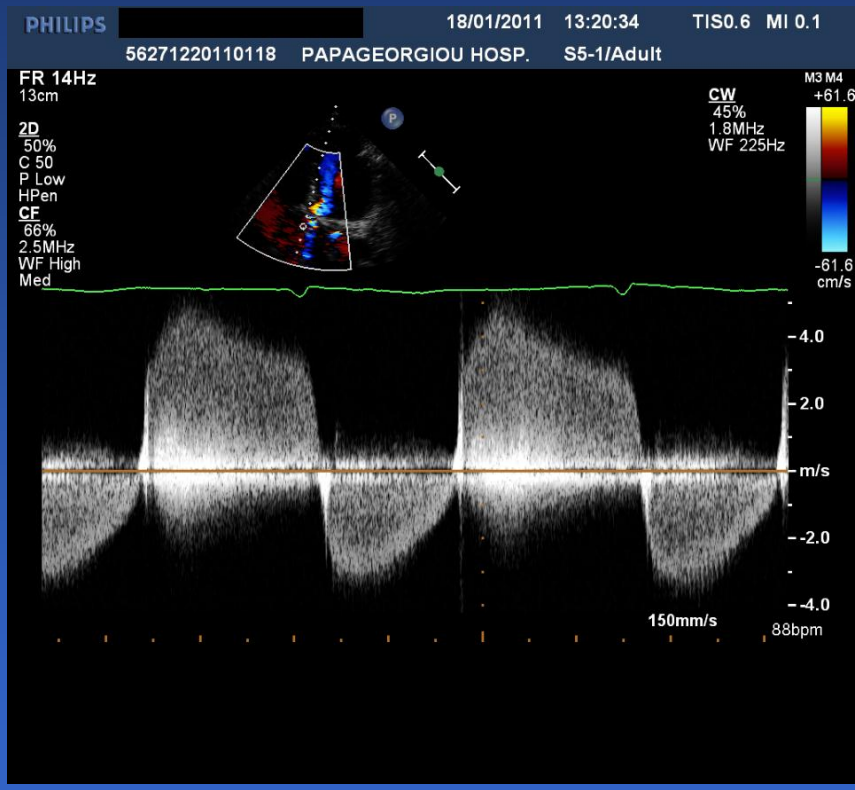




Takenaka K et al. Am J Cardiol 1986

Continuous Wave AR Signal density

- Reflects the volume of regurgitation
- Faint signal → mild AR



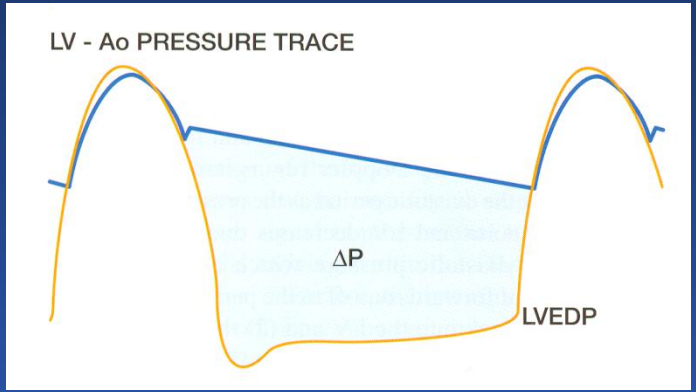
Limitations

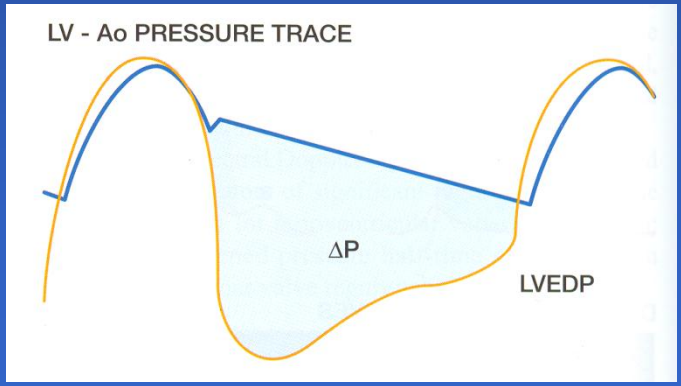
- Eccentric jets
- Significant overlap between moderate and severe AR

In practice it is complementary to the all the above parameters

Aortic deceleration time (PHT)

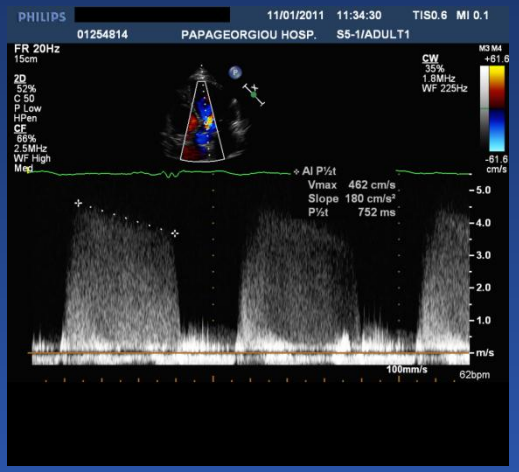
- Reflects the rate of equalization of aortic and LV diastolic pressures
- ↑ severity AR → Slope become steeper
- Very useful in acute AR

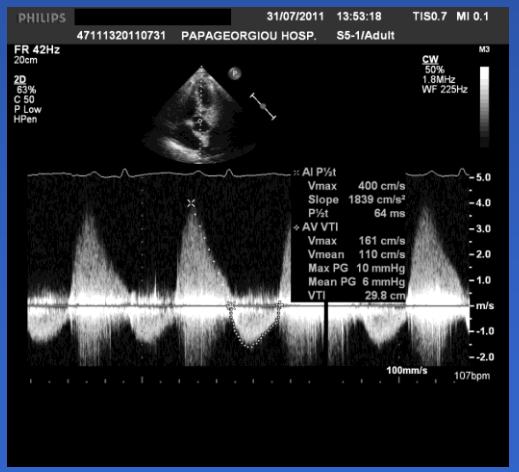




Aortic deceleration time (PHT)

- PHT Values
  > 500 ms → mild AR
  < 200 ms → severe AR





Limitations

- Stiff, **non-compliant ventricle**
- Tends to **normalize** with **chronic** LV adaptation to severe AR

In clinical practice it is used complementary with the other parameters

Left ventricular size and function

- Pressure and volume overload
- In acute AR → LV usually has normal size
- In chronic AR → LV progressively dilates and eventually fails
- Clinical decision making

Summary of echo assessment of AR

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

EAE recommendations. Eur J Echocardiogr 2010

Proposed algorithm

Visual Colour Flow assessment → **mild AR**

↓ **> mild AR**

Vena Contracta > 6 mm and/or
Holodiastolic flow reversal in aorta
(dense signal and PHT < 500)

→ **severe AR**
YES
NO
→ **moderate AR**

↓ **conflicting or suboptimal values**

Impaired LV size and function
(PISA and/or Doppler Volumetric method)

→ **severe AR**
YES
NO
→ **?moderate AR**

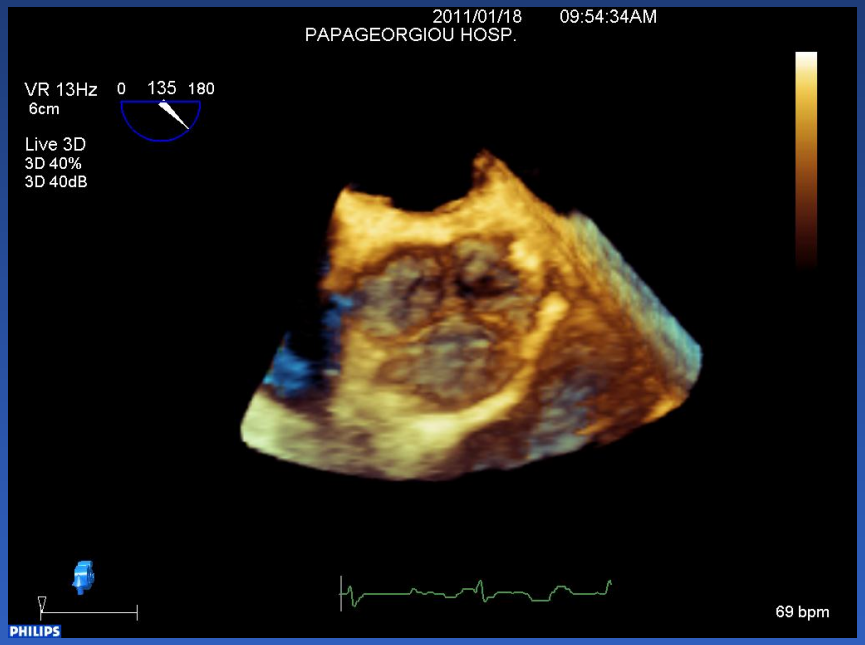
Comparison of Semiquantitative and Quantitative Assessment of Severity of Aortic Regurgitation: Clinical Implications

area ≥ 30 mm^2 or regurgitant volume ≥ 60 mL). Overall, semiquantitative methods had good specificity but poor sensitivity, except the vena contracta, which had good sensitivity and specificity. Sensitivity, specificity, and positive and negative predictive values of the recommended thresholds for severe AR of the four semi-quantitative methods were 53%, 89%, 77%, and 73% for left ventricular cardiac output ≥ 10 L/min; 12%, 100%, 100%, and 52% for pressure half-time < 200 msec; 45%, 87%, 79%, and 60% for diastolic flow reversal ≥ 18 cm/sec; and 81%, 83%, 78%, and 85% for vena contracta ≥ 6 mm, respectively.

Mesika-Zeitoun D. JASE 2011

What about new echo modalities?

- 3D Promising but not yet of proven benefit in clinical practice
- TDI or strain rate for early LV dysfunction
- The additional value of exercise echocardiography is also not well established

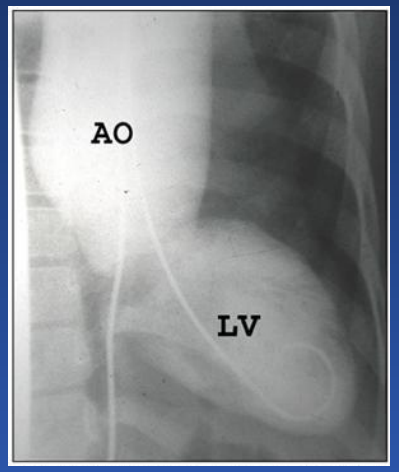


Fang L et al. Echocardiography 2005
Vinereanu D et al. Heart 2001
Pierard LA et al. Heart 2007

Other modalities

Cardiac catheterisation

- Still commonly used
- Does not provide true quantitation



MRI

- Very promising
- Not widely available

Their use is limited in cases of uncertainty with echo

Cawley PJ et al. Minerva Cardioangiol 2009

ACC/AHA guideline summary: Diagnosis and initial evaluation of aortic regurgitation (AR)

Class I - There is evidence and/or general agreement that the following tests are indicated for the diagnosis and initial evaluation in patients with AR

- Echocardiography
  1. To confirm the diagnosis and to estimate the severity of acute or chronic AR.
  2. To determine the cause of chronic AR, including assessment of valve morphology and the size and morphology of the aortic root.
  3. To determine left ventricular function and detect left ventricular hypertrophy and left ventricular dimensions or volumes in chronic AR.
  4. Among patients with an enlarged aortic root, to assess both AR and the severity of aortic dilatation.
  5. Among asymptomatic patients with chronic AR, to periodically reevaluate left ventricular size and function.
  6. To reassess mild, moderate, or severe AR in patients who have new or changing symptoms.
- Radionuclide angiography or magnetic resonance imaging
  1. For the initial and follow-up assessment of left ventricular volume and function at rest when echocardiography is suboptimal.
- Cardiac catheterization
  1. With aortic root angiography and measurement of left ventricular pressure to assess the severity of AR, left ventricular function, and aortic root size when noninvasive tests are inconclusive or provide discrepant results from clinical findings.

Class IIa - The weight of evidence or opinion is in favor of efficacy of the following tests in patients with AR in the following settings

- Exercise stress testing in chronic AR
  1. To assess functional capacity and symptom response in patients with a history of equivocal symptoms.
  2. To assess symptoms and functional capacity prior to participation in athletic activity.
- Magnetic resonance imaging
  1. To assess the severity of AR when echocardiography is not satisfactory.

Class IIb - The weight of evidence or opinion is less well established for the following test in patients with AR in the following setting

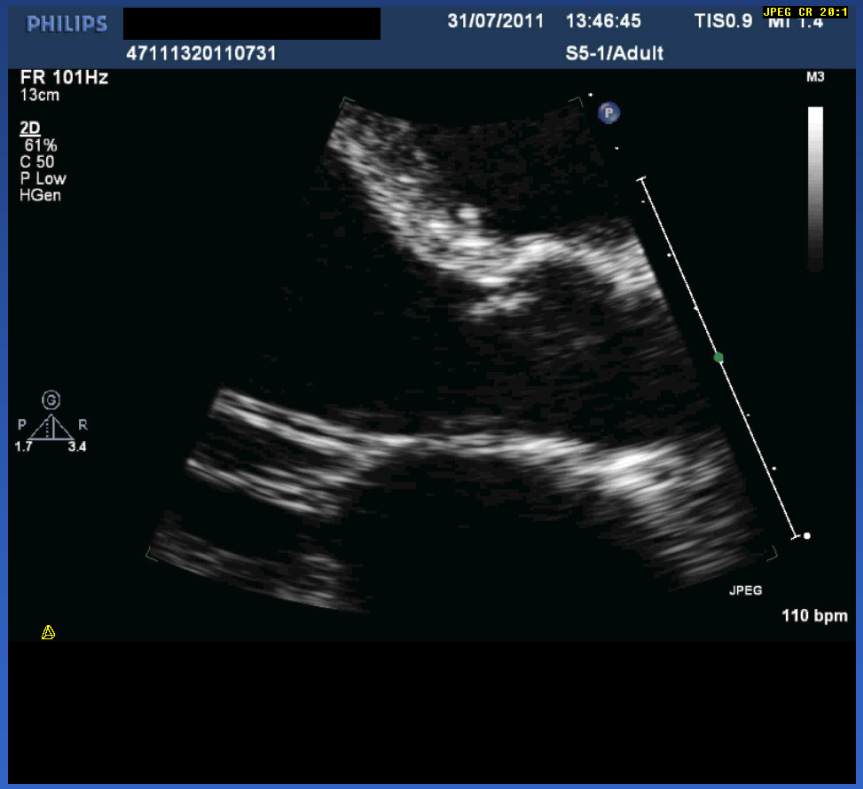
- Exercise stress testing with radionuclide angiography
  1. To assess left ventricular function in asymptomatic or symptomatic patients with chronic AR.

Class III - There is evidence and/or general agreement that the following test is not useful in patients with AR in the following settings

- Cardiac catheterization
  1. When noninvasive tests are adequate and consistent with clinical findings and coronary angiography is not required.
  2. In asymptomatic patients in whom noninvasive tests are adequate.

AHA/ACC Guidelines 2006

Case 1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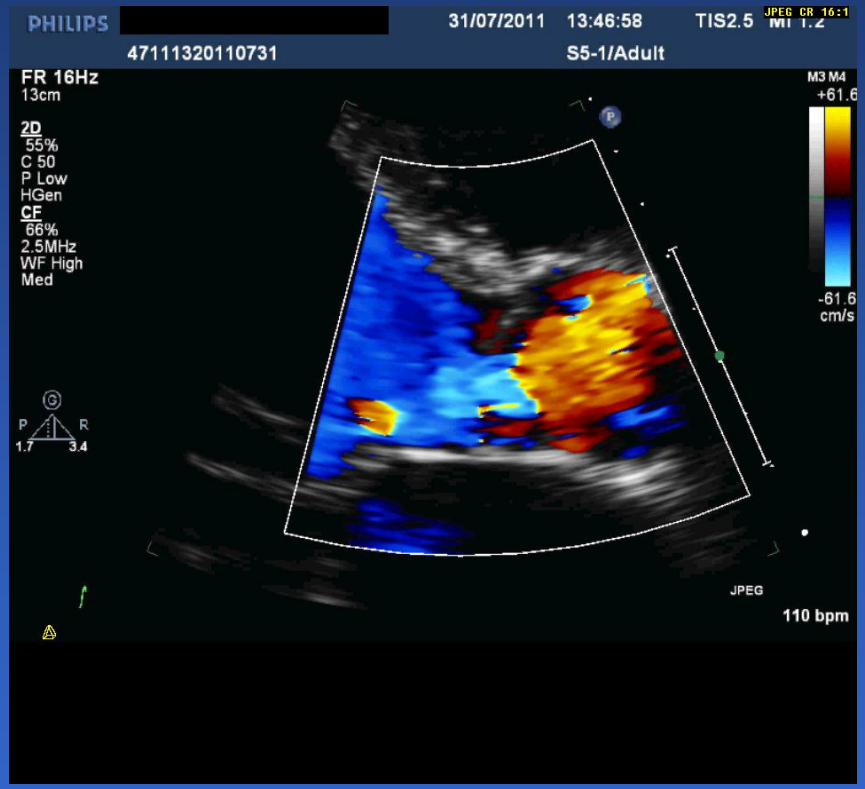
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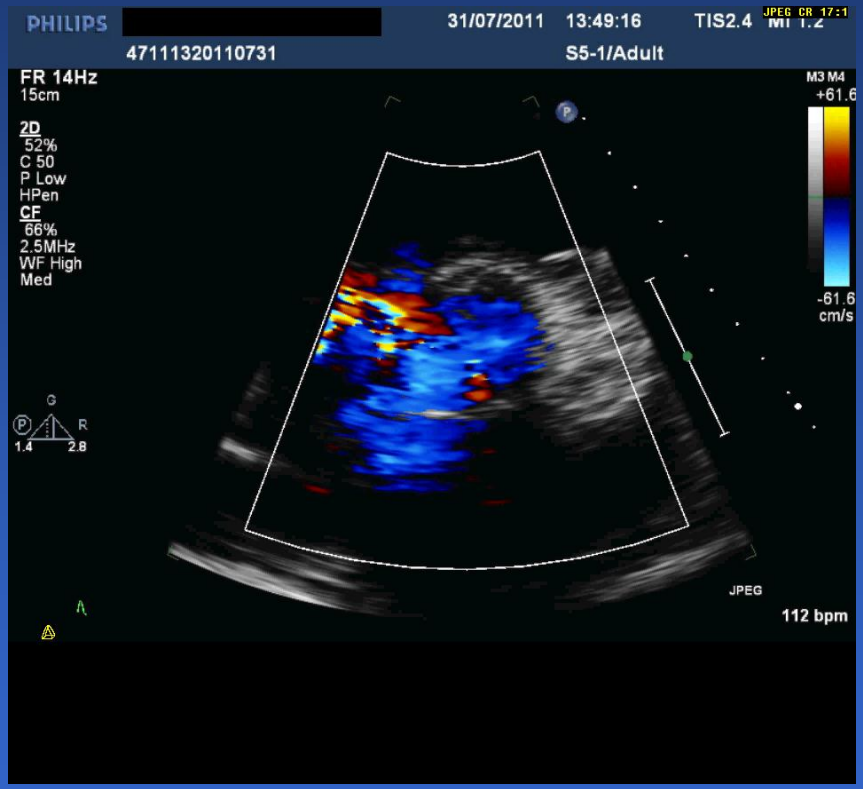
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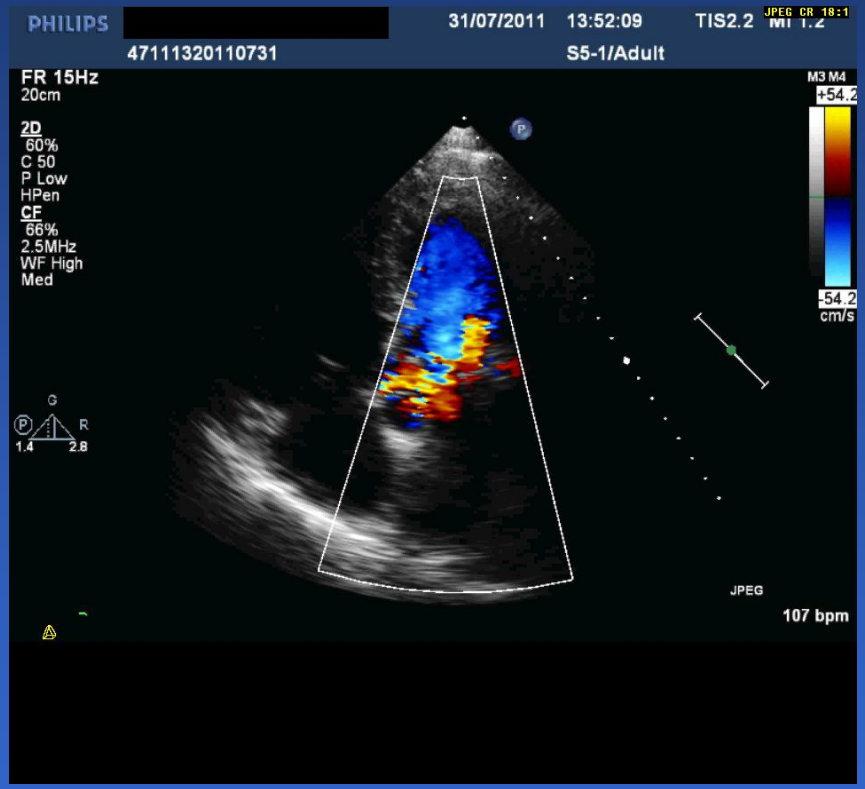
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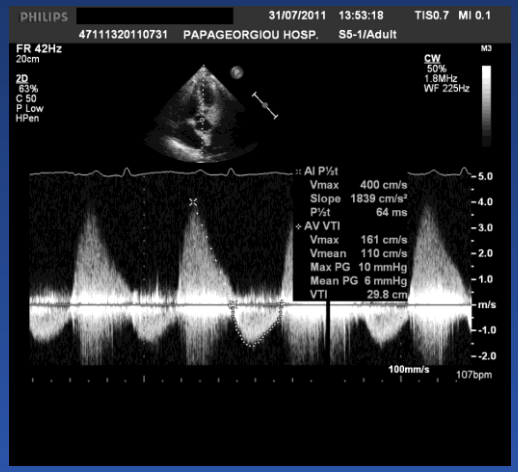
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P½t 64 ms
AV VTI
Vmax 161 cm/s
Vmean 110 cm/s
Max PG 10 mmHg
Mean PG 6 mmHg
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m/s
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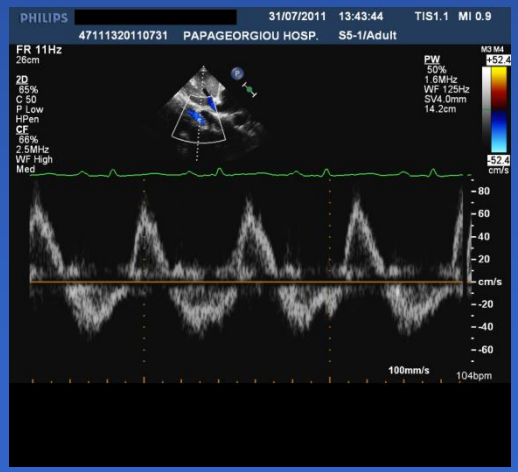
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14.2cm
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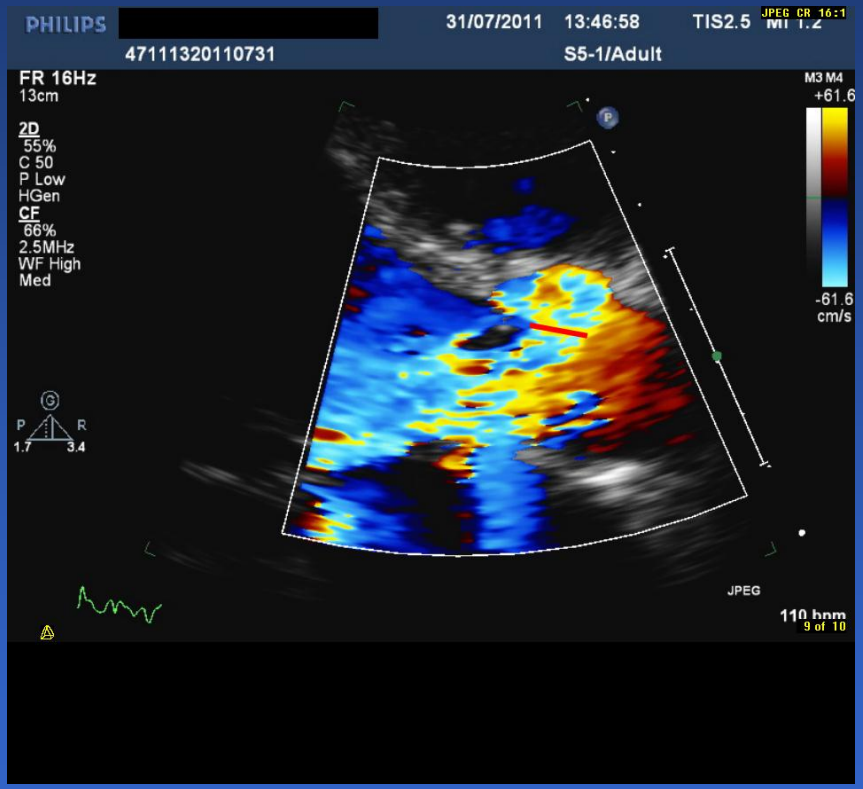
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R
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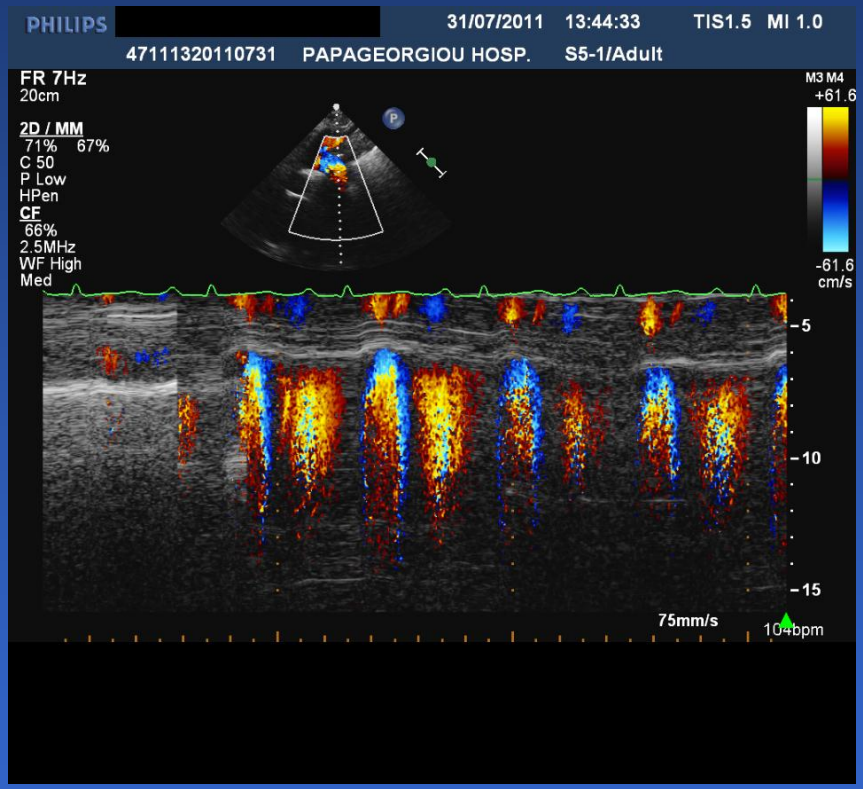
9 of 10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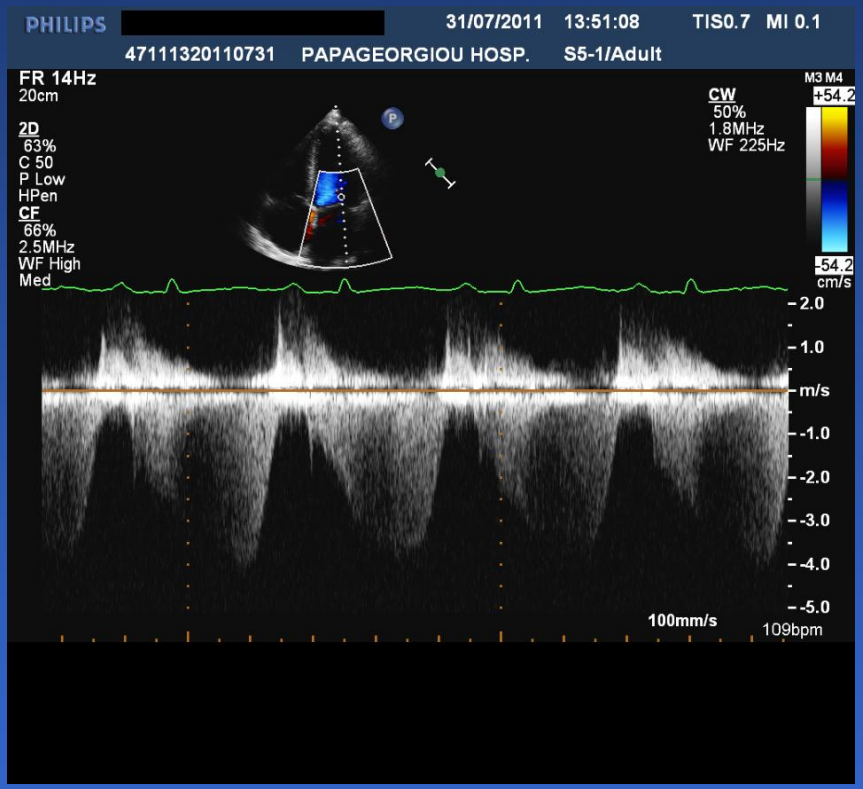
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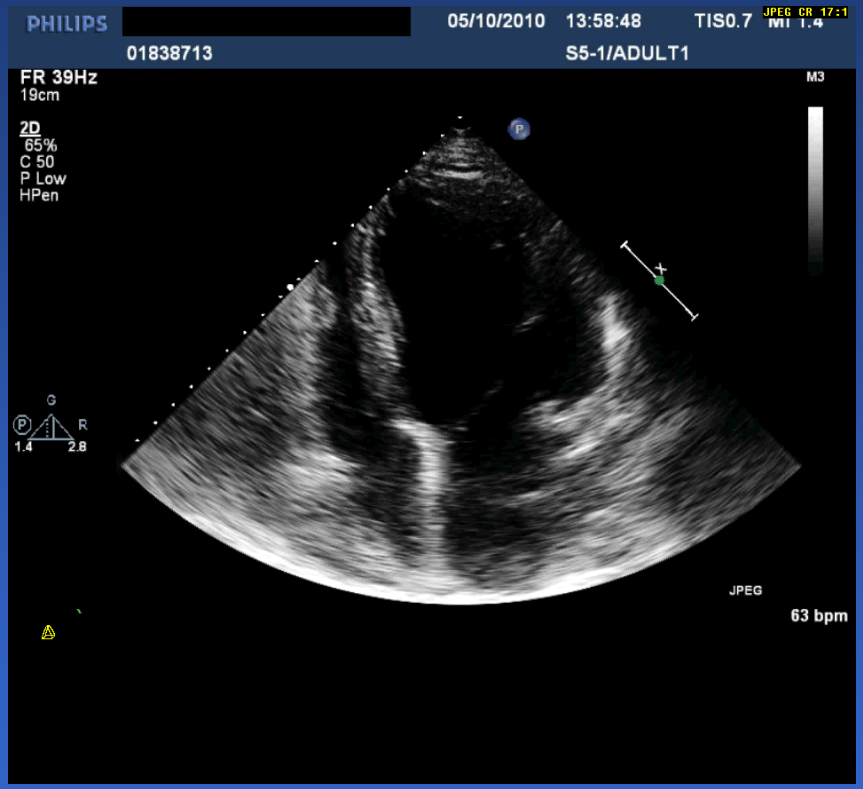
Case 2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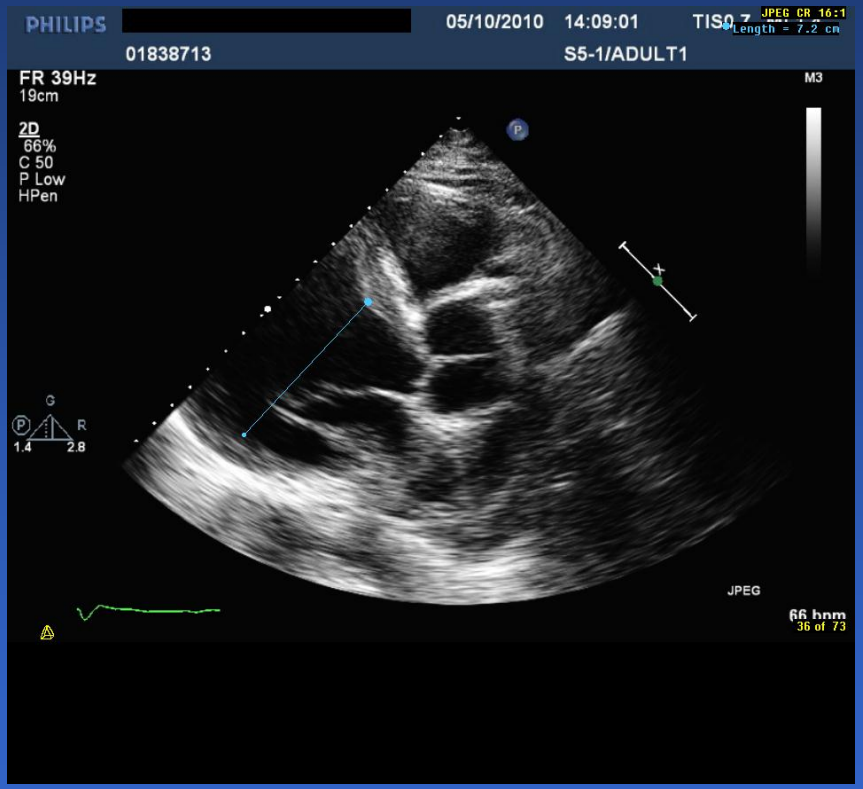
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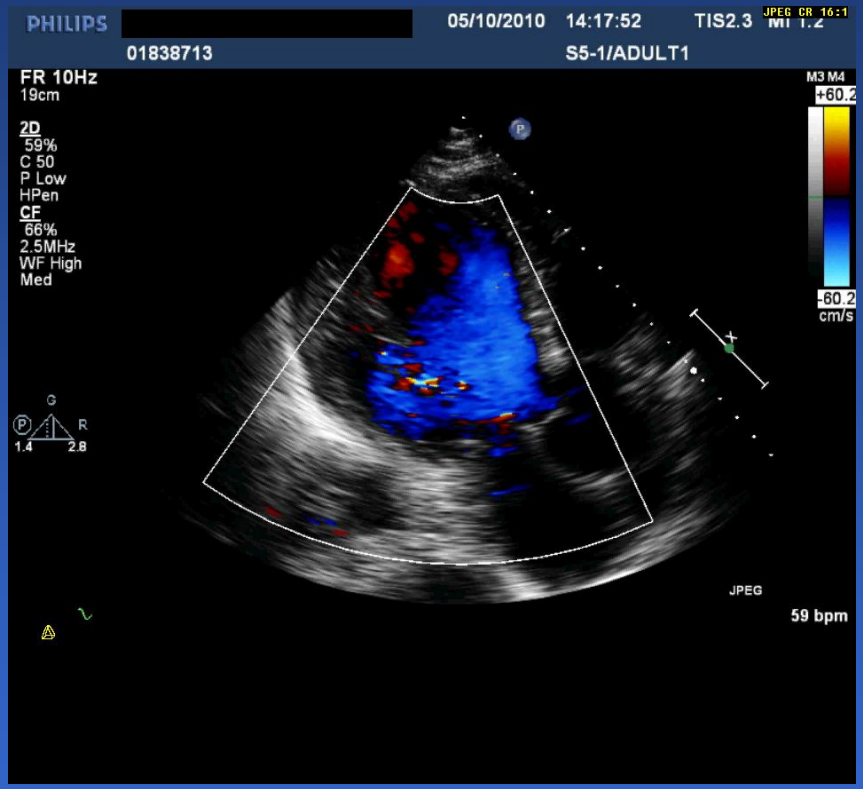
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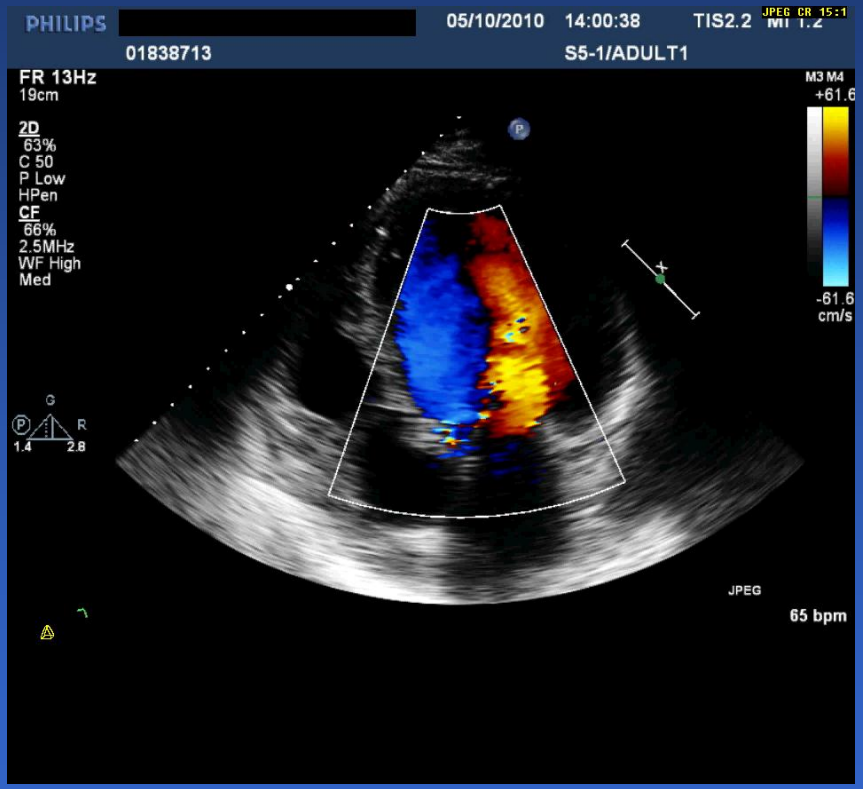
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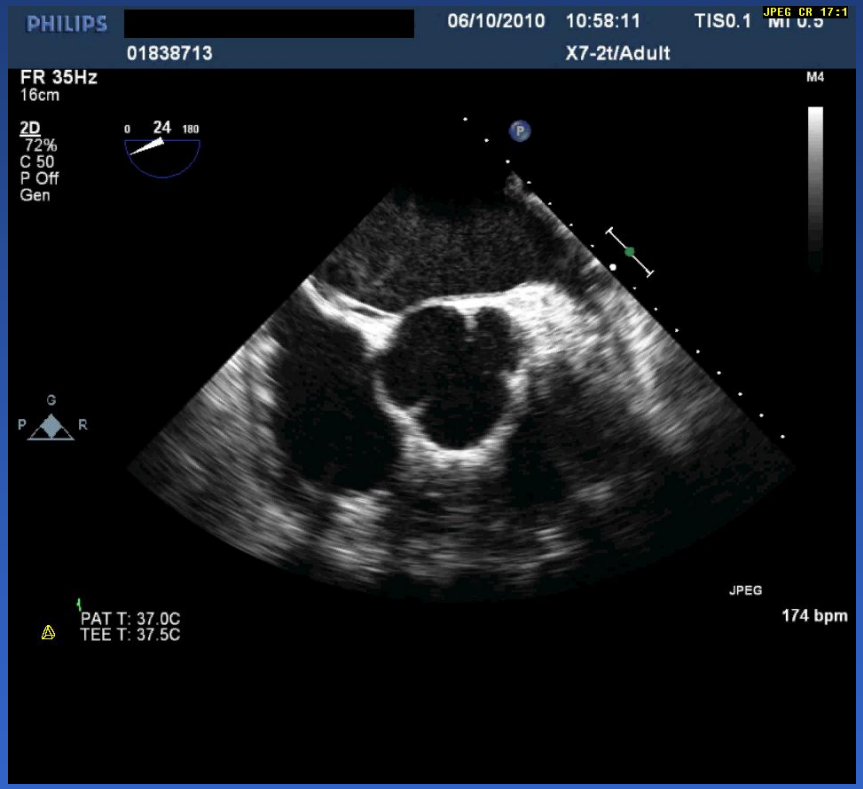
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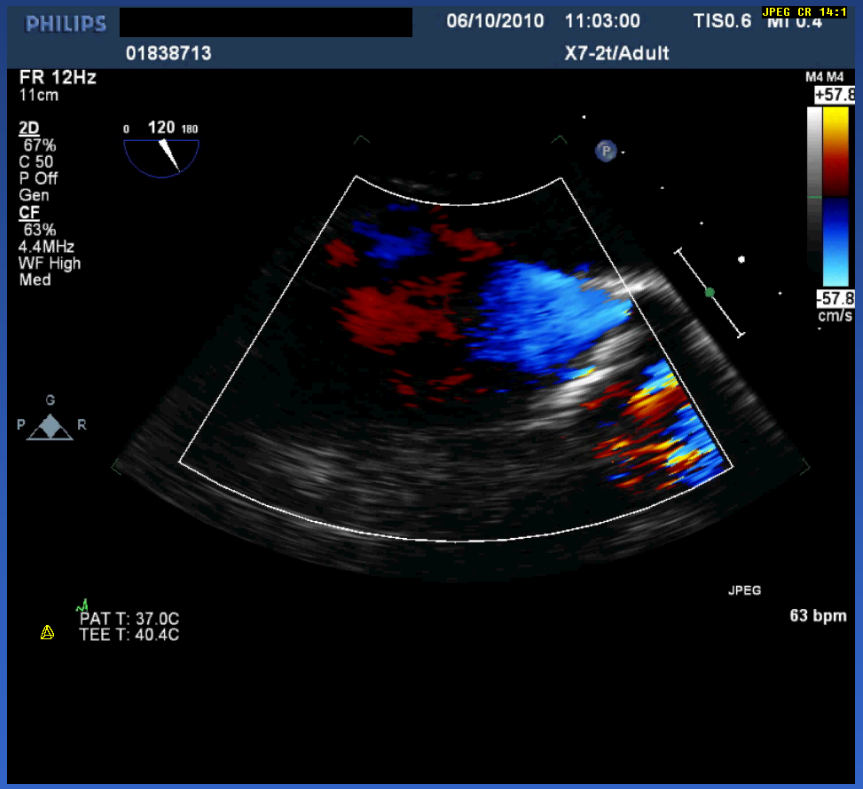
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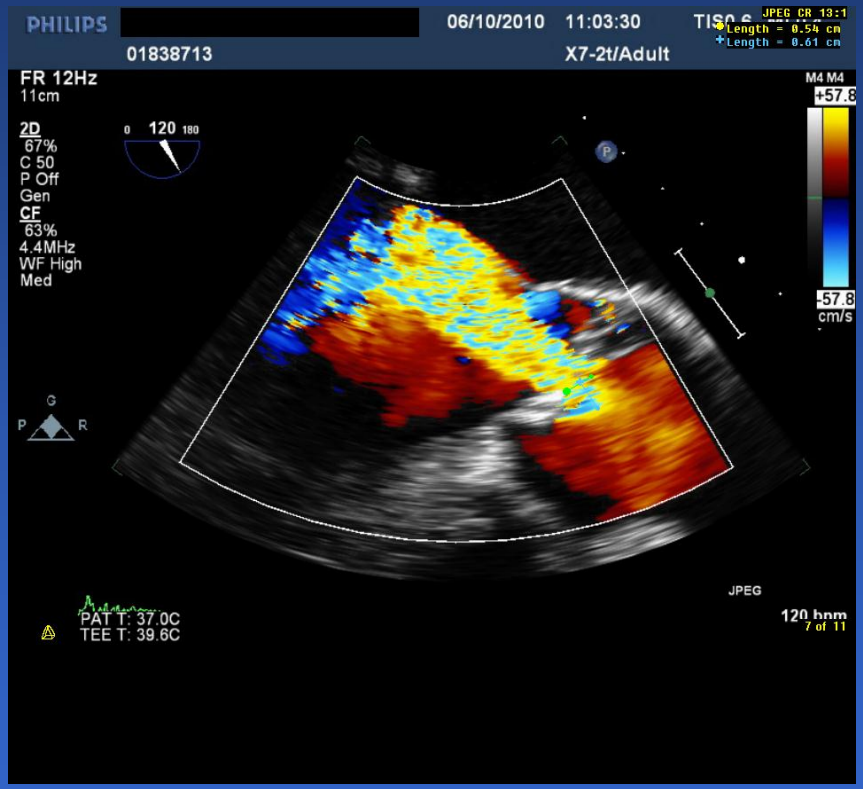
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TEE T: 39.6C
JPEG
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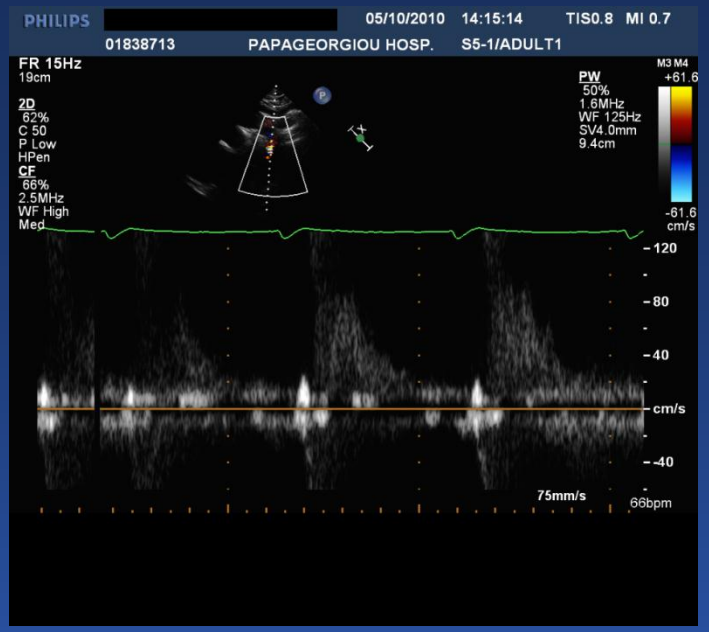
7 of 11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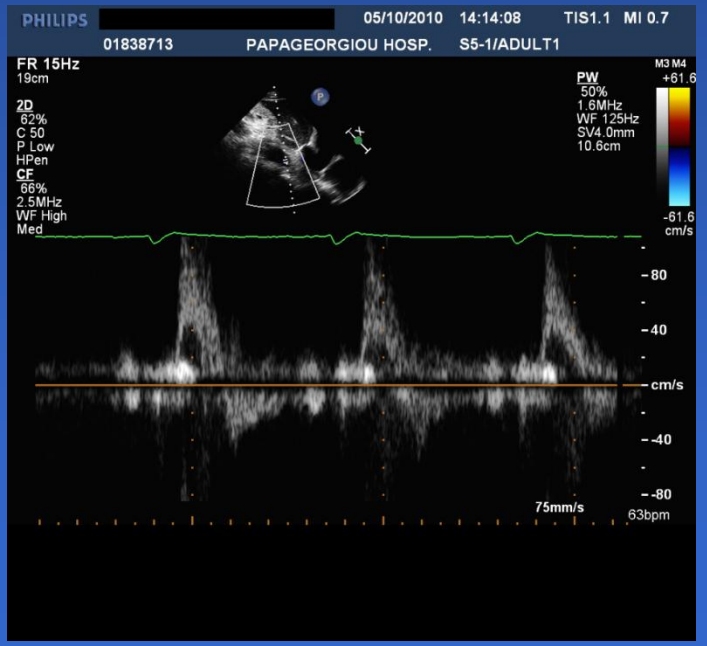
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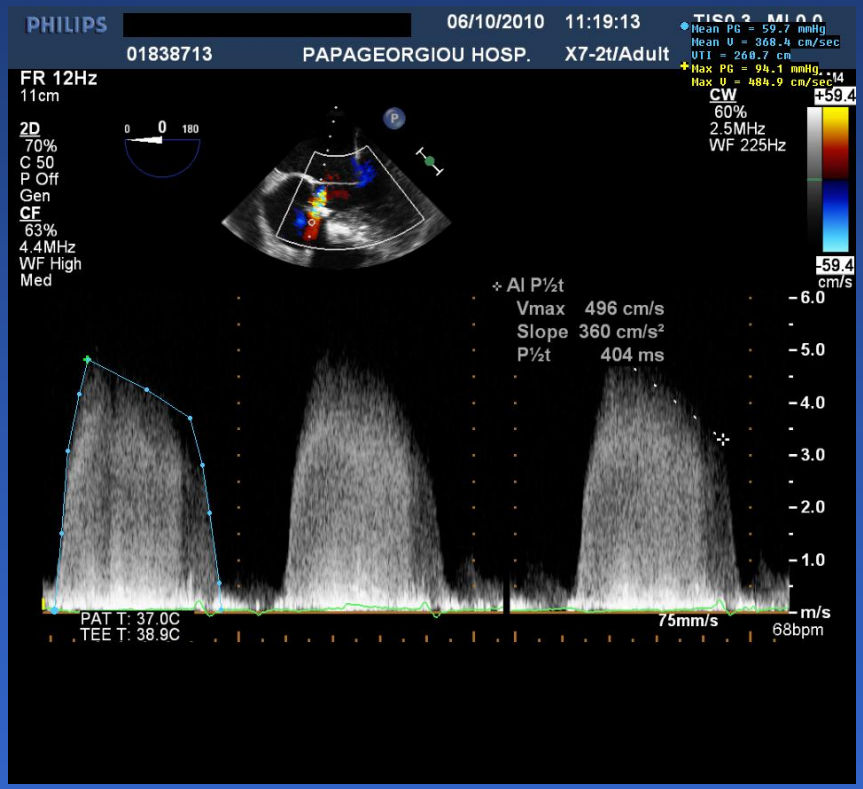
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11cm
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C 50
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-59.4
cm/s
AI P½t
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Slope 360 cm/s²
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6.0
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TEE T: 38.9C
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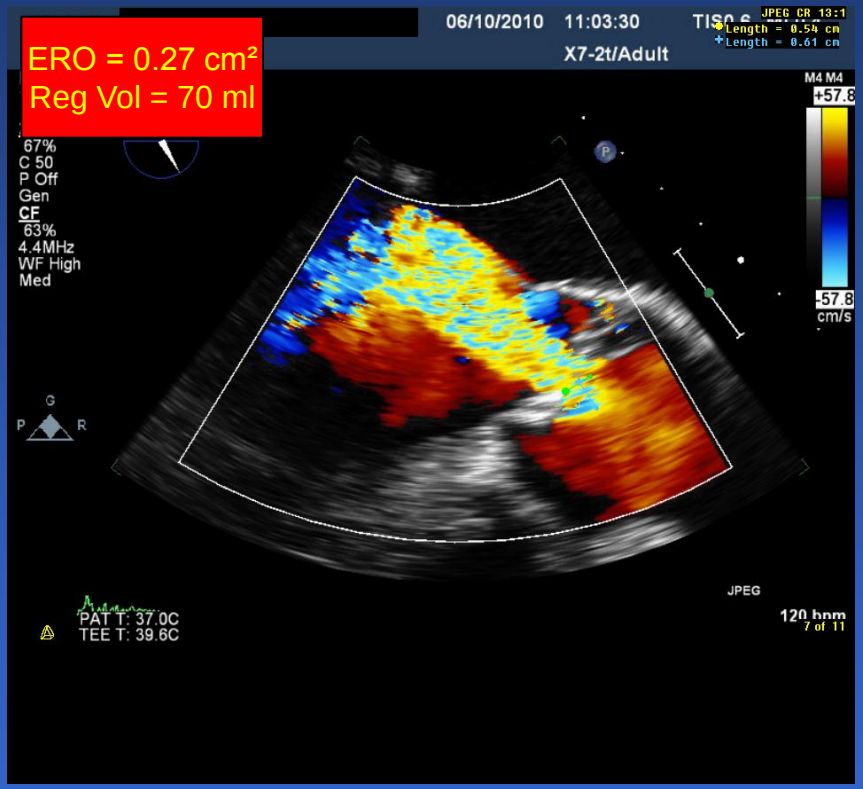
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-57.8
cm/s
PAT T: 37.0C
TEE T: 39.6C
JPEG
120 bpm
7 of 11

Management of AR

Treatment options for acute severe AR

- **Death is common** in acute severe AR, even with intensive medical management

- **Temporary interventions** as a bridge to surgery:
- Nitroprusside
- Inotropic agents (dopamine or dobutamine)

Intra-aortic balloon counterpulsation is contraindicated

Treatment = urgent surgical intervention

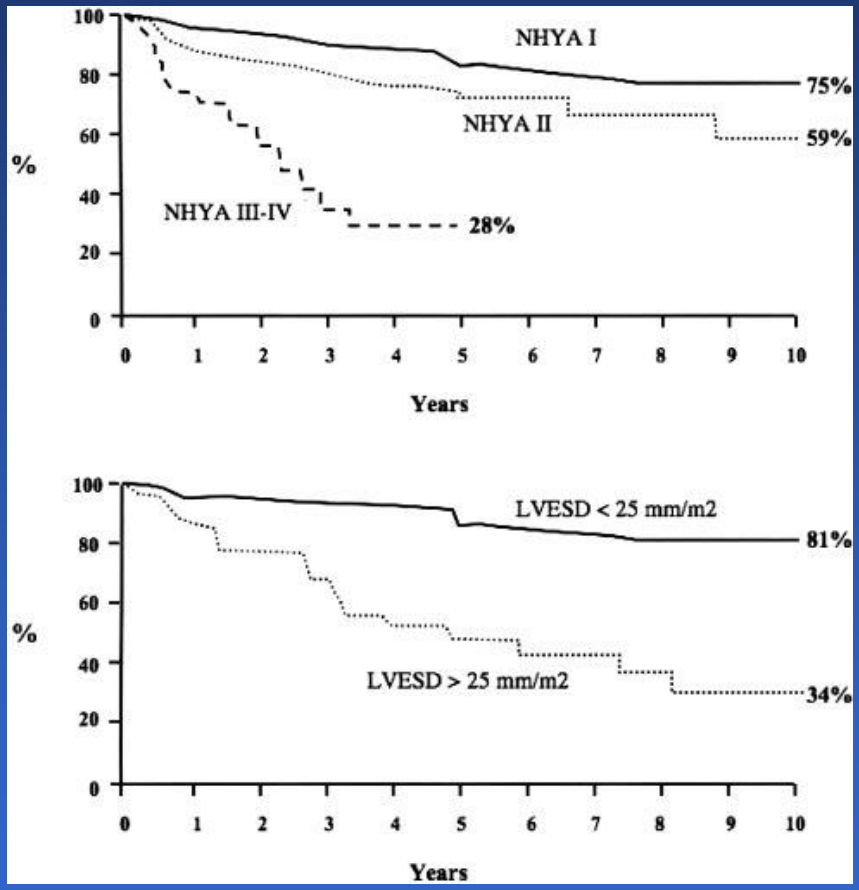
ESC Guidelines 2007

Natural History of chronic AR

Natural history of chronic aortic regurgitation mostly based upon data from nine series with a total of 593 patients followed for a mean of 6.6 years

Asymptomatic patients with normal left ventricular (LV) systolic function	
Progression to symptoms and/or LV dysfunction	Less than 6 percent/year
Progression to symptoms, LV dysfunction, or death	
According to LV end-systolic dimension*	
>50 mm	19 percent/year
40 to 50 mm	6 percent/year
<40 mm	0 percent/year
Progression to asymptomatic LV dysfunction	Less than 3.5 percent/year
Sudden death	Less than 0.2 percent/year
Asymptomatic patients with LV systolic dysfunction	
Progression to cardiac symptoms	More than 25 percent/year
Symptomatic patients	
Mortality rate	More than 10 percent/year

ACC/AHA Guidelines 2006

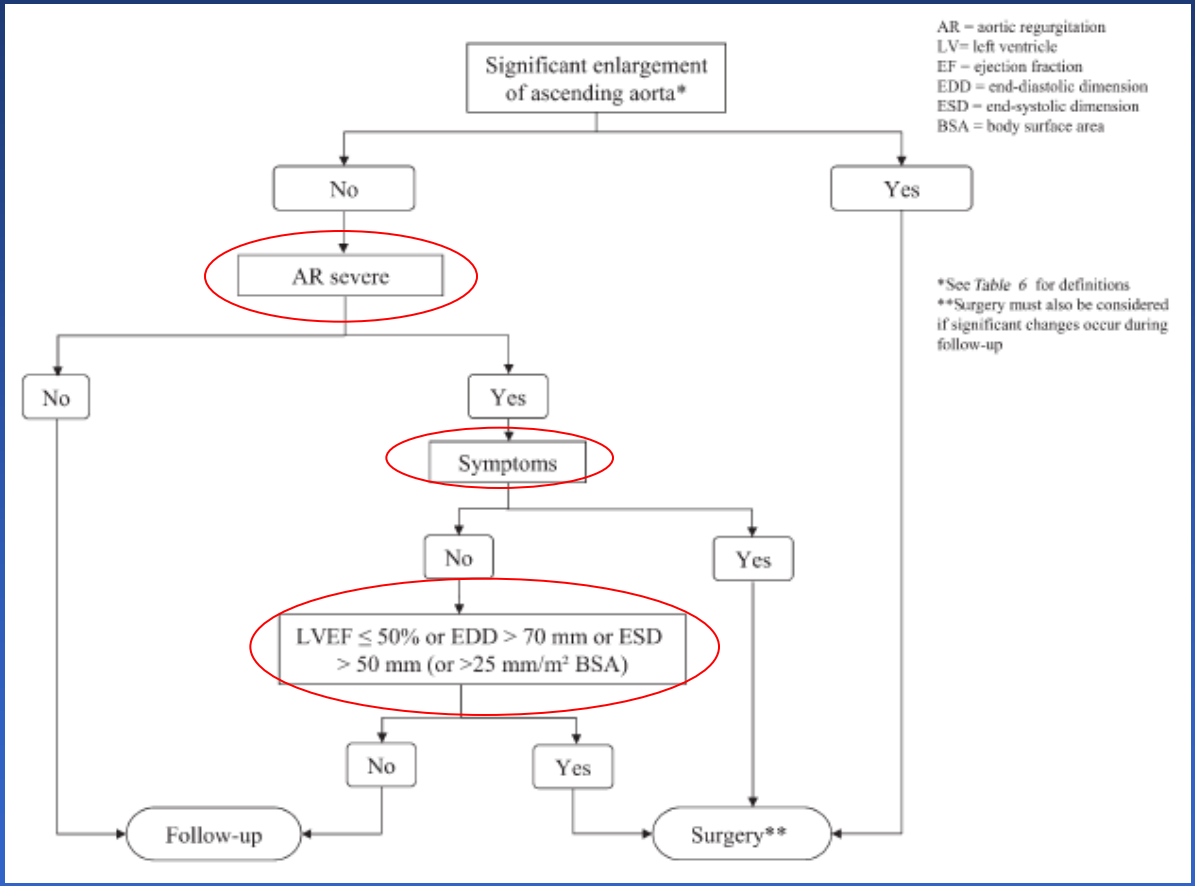


Dujardin KS et al. Circulation 1999

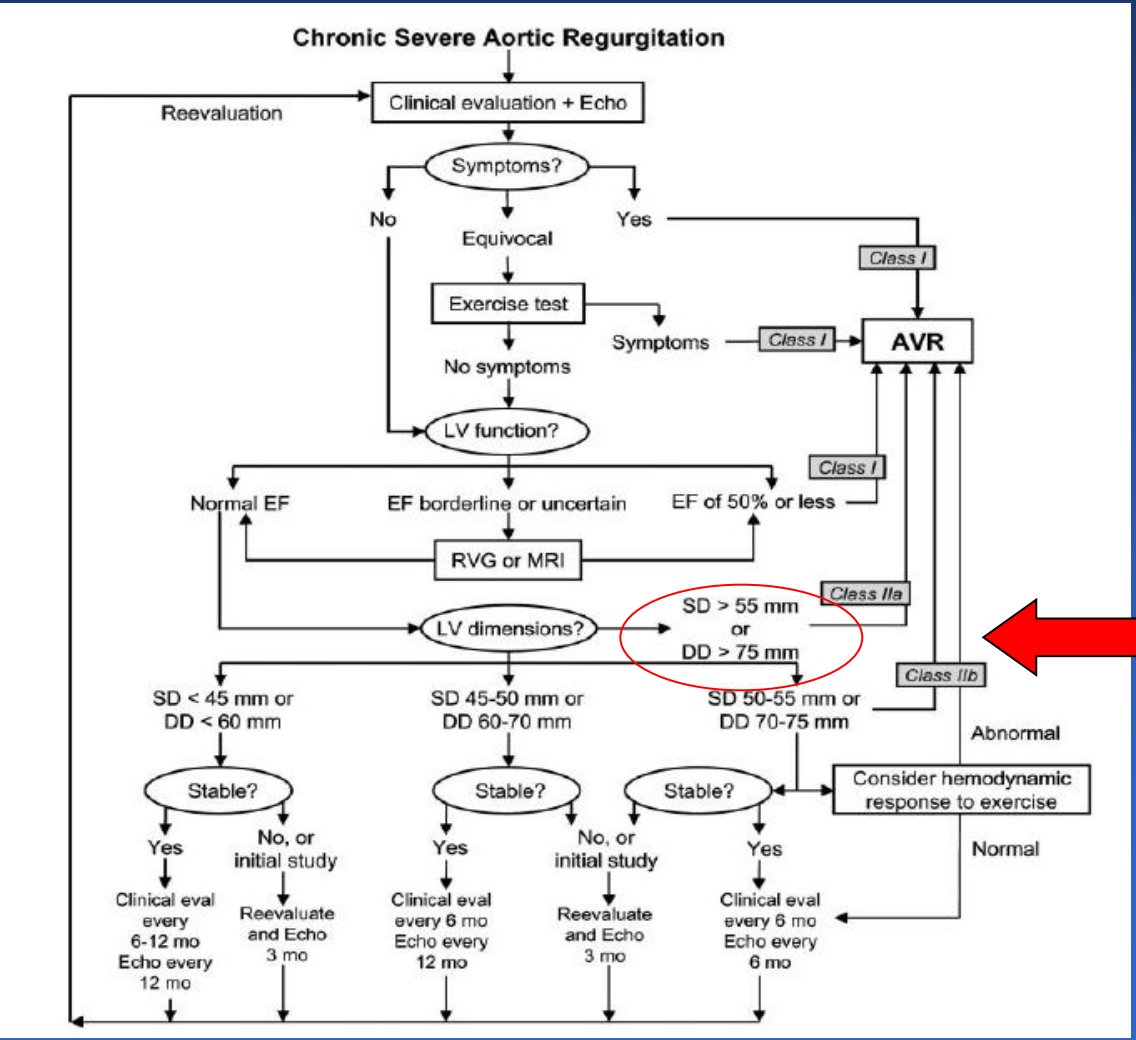
Treatment of chronic severe AR

- The optimal time for surgical intervention is when the combined risk of surgery and a prosthetic valve is lower than the risk of continued watchful waiting
- The operative mortality of isolated AVR is ≈ 4% (and higher for concomitant aortic root replacement or CABG)

ESC Guidelines (2007)



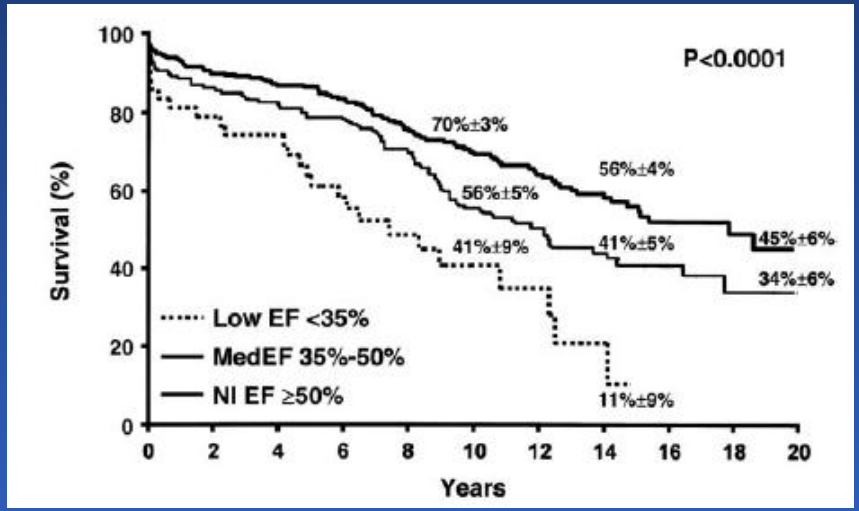
ACC/AHA Guidelines 2006



Difference from ESC Guidelines

Benefit from operation in patients with dilated LV or low EF

- Operative mortality increases as EF falls (14% when EF<35%)
- Improvement of symptoms and LV function in all groups
- It is almost never “too late” to operate in chronic severe AR
- Surgical results have improved with time



Chaliki HP et al. Circulation 2002

Medical treatment of chronic severe AR

- Limited value

- Mainly for:
- control of hypertension
- control of heart failure symptoms when surgery is contraindicated or until the operation
- Marfan patients with aortic dilatation (b-blockers)

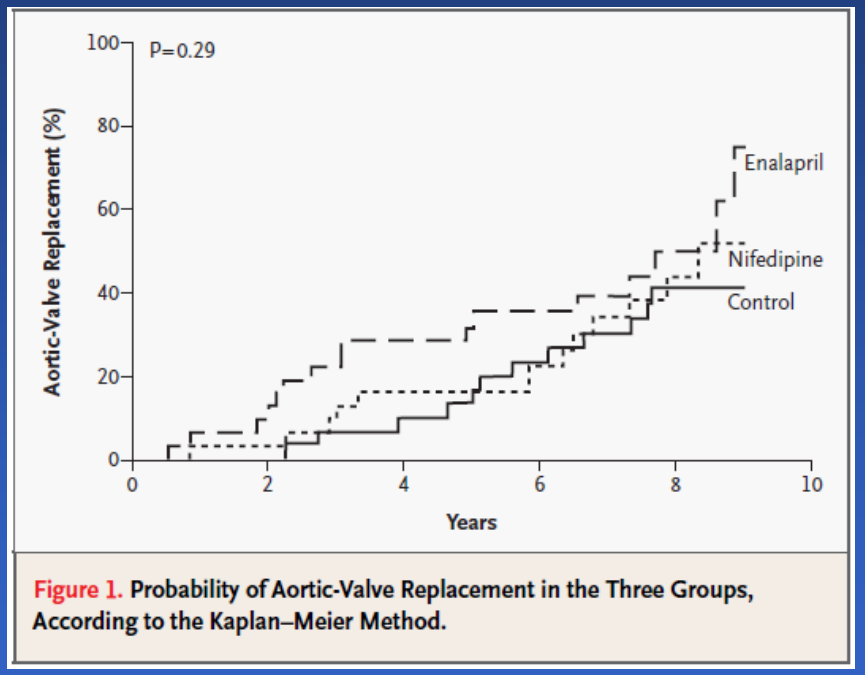
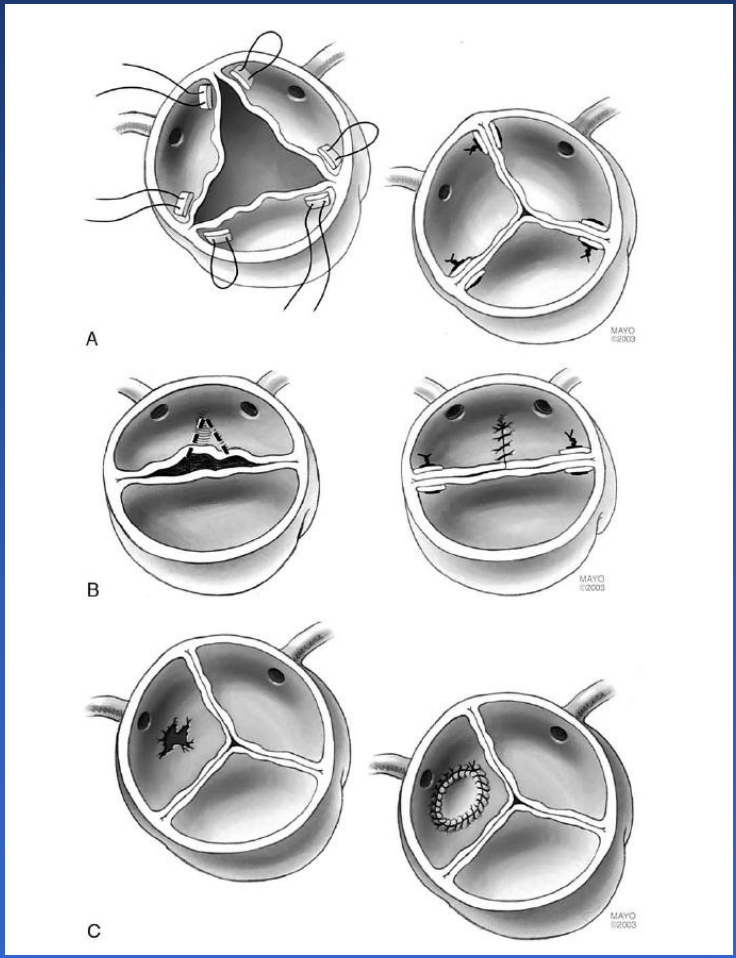


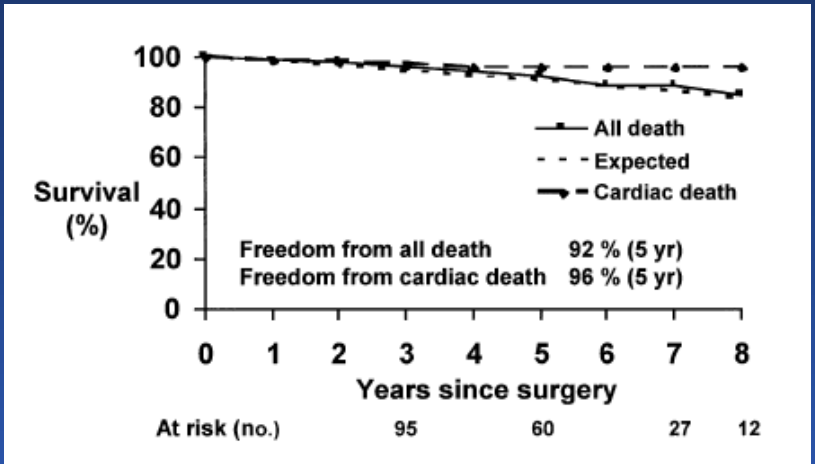
Figure 1. Probability of Aortic-Valve Replacement in the Three Groups, According to the Kaplan–Meier Method.

Evangelista A et al. NEJM 2005

The role of vasodilators in the asymptomatic patients without hypertension in order to delay surgery is unproved

AV Repair?





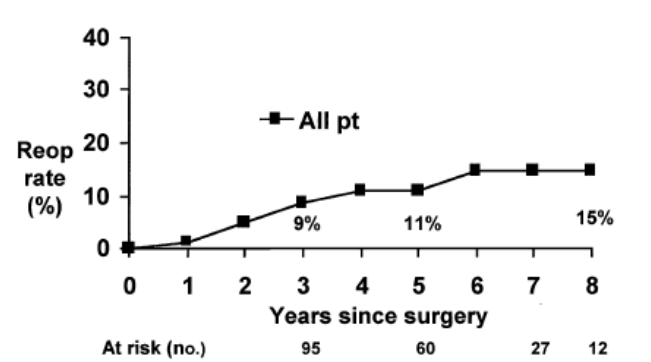


Figure 3. Cumulative risk of aortic valve reoperation.

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