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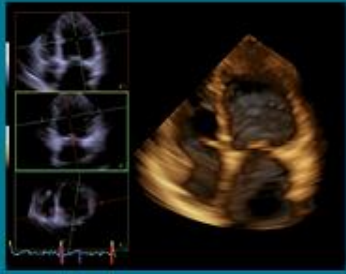
October 24-25, 2014



Aortic regurgitation. Physiopathology

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Hospital Vall d'Hebron. Barcelona
Eurovalve 2014





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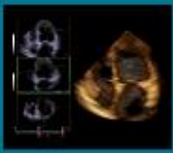
Faculty disclosure

Pilar Tornos

I have **no financial relationships** to disclose.



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Acute AR

Etiology:

Infective endocarditis, aortic dissection, trauma,
post balloon valvuloplasty, post surgical commisurotomy,
idiopathic fenestration or cusp rupture

Pathophysiology:

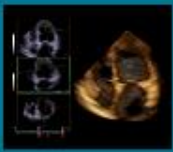
Sudden large regurgitant volume is imposed on a normal size LV



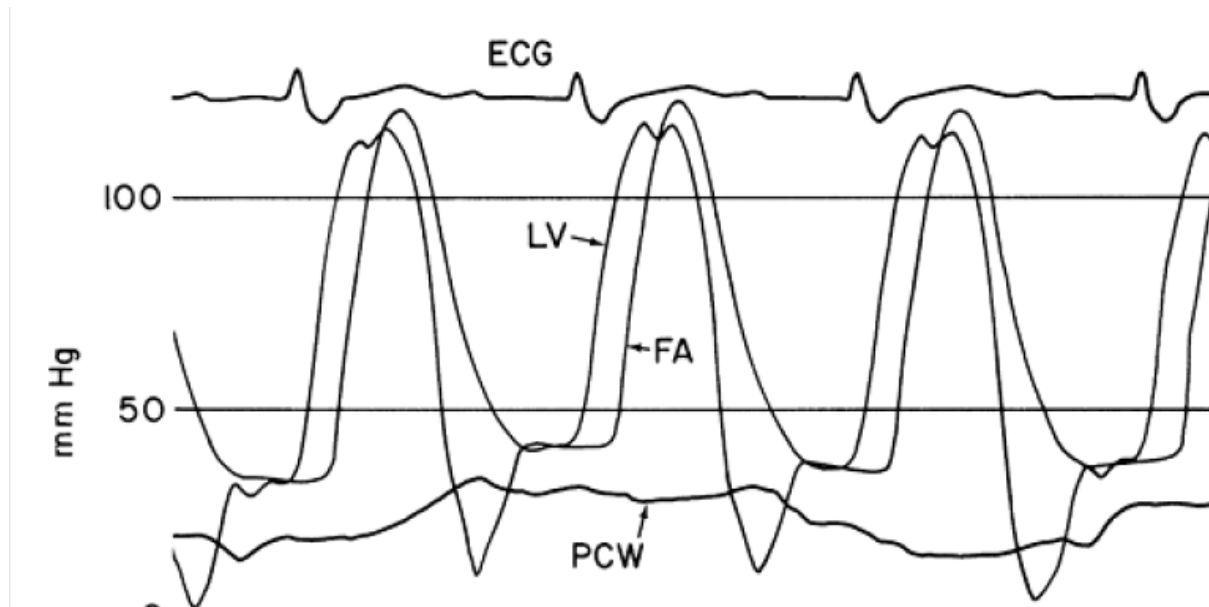
Rapid increase in LVEDP and LAP
Equalization of aortic and LV pressures in diastole



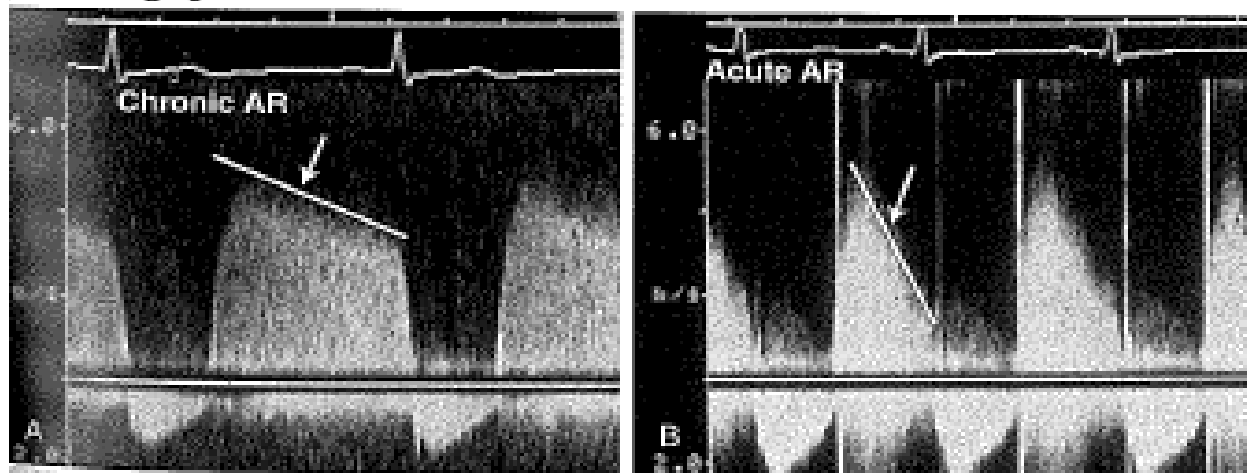
Pulmonary edema, cardiogenic shock
Diminished myocardial perfusion pressure
In the subendocardium



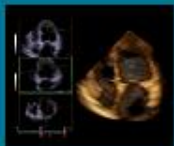
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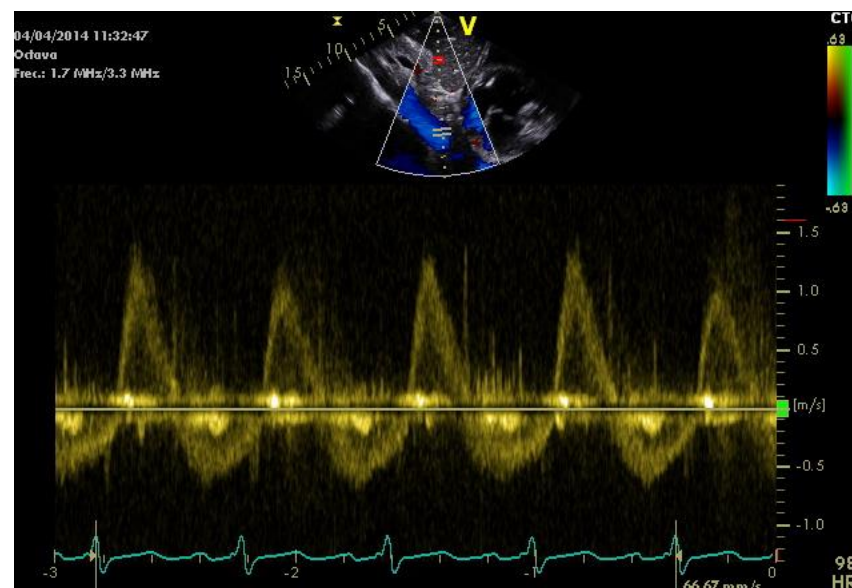
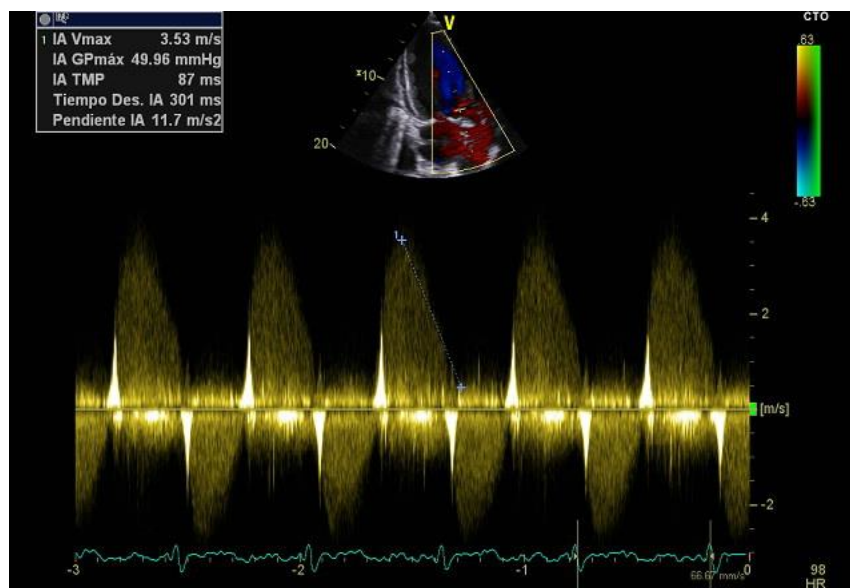
Taken from Grossman , 7 ed

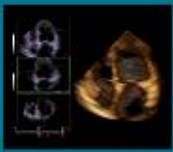


Taken from Otto, 2 edi



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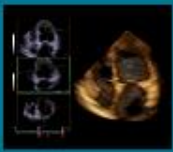




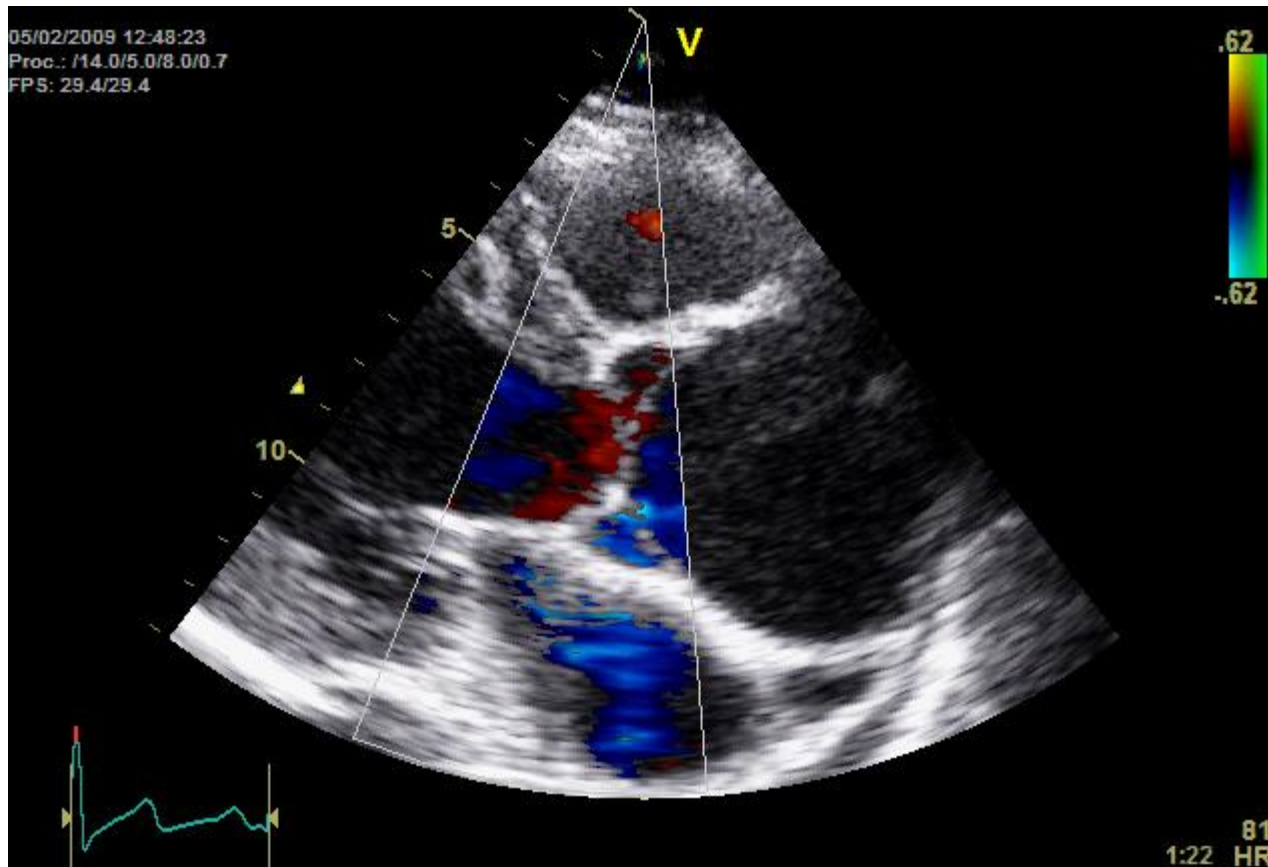
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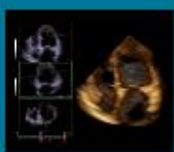


	Chronic AR compensated	Chronic AR decompensated	Acute AR
Physiology LV Volume Ejection Fraction LV EDP	Increased (ESD<55) Normal (>55%) Normal	Increased (ESD>55) Normal or decreased Normal	Normal Normal or decreased Increased
Physical exam Diastolic murmur Pulse pressure Peripheral signs	Holodiastolic, Wide Present	Holodiastolic Wide Present	Early diastole Normal Absent
Clinical Presentation	Asymptomatic	Gradual onset of symptoms,	Sudden onset, pulmonary edema



Acute AR in aortic dissection





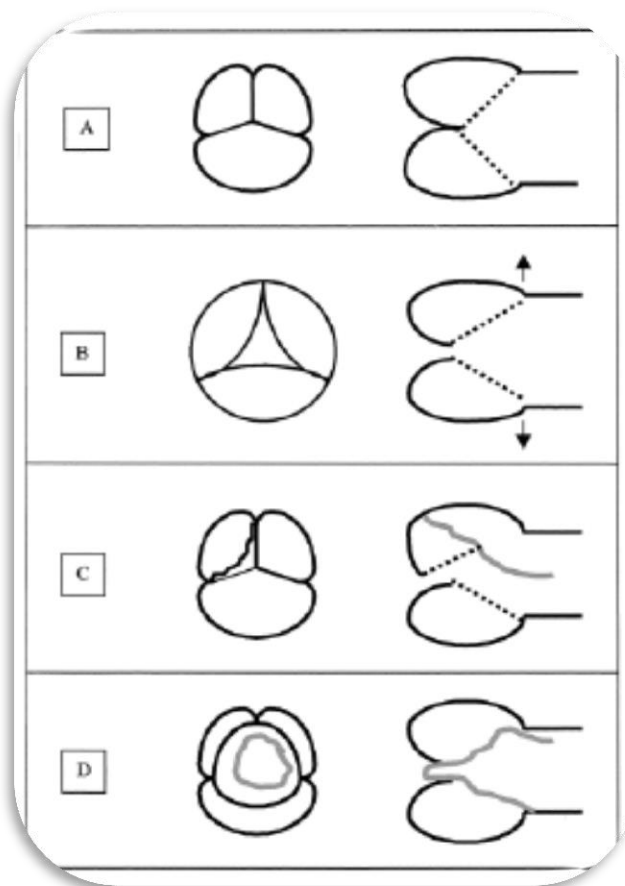
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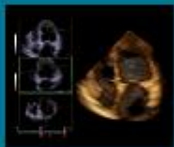
Acute AR in Aortic Dissection.



	Degree of Aortic Regurgitation	
	None/Trace/Mild (n = 27)	Moderate/Severe (n = 22)
Incomplete leaflet closure	0	12
Aortic leaflet prolapse	2	8
Intimal flap prolapse	2	3
Bicuspid aortic valve	3	3
Leaflet thickening	17	5



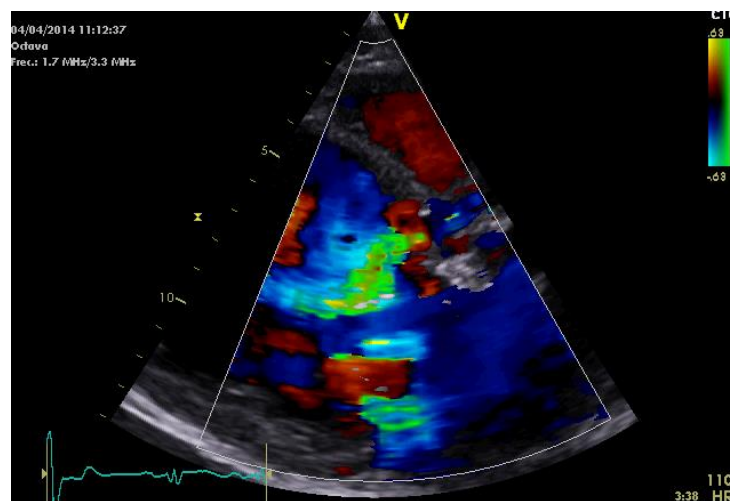
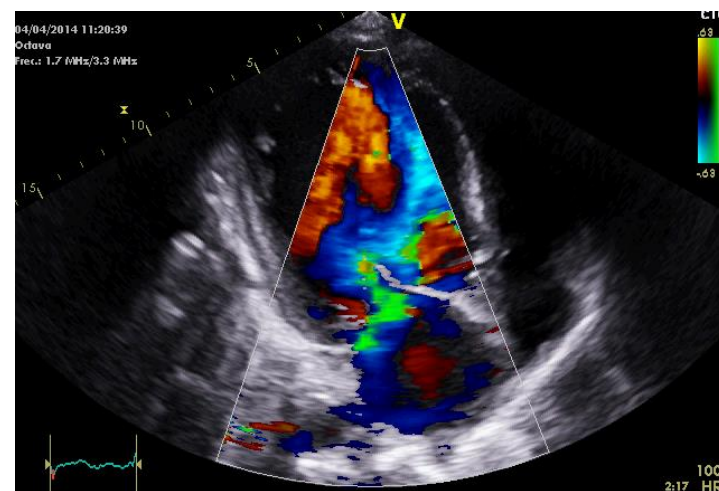
Movsowitz HD, et al. JACC 2000;36:884

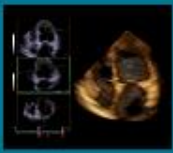


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Acute AR in IE





Chronic AR. Pathophysiology

VOLUME-PRESSURE OVERLOAD

Increased stroke volume

Increased afterload

Ventricular dilatation

Increase in wall thickness

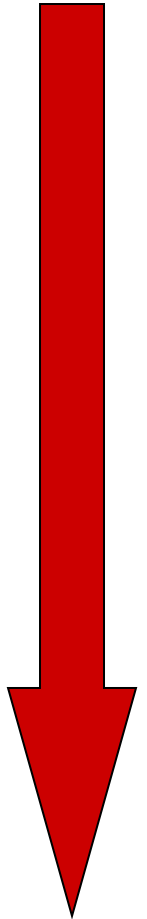
Wall stress maintained

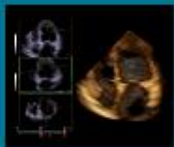
Increase in LV dimension and systolic pressure

Wall thickening fails to keep pace

Increase in wall stress

Excessive afterload: depression of contractility



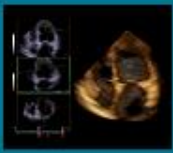


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Stage	Definition	Description
A	At risk	Patients with risk factors for the development of VHD
B	Progressive	Patients with progressive VHD (mild-to-moderate severity and asymptomatic)
C	Asymptomatic severe	Asymptomatic patients who have reached the criteria for severe VHD C1: Asymptomatic patients with severe VHD in whom the left or right ventricle remains compensated C2: Asymptomatic patients who have severe VHD, with decompensation of the left or right ventricle
D	Symptomatic severe	Patients who have developed symptoms as a result of VHD

Stages of Progression of VHD. ACC/ AHA Guidelines 2014



CHRONIC AR

Stage A: At risk of AR

Stage B: Mild- moderate AR

Stage C1: Compensated severe AR (balanced
preload/hypertrophy/afterload)
FE>50%, LVESD < 50 mm

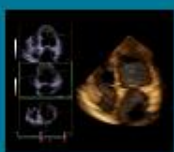
Stage C2: Decompensated severe AR
(progressive LV enlargement, decline EF)
FE<50% or LVESD >50 mm or >25 mm/m²

Stage D: Irreversible LV dysfunction



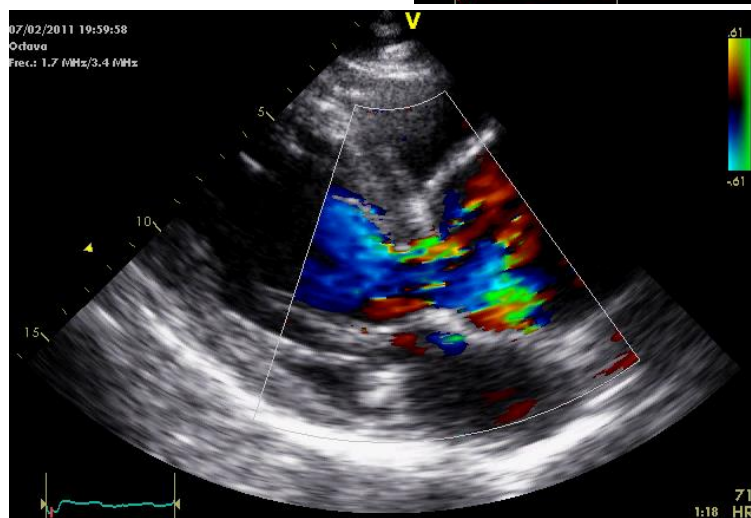
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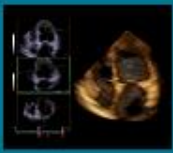
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Chronic AR. Stages A and B





CHRONIC AR

Stage A: At risk of AR

Stage B: Mild- moderate AR

Stage C1: Compensated severe AR (balanced
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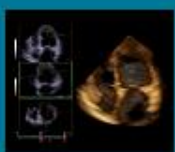
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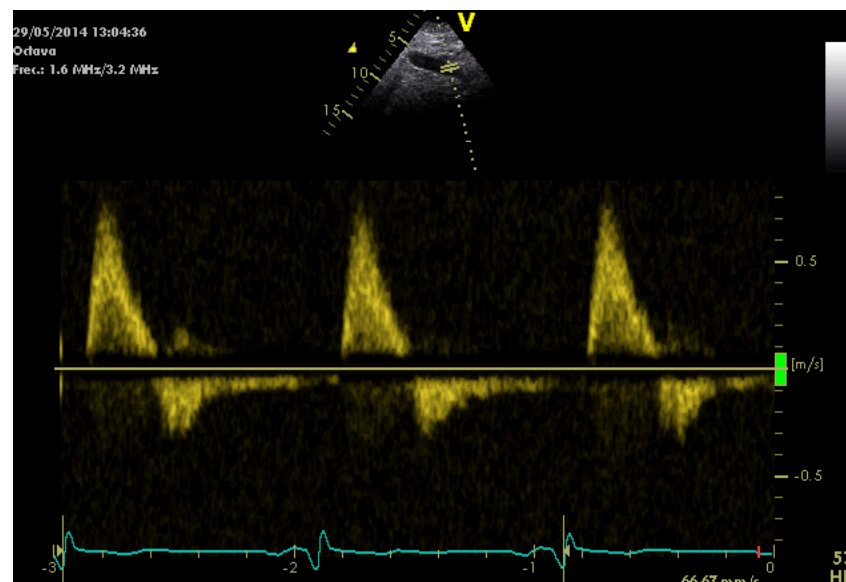
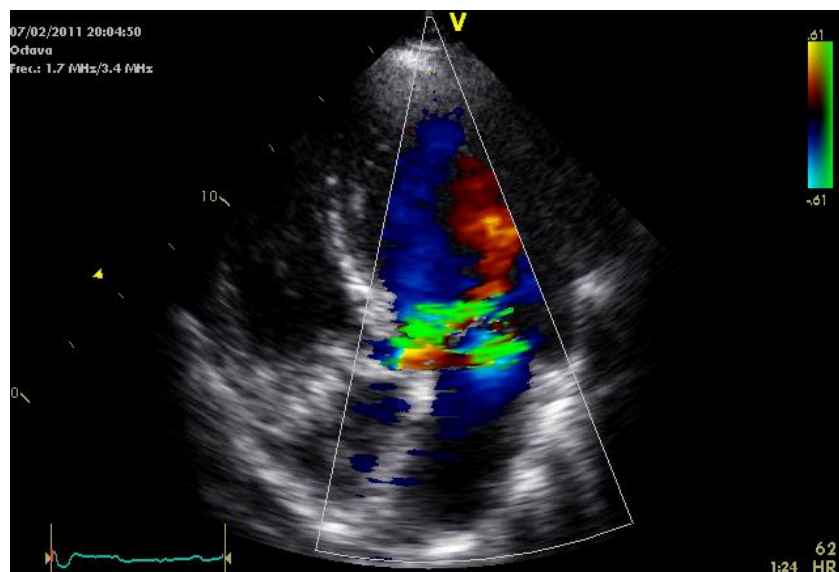


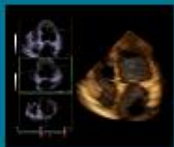
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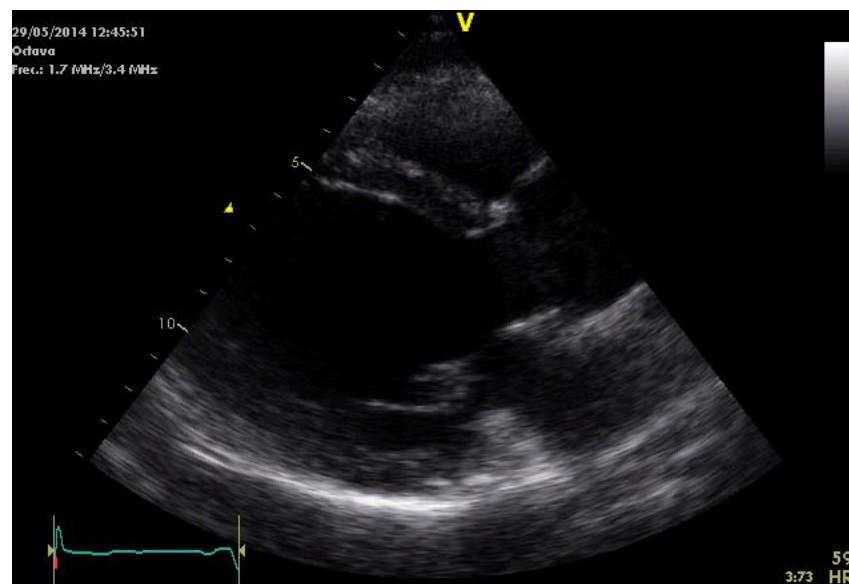
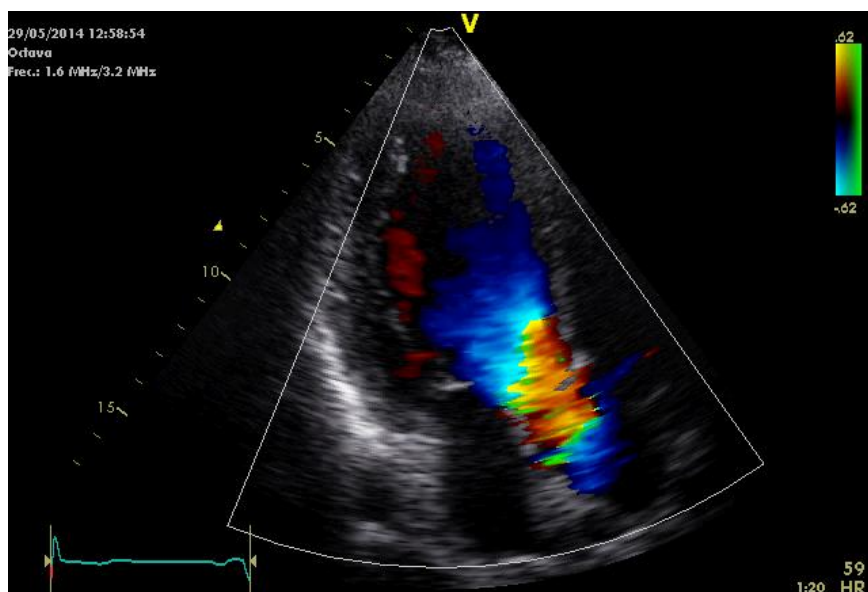


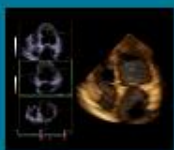
Chronic AR. Stage C1





Chronic AR. Stage C1

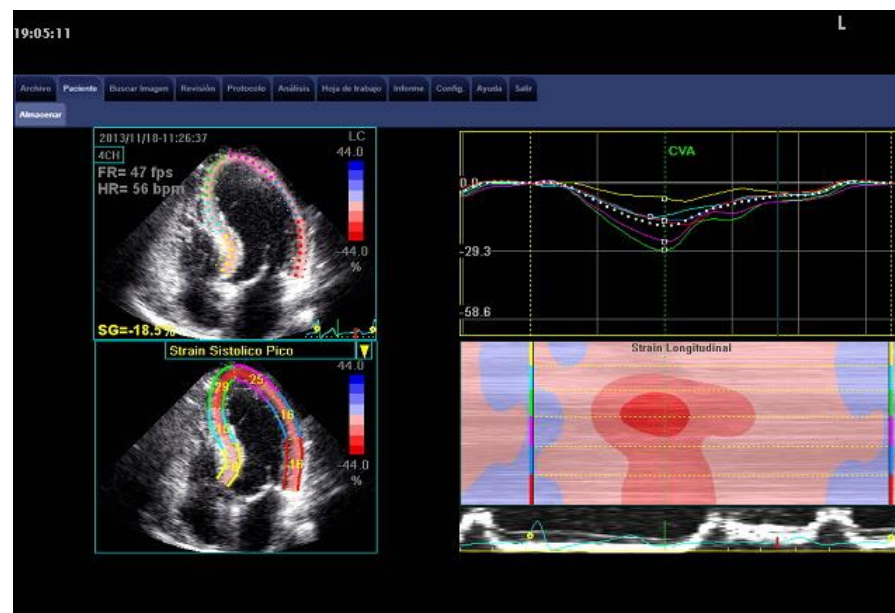
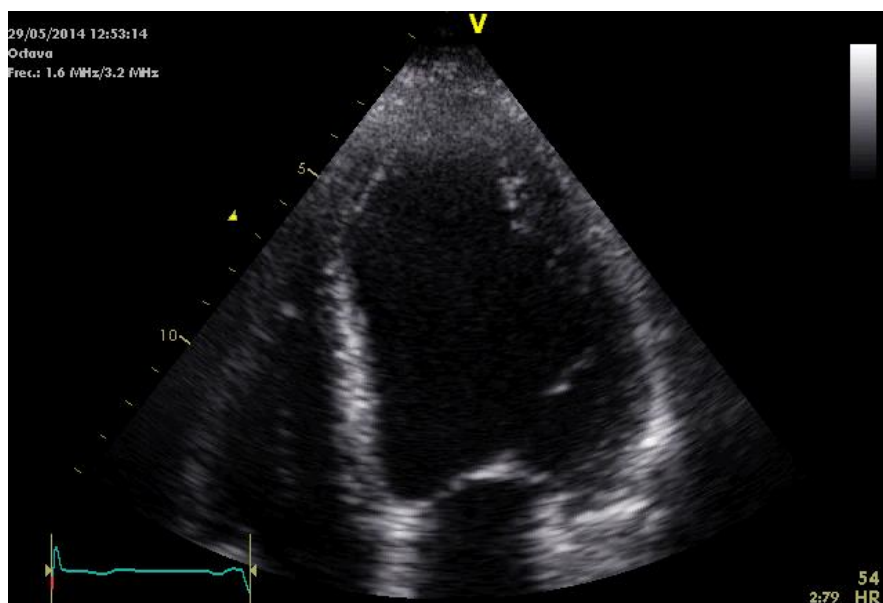


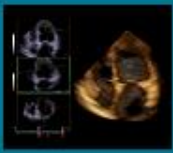


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Chronic AR Stage C1





CHRONIC AR

Stage A: At risk of AR

Stage B: Mild- moderate AR

Stage C1: Compensated severe AR (balanced preload/hypertrophy/afterload)
FE>50%, LVESD < 50 mm

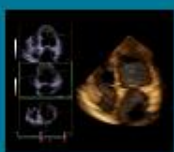
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FE<50% or LVESD >50 mm or >25 mm/m²

Stage D: Irreversible LV dysfunction



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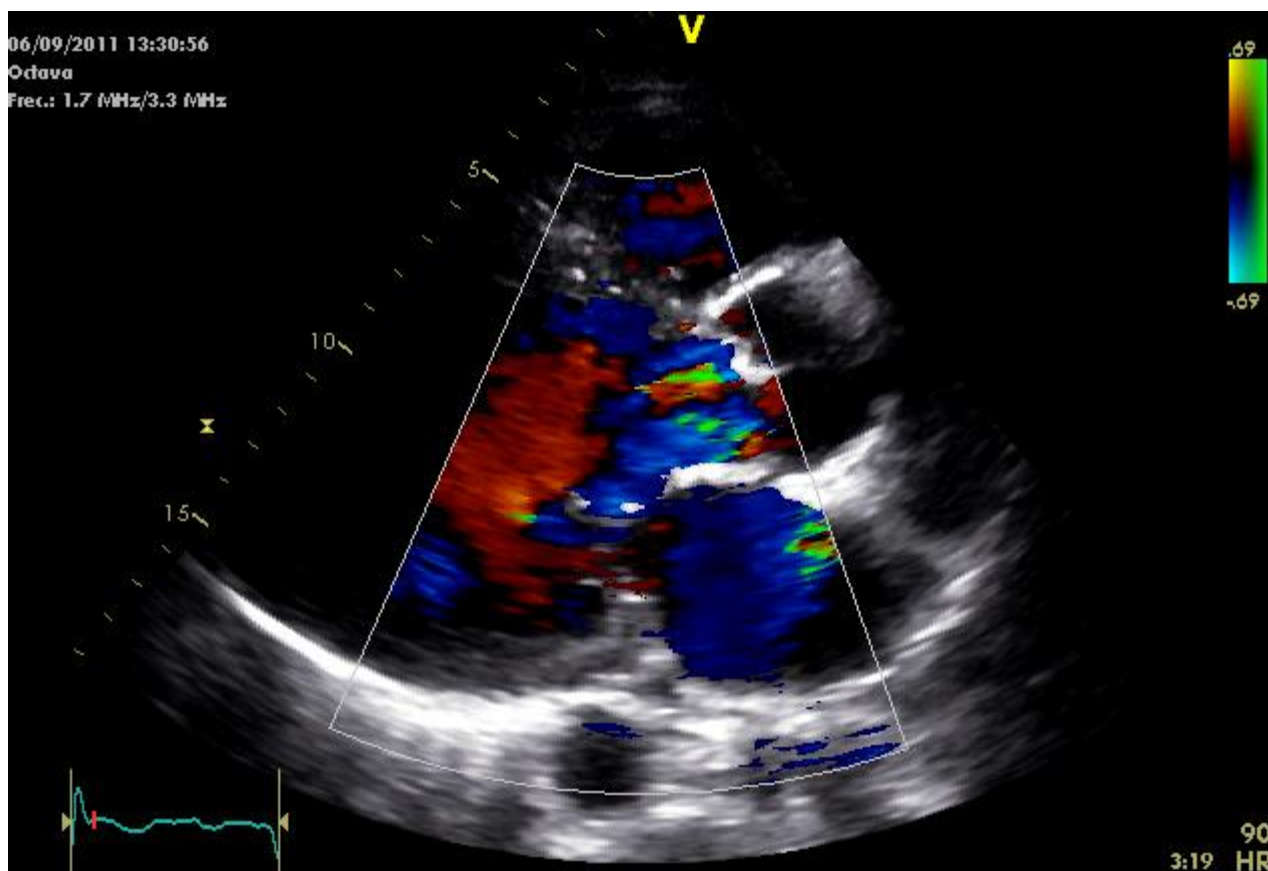
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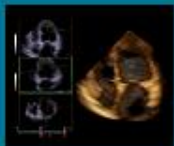


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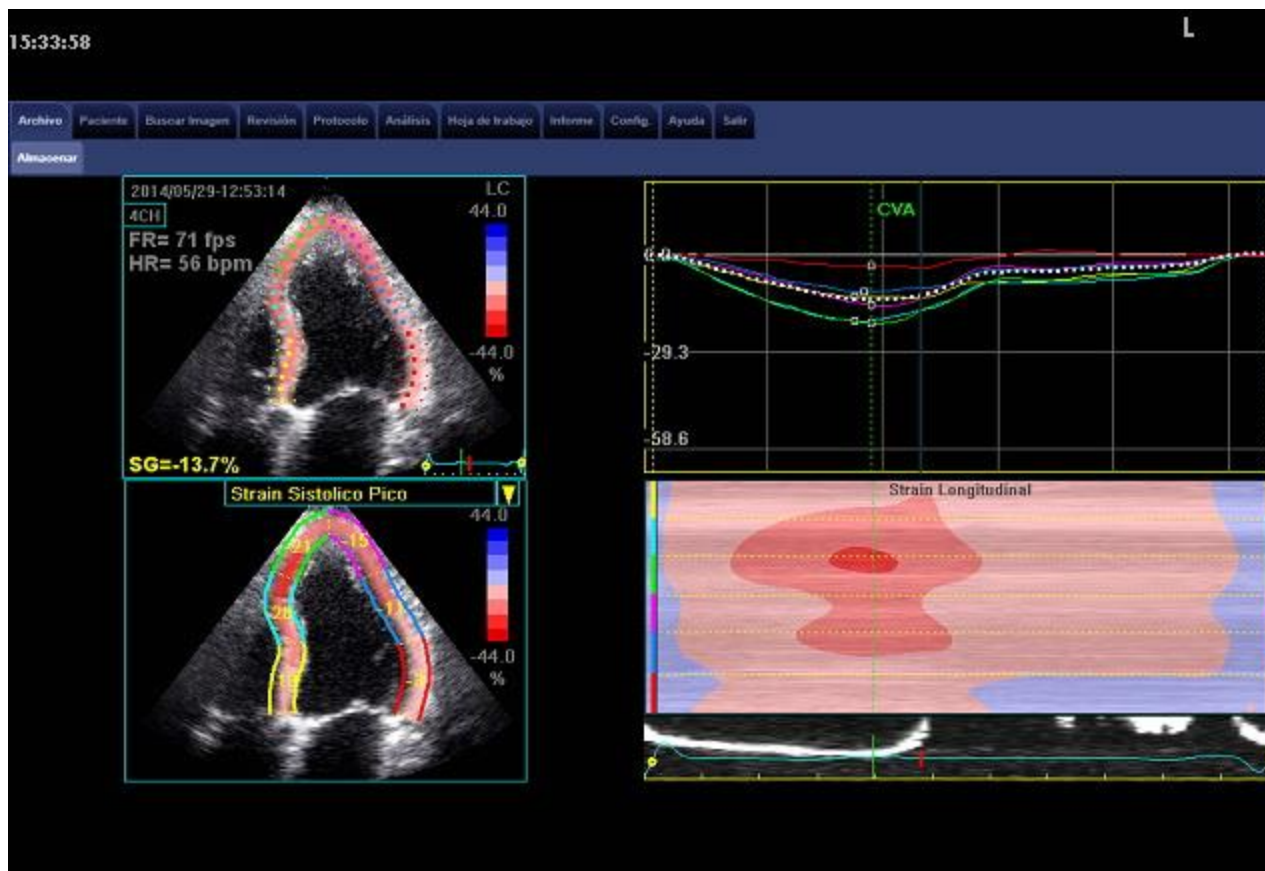


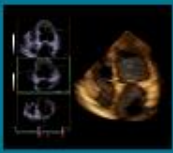
Chronic AR. Stage C2





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CHRONIC AR

Stage A: At risk of AR

Stage B: Mild- moderate AR

Stage C1: Compensated severe AR (balanced
preload/hypertrophy/afterload)
FE>50%, LVESD < 50 mm

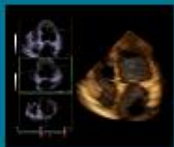
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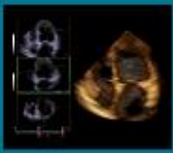
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Chronic AR. Stage D





Final comments

- The pathophysiology of both acute and chronic AR is well established
- Clinical and Echo-Doppler evaluation are needed for diagnosis and follow up
- Knowledge of pathophysiology is crucial for the correct management of patients with AR