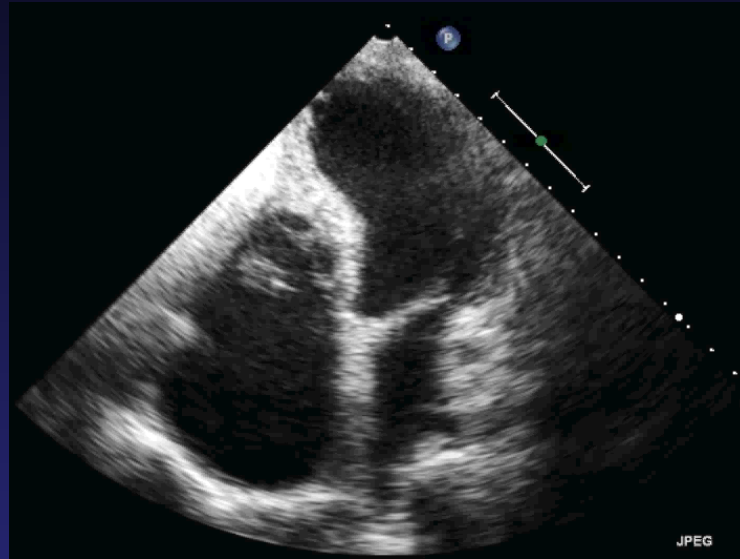


Mystery Case



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Vice President, American Society of Echocardiography

Professor of Medicine

Northwestern University Feinberg School of Medicine

Medical Director, Echocardiography Laboratory

Northwestern Memorial Hospital

- *No Disclosures*

History

- 74 year old female who presents with progressive LE edema
- She denies shortness of breath and chest pain

History

- She has a long standing history of hypereosinophilia treated with steroids
- PMH
 - Asthma
 - Nasal polyps
 - ASA allergy
 - sinusitis
- She has seen a cardiologist only intermittently

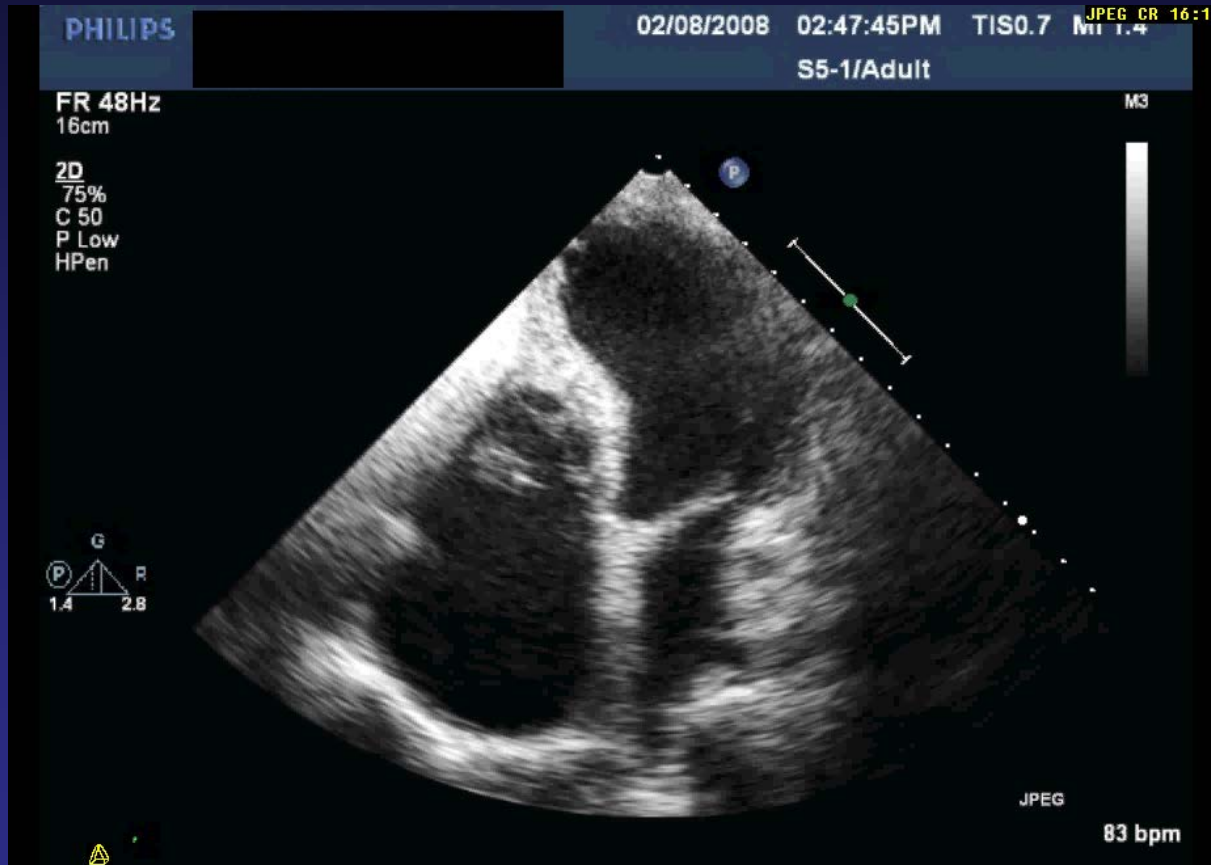
PLAX 2008



PSAX 2008



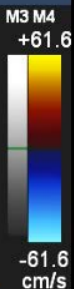
4 Chamber View 2008



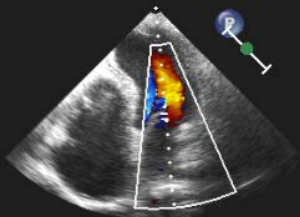
FR 19Hz
15cm

9:38:14

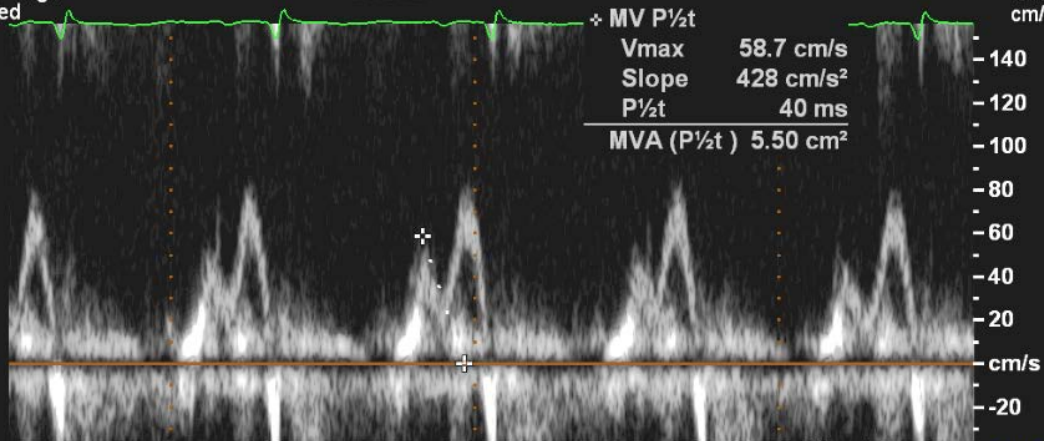
M3 M4
PW
50%
1.6MHz
WF 125Hz
SV4.0mm
7.7cm



2D
75%
C 50
P Low
HPen
CF
66%
2.5MHz
WF High
Med



MV P_{1/2}t
Vmax 58.7 cm/s
Slope 428 cm/s²
P_{1/2}t 40 ms
MVA (P_{1/2}t) 5.50 cm²

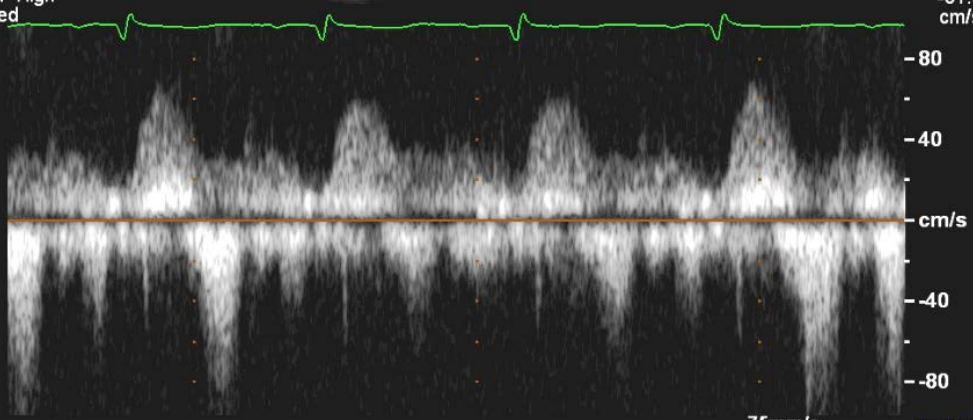
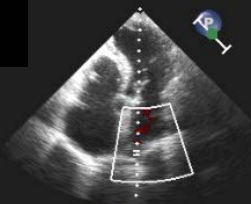
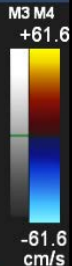


75mm/s 86L 1 of 1

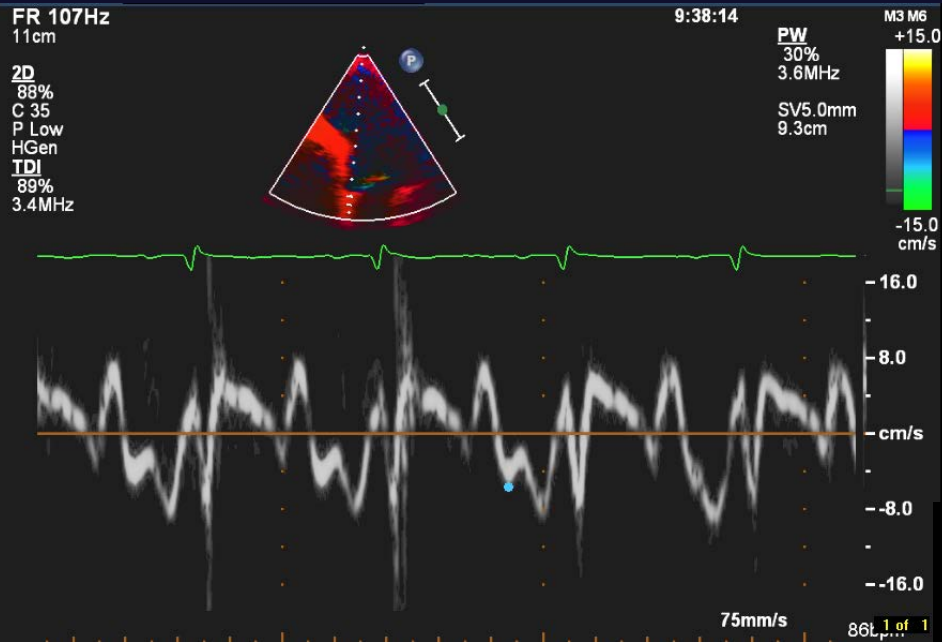
C 50
P Low
HPen
CF
66%
2.5MHz
WF High
Med

9:38:14

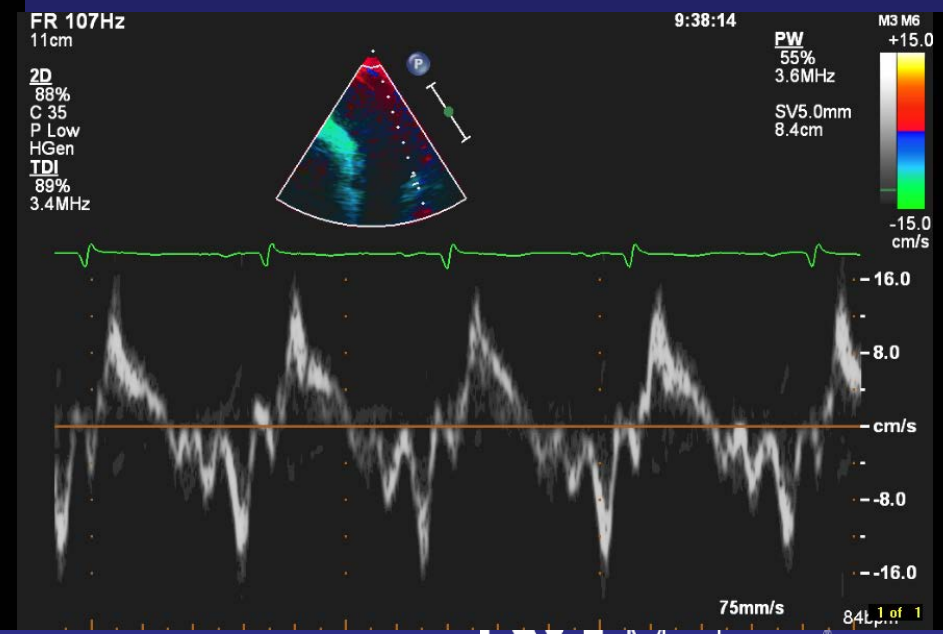
M3 M4
PW
50%
1.6MHz
WF 125Hz
SV4.0mm
13.8cm



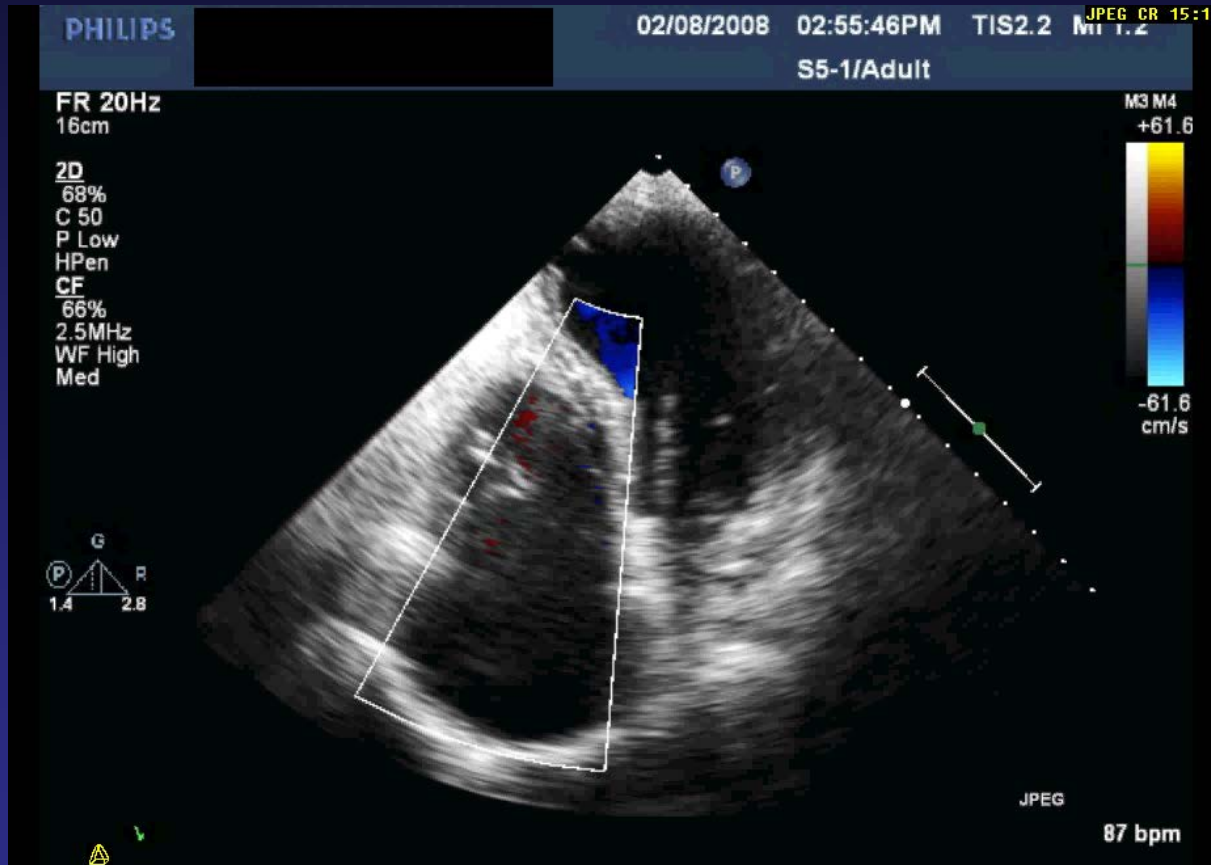
75mm/s 86L 1 of 1



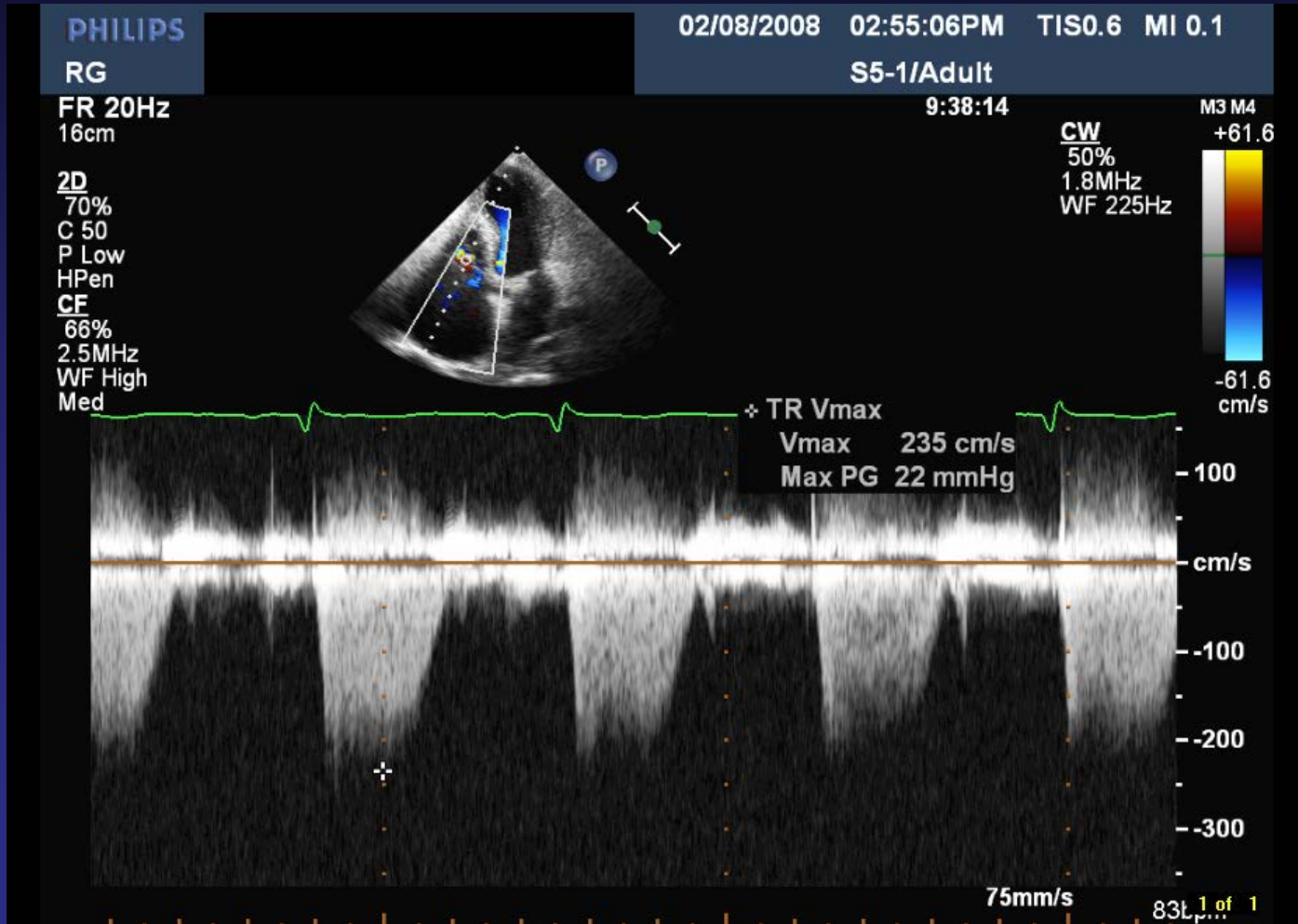
$$E/E' = 7.3$$



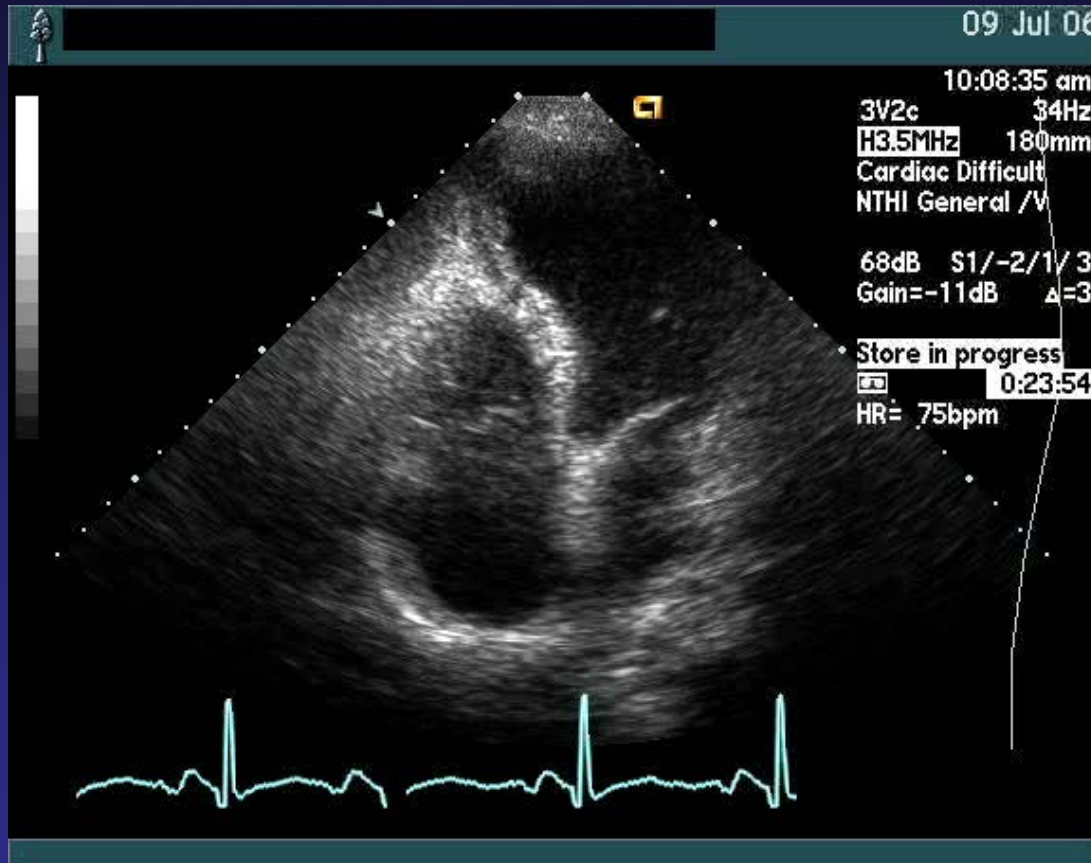
4 Chamber View 2008



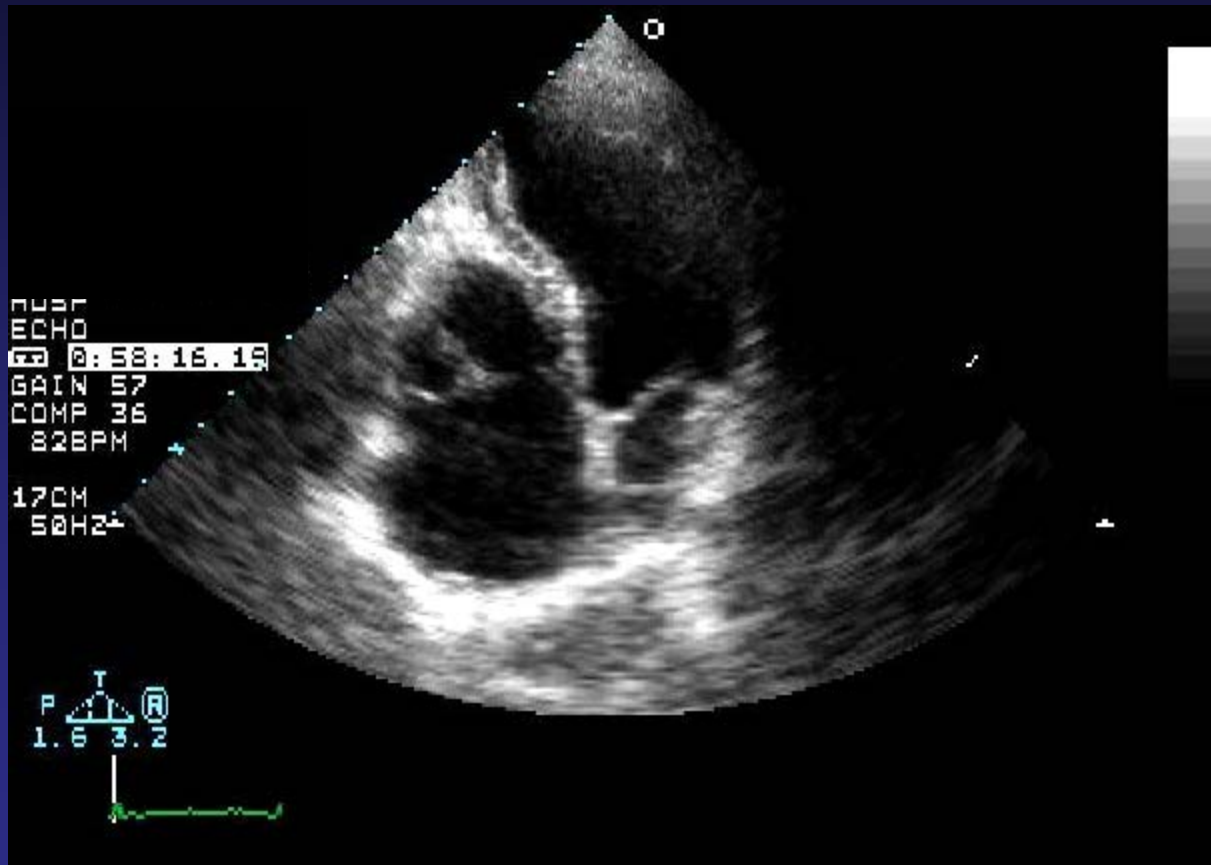
RV Systolic Pressure 2008



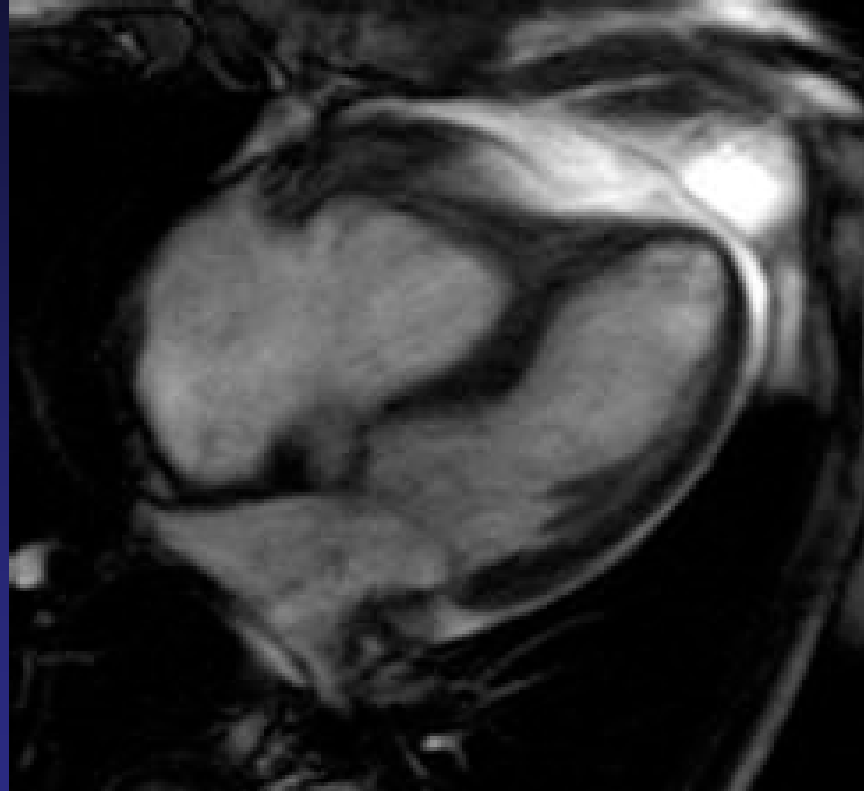
4 Chamber View 2006



4 Chamber View 2002



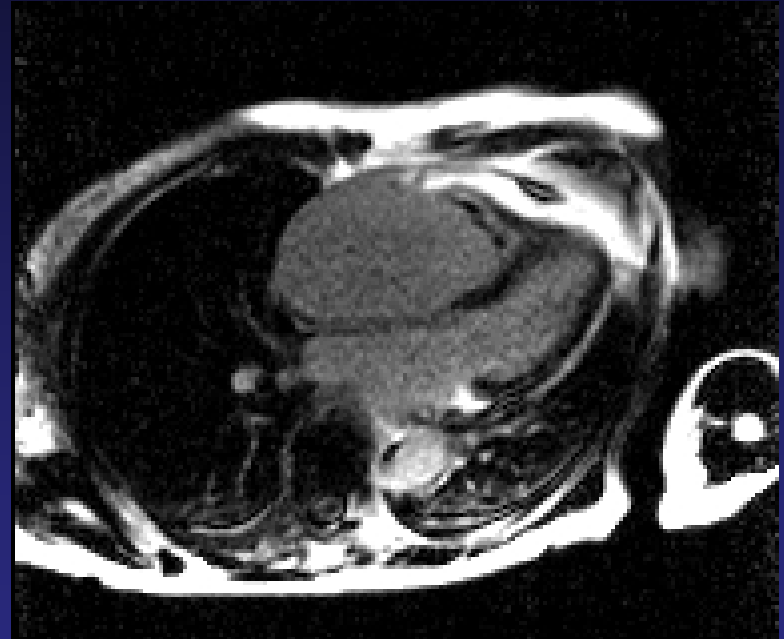
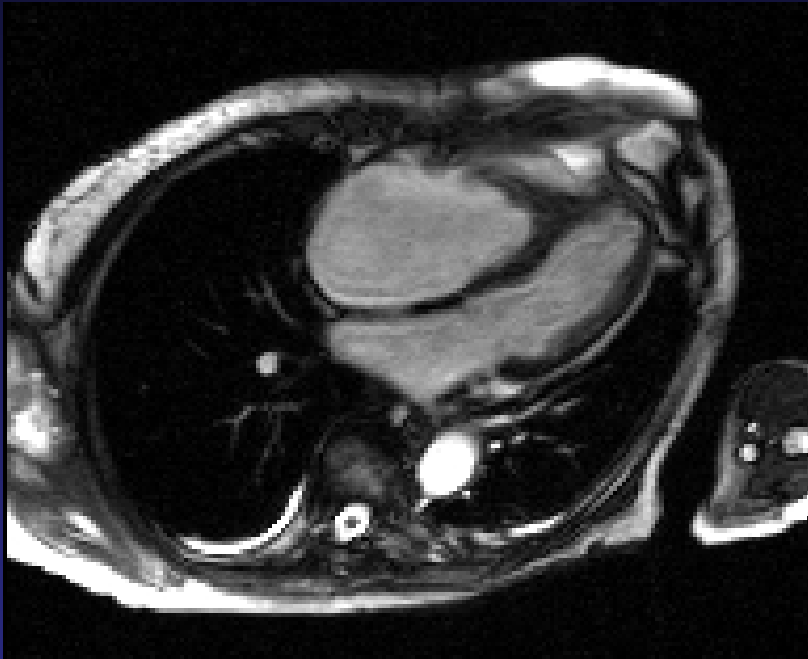
Cine MRI 2008



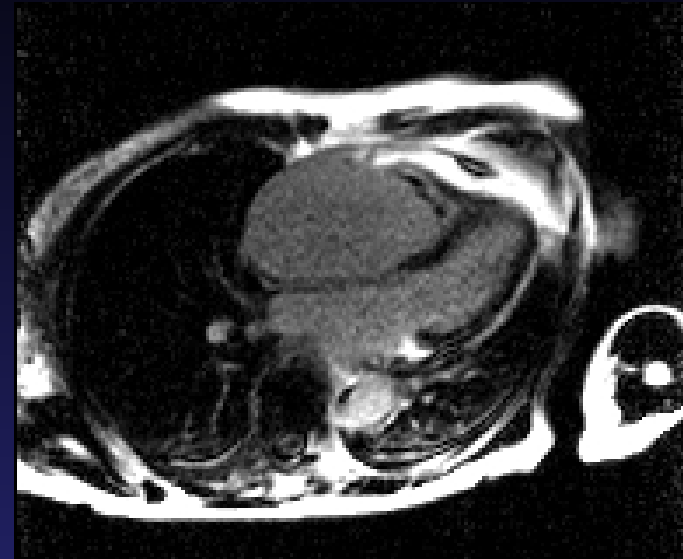
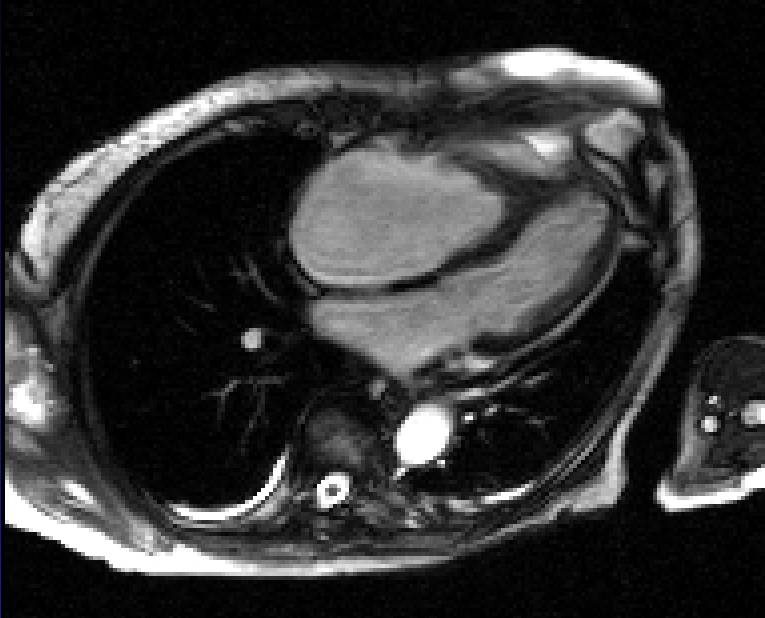
What is the Mass in the RV Apex?

- A. Tumor
- B. Thrombus
- C. Fibrosis
- D. B and C

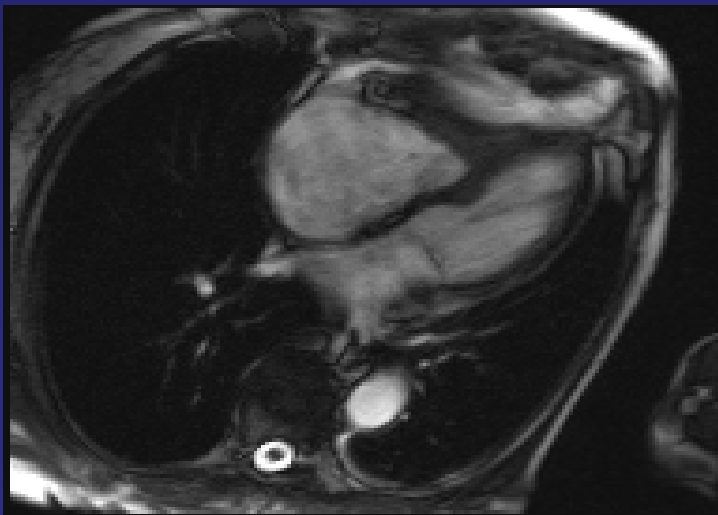
MR Cardiac July 2006



MR Cardiac July 2006



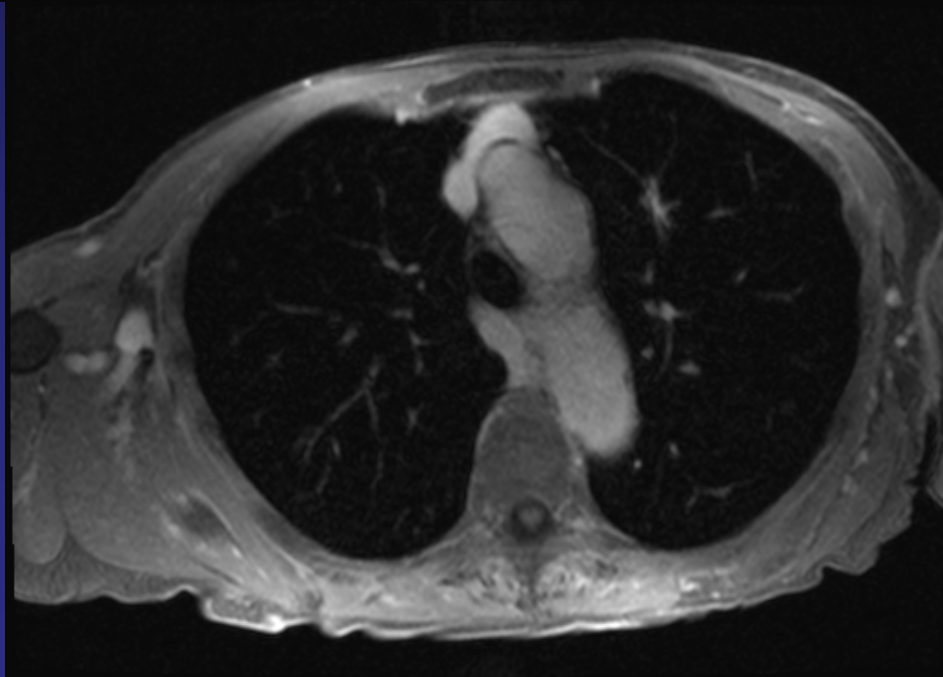
MR Cardiac March 2008







2008

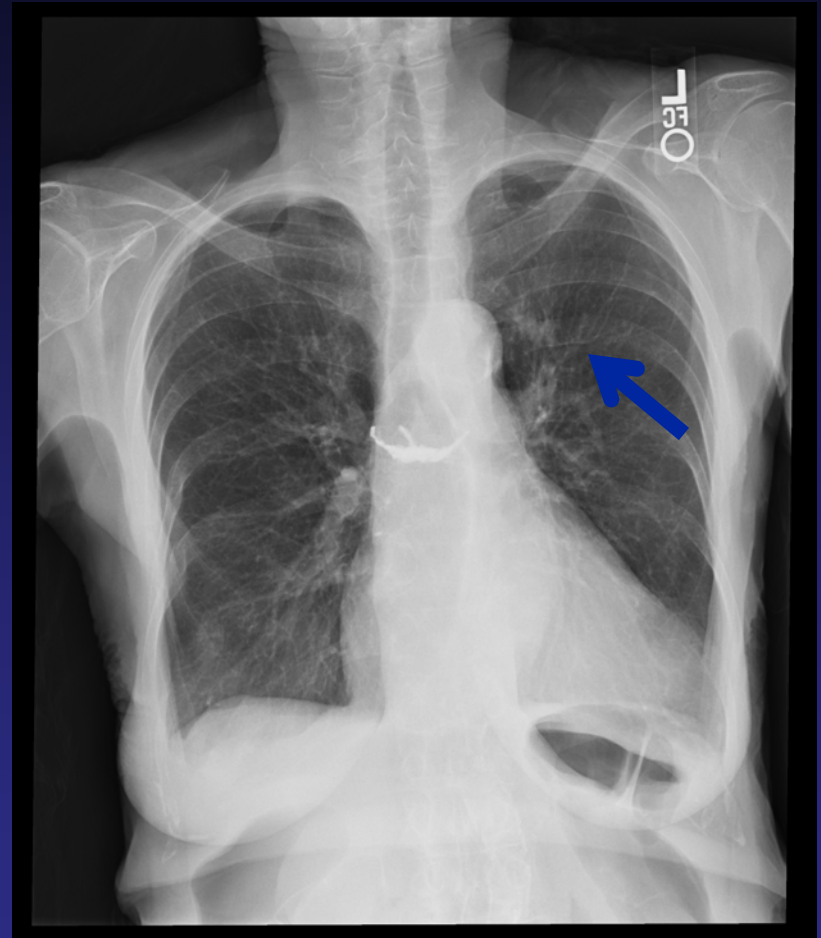
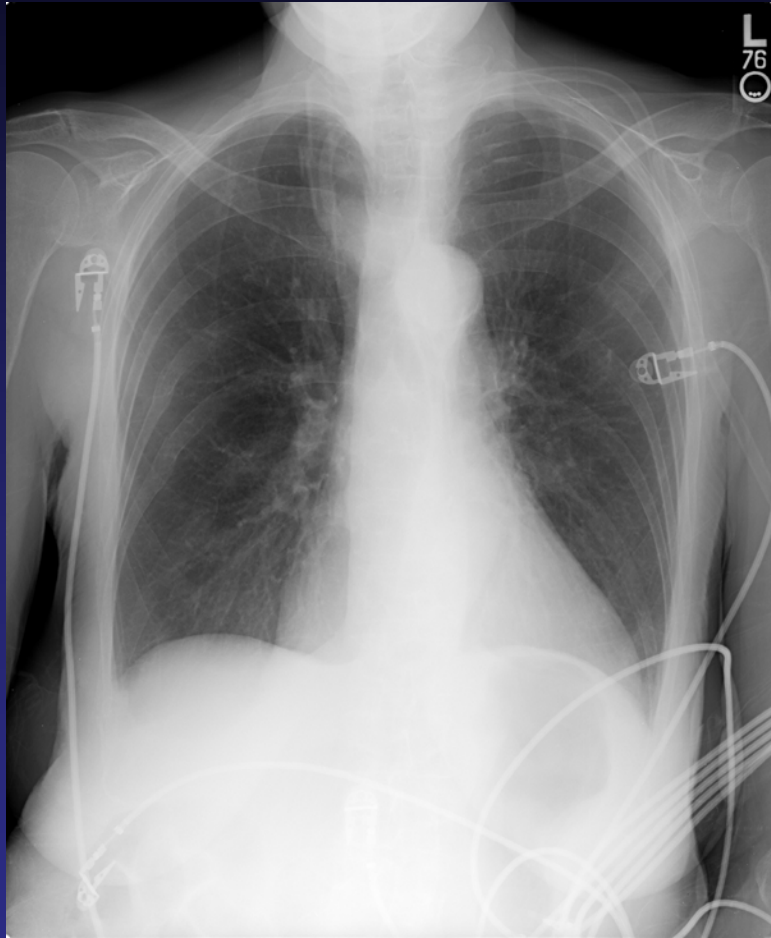


2006

Chest X-Ray

2006

12/2007



Loffler's Syndrome

- First described in 1932 by Wilhelm Loffler
- Eosinophilic pneumonia caused by the parasites *Ascaris lumbricoides*, *Strongyloides stercoralis* and the hookworms *Ancylostoma duodenale* and *Necator americanus*
- Loffler's endocarditis: Cardiac damage due to idiopathic or parastic hypereosiniphilia

Table 2. Eosinophilic Cardiomyopathies.

Feature	Hypersensitivity Myocarditis	Acute Necrotizing Eosinophilic Myocarditis	Löffler's Endocarditis	Endomyocardial Fibrosis
Demographics	Any	Occasional history of allergic disorders	Male, temperate climates	Tropical climates
Precipitant	Medication	Medication, viral infection, or any cause of eosinophilia	Any cause of eosinophilia, including drug hypersensitivity reaction, parasitic infection, allergic disorder, vasculitides (e.g., Churg–Strauss syndrome, polyarteritis nodosa), hypereosinophilic syndrome, eosinophilic leukemia, other malignancies (e.g., Hodgkin's disease)	Any cause of eosinophilia, including drug hypersensitivity reaction, parasitic infection, allergic disorder, vasculitides (e.g., Churg–Strauss syndrome, polyarteritis nodosa), hypereosinophilic syndrome, eosinophilic leukemia, other malignancies (e.g., Hodgkin's disease)
Tempo	Acute and transient	Acute and severe	Subacute	Gradual
Clinical presentation	Usually fever and rash Mild heart failure Arrhythmias	Sometimes fever and rash Chest pain Fulminant heart failure	Usually fever and rash Right-sided more often than left-sided heart failure Embolic events	Right-sided more often than left-sided heart failure
Peripheral eosinophilia	Sometimes	Usually	Almost always	Rarely
Biomarkers of necrosis	Modestly elevated	Elevated	Normal	Normal
ECG	Nonspecific ST-segment and T-wave abnormalities	Sinus tachycardia ST-segment elevation Low voltage	Nonspecific ST-segment and T-wave abnormalities	Nonspecific ST-segment and T-wave abnormalities
Echocardiography	Mild systolic dysfunction Occasional pericardial effusion	Severe systolic dysfunction Increased wall thickness Pericardial effusion	Restrictive cardiomyopathy Mural thrombi Thrombotic vegetations Mitral and tricuspid regurgitation	Restrictive cardiomyopathy Organized mural thrombi Mitral and tricuspid regurgitation
Pathology	Eosinophilic infiltrate Mild necrosis	Eosinophilic infiltrate Severe necrosis Occasional giant cells	Mild eosinophilic infiltrate Endomyocardial thickening and fibrosis Overlying thrombosis	Occasionally eosinophilic infiltrate Endomyocardial thickening and fibrosis Occasional organized thrombosis
Prognosis	Self-limited	Poor	Irreversible	Irreversible

Sabatine MS et al. N Eng J Med 2007;357:2167-

Hypereosinophilic Syndromes

- Disorders marked by sustained overproduction of eosinophils with damage to multiple organs due to eosinophilic infiltration and mediator release
- Dx: Clinical features, eos $>$ 1500/mm³, no other causes of high eos, end organ damage
- Common organs involved: heart, lungs, skin, neuro, eyes
- Most common in men 20-50 yrs old

Stages of Cardiac Involvement

- Early necrotic phase
 - Eosinophilic infiltration causing microabscesses
- Intermediate thrombotic-necrotic phase
 - Thrombus formation on surfaces of denuded endomyocardium
- Fibrotic phase
 - Endomyocardial fibrosis

What is the Mass in the RV Apex?

- A. Tumor
- B. Thrombus
- C. Fibrosis
- D. B and C

Cardiac Involvement

- Necrosis/thrombosis/fibrosis most common in RV and LV apex
- Fibrosis can lead to restrictive cardiomyopathy
- Fibrosis can entrap chordal apparatus and lead to valve dysfunction
- Patients with cardiac symptoms refractory to medical therapy may require valve replacement, endomyocardectomy or thrombectomy
- Mortality is often due to cardiac decompensation



Thank You