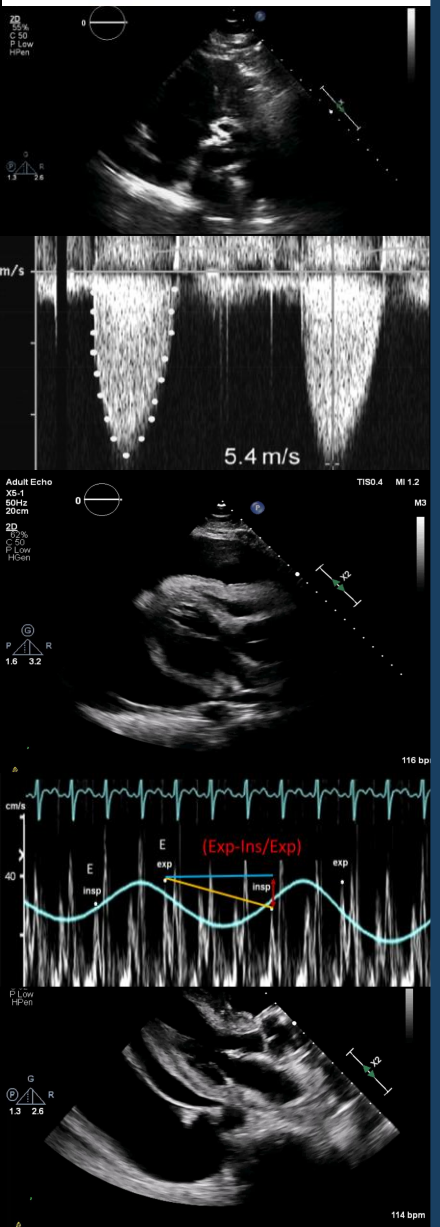
Iowa CHAPTER



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CARVER COLLEGE OF MEDICINE
University of Iowa Health Care

Cardiology Consults: Symptomatic Severe Aortic Stenosis & Cardiac Tamponade

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Disclosures

- None

Consult #1

Consult #1 - Objectives

In relation to severe aortic stenosis (AS) at the conclusion of this activity, participants will be able to:

1. Be able to diagnose severe AS based on physical exam

2. Understand natural history and pathophysiology of AS

3. Learn the basics of echocardiographic assessment and how to interpret Echo report/measurements

Consult #1

A 78 yo M with no significant past medical history, now presents with syncope.

Physical Exam:

BP 131/63 | Pulse 55 | Ht 5' 4” | Wt 70.3 kg (154 lb 15.7 oz) | SpO2 96% | BMI 26.59 kg/m²

- Lound 3/6 systolic ejection murmur at the apex, R clavicle and 2nd ISC radiating to the neck (LR+/95% CI: 9-154; *Etchells E et al. JAMA 1997;277(7):564-571*)

- Usually peaks in early- or mid-systole; if absent or soft S2 = severe AS
- Apical murmur due to *Galliverdin phenomenon*
- Carotid pulse is *parvus et tardus*, delayed carotid upstroke

Consult #1 - Transthoracic echo images obtained, pending report

Technical Comments
Echo image quality: good to marginal.

LV EF?

MMode/2D Measurements & Calculations

IVSd: 0.99 cm
LVIDd: 4.8 cm
LVIDs: 3.2 cm
LVPWd: 0.98 cm
IVS/LVPW: 1.0
Ao root diam: 2.3 cm
LA dimension: 5.0 cm

asc Aorta Diam: 3.3 cm
LA/Ao: 2.1
LVOT diam: 2.0 cm
LVOT area: 3.0 cm^2
EDV(MOD-sp4): 112.0 ml
ESV(MOD-sp4): 41.5 ml
EDV(MOD-sp2): 76.6 ml
ESV(MOD-sp2): 29.7 ml

SV(MOD-sp4): 70.5 ml
LA Area (A2C): 30.4 cm^2
LA Area (A4C): 31.0 cm^2
LAV (AL-bp): 118.8 cm^3

LAV (AL-bp) Indexed: 67.0 ml/m^2

Doppler Measurements & Calculations

MV E max vel: 114.0 cm/sec
MV A max vel: 78.7 cm/sec
MV E/A: 1.4
MV dec time: 0.28 sec
Ao V2 max: 431.6 cm/sec
Ao max PG: 74.5 mmHg
Ao V2 mean: 314.4 cm/sec
Ao mean PG: 43.9 mmHg
Ao V2 VTI: 106.3 cm
AVA(LD): 0.66 cm^2
AVA(VD): 0.65 cm^2
AI dec slope: 81.0 cm/sec^2
AI P1/2t: 1227 msec

LVV1 max: 92.6 cm/sec
LVV1 mean: 64.8 cm/sec
LVV1 VTI: 23.0 cm
TR max vel: 258.4 cm/sec
TR max PG: 26.7 mmHg

Reference Values:

LVEF: 50-70%

SV: 80-120 ml
SVi: >35 ml/m^2

LVOT VTI: 18-22 cm

IVSd: <1.0 cm
LVPWd: <1.0 cm

Ao V2 max: ≤2.5 m/sec

Ao V2 mean gr: <20 mmHg

AVA: 2.0-2.5 cm^2

DI (V1/V2): >0.5

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Grading AS Severity

Table 3 Recommendations for grading of AS severity

	Aortic sclerosis	Mild	Moderate	Severe
Peak velocity (m/s)	≤2.5 m/s	2.6–2.9	3.0–4.0	≥4.0
Mean gradient (mmHg)	–	<20	20–40	≥40
AVA (cm^2)	–	> 1.5	1.0–1.5	<1.0
Indexed AVA (cm^2/m^2)	–	>0.85	0.60–0.85	<0.6
Velocity ratio (or DI)	–	> 0.50	0.25–0.50	<0.25

Normal LVEF *

- Echo Doppler can NOT overestimate the gradient (unless Hb <8), so AV gr ≥40 mmHg = SEVERE AS

J Am Soc Echocardiogr 2017;30:372-92

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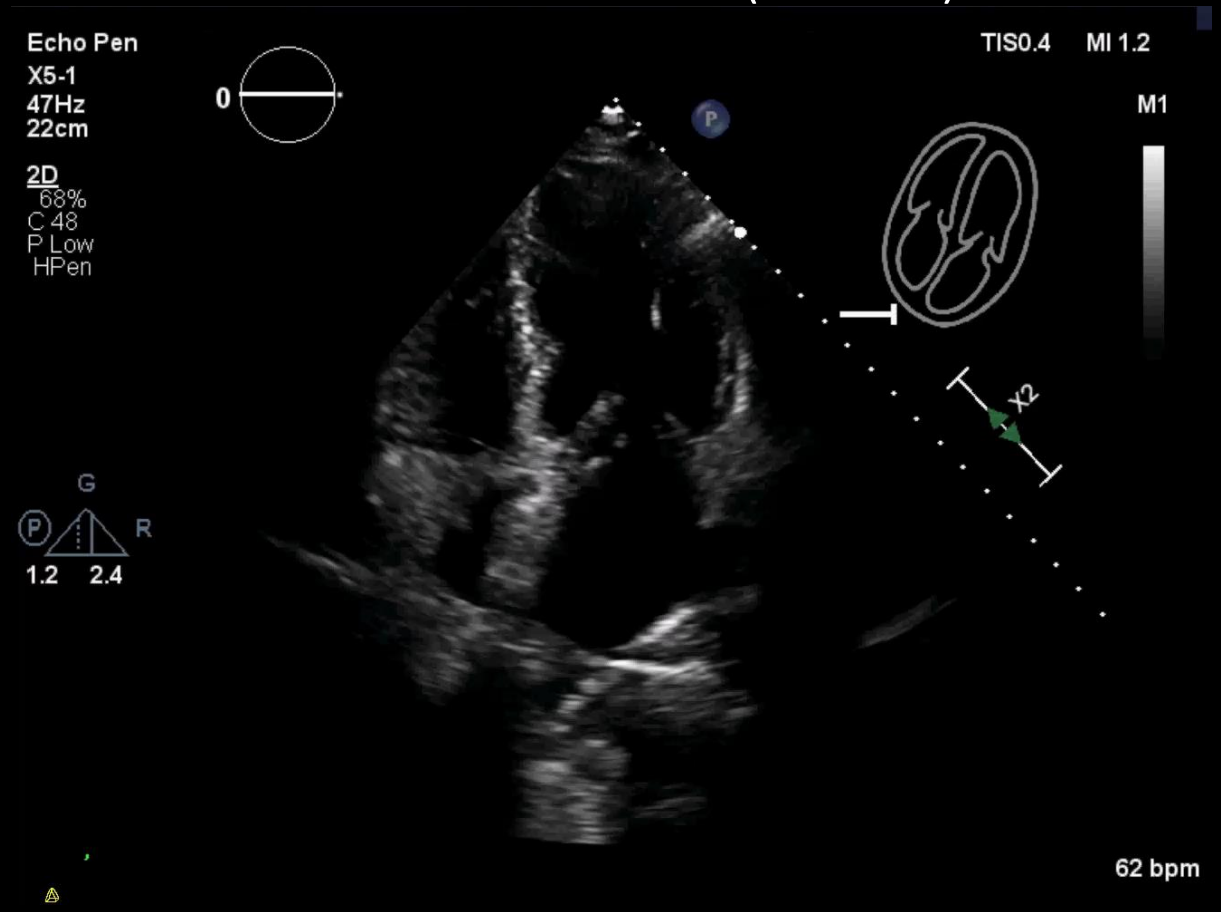
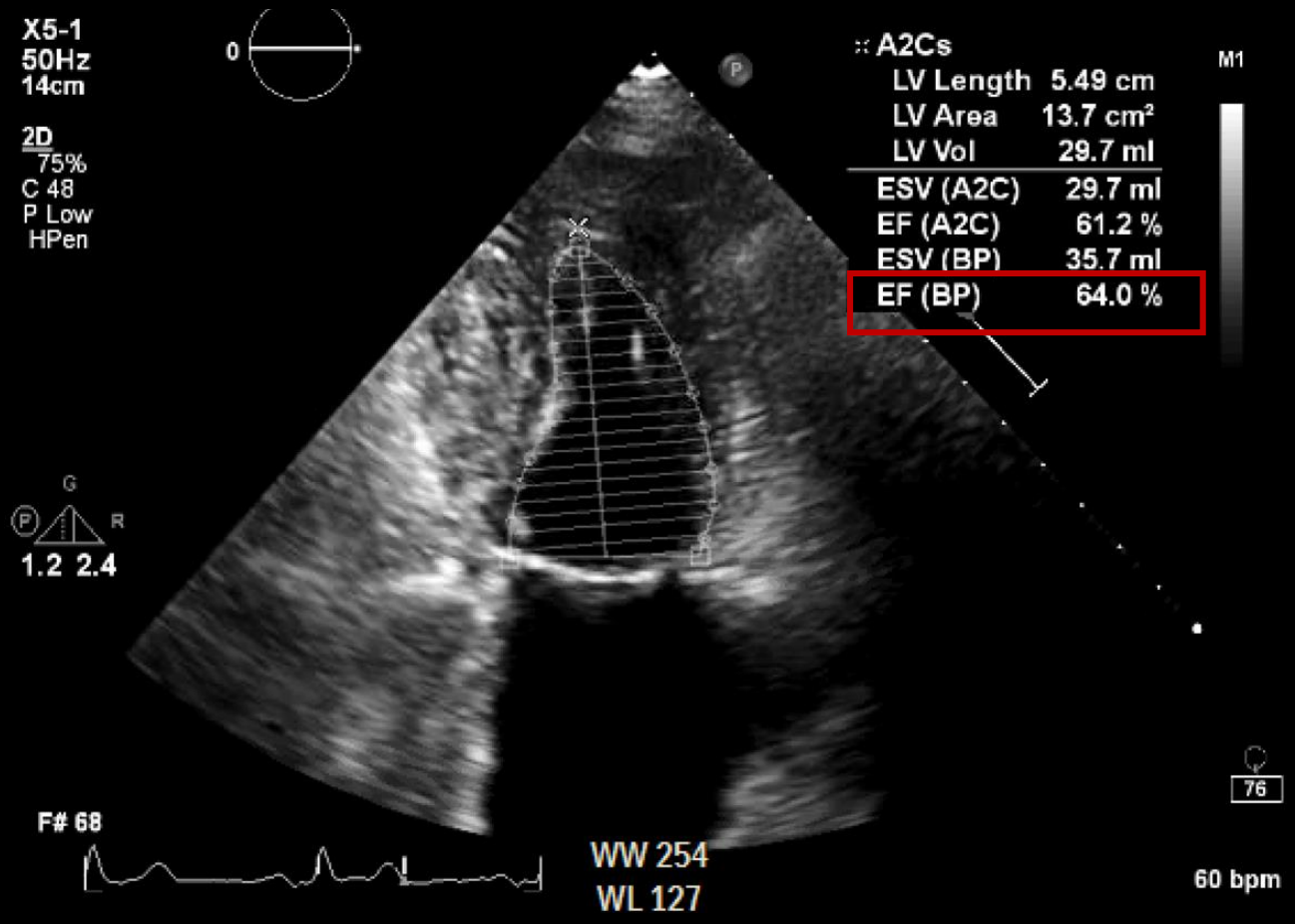
1. LV systolic function

Ejection Fraction

Preserved (EF ≥40%)

Decreased (EF <40%)





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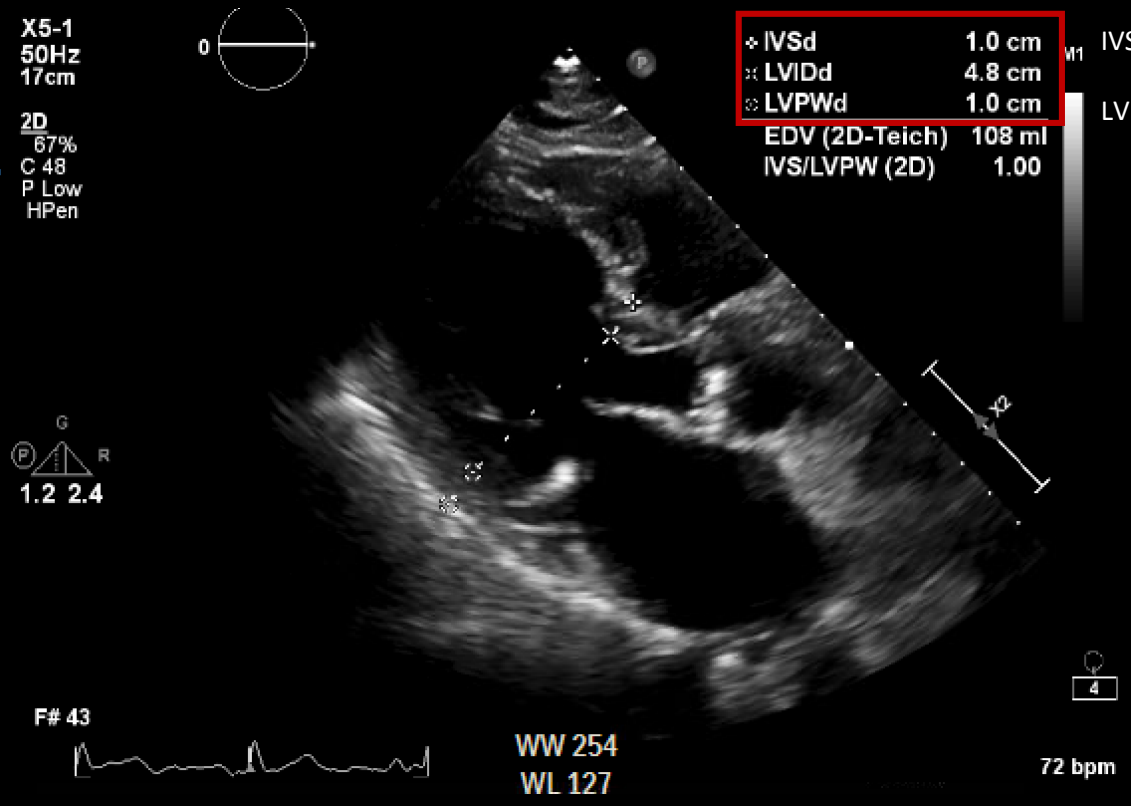
2. LV hypertrophy



IVS: interventricular septum (N: <1.0 cm)

LVPWd: LV posterior wall diameter (N:<1.0 cm)

The LaPlace's law:

$$\downarrow \text{Wall stress} = \frac{\text{Pressure} \times \text{Radius}}{\uparrow \text{Wall Thickness}}$$

Severe AS without LVH ?

Heart 1996;76:250–255

Severe aortic stenosis without left ventricular hypertrophy: prevalence, predictors, and short-term follow up after aortic valve replacement

Christian Seiler, Rolf Jenni

194 pts: **90%** - LVH, **10%** no LVH, **4%** had no macroscopically detectable hypertrophic adaptation

** small body size was an independent risk*

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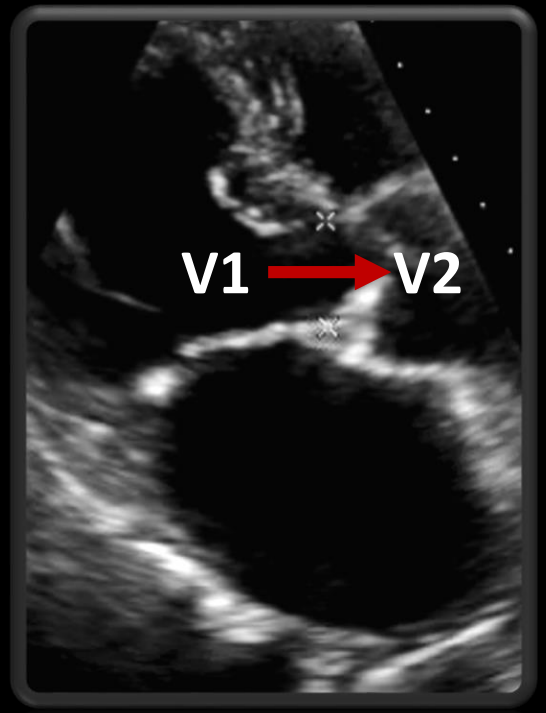
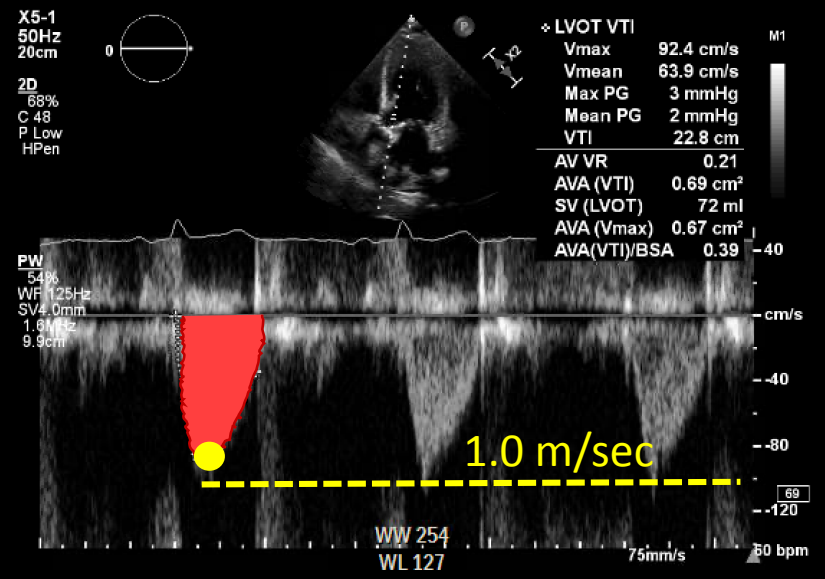
3. Aortic Valve hemodynamics



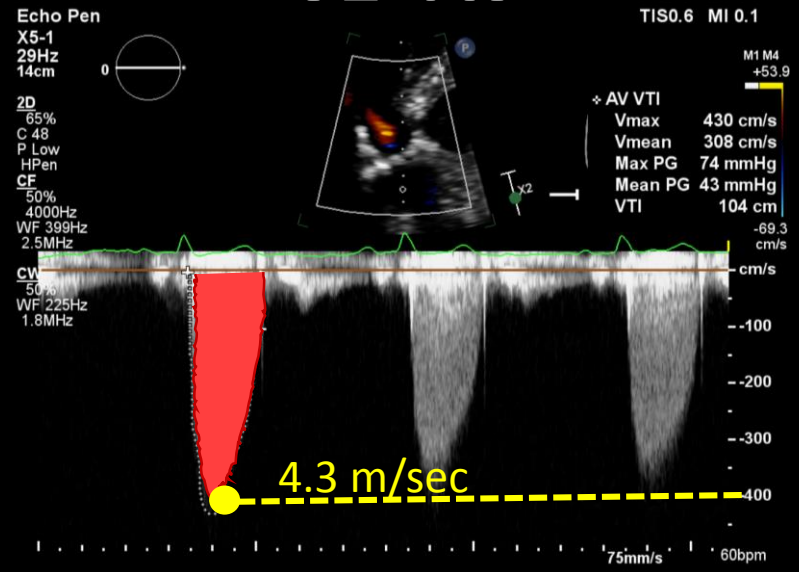
LV V1 VTI: 22.8 cm

V1=LVOT



Ao V2 VTI: 104 cm
Ao mean PG: 43 mmHg

V2=AV



Severe AS: Peak AV vel ≥ 4.0 m/sec

Severe AS: Mean AV gradient ≥ 40 mmHg

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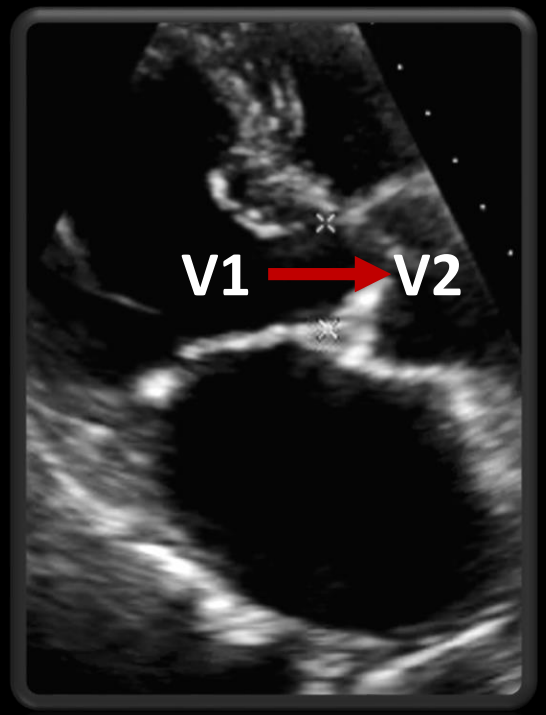
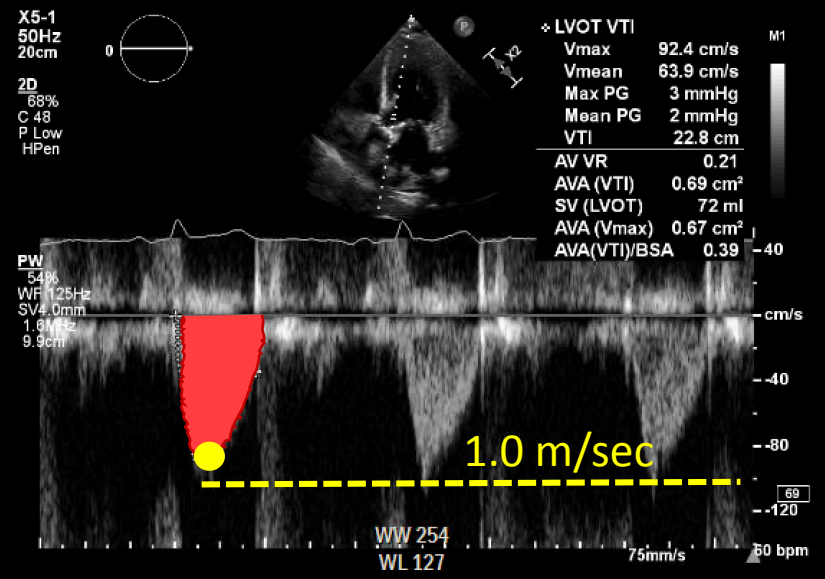
2. Aortic valve hemodynamics

LV V1 max: 92.4 cm/sec = 0.9 m/sec
LV V1 VTI: 22.8 cm

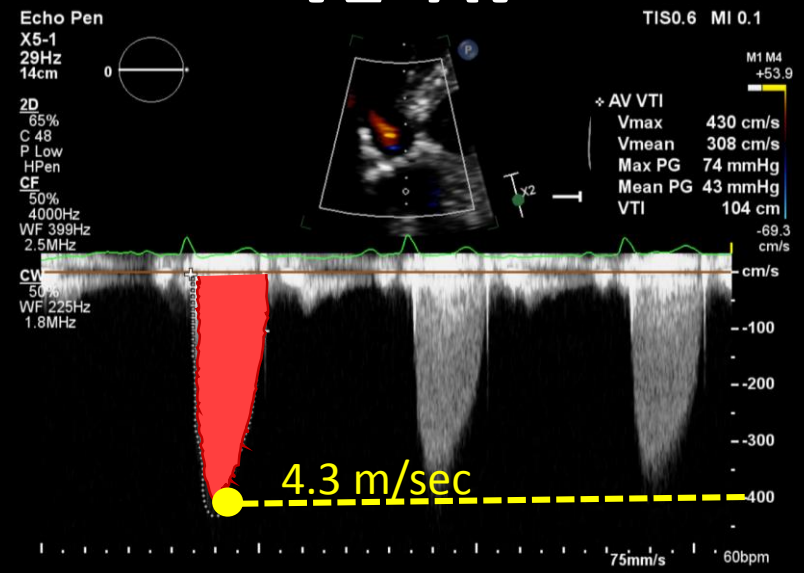
Ao V2 max: 430 cm/sec = 4.3 m/sec
Ao V2 VTI: 104 cm
Ao mean PG: 43 mmHg



V1=LVOT



V2=AV



$$\text{Dimensionless Index (Velocity Ratio)} = \frac{V1}{V2} = \frac{VTI_{LVOT}}{VTI_{AV}}$$

Severe AS: DI ≤ 0.25

$$= \frac{23}{104} = 0.22$$

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Otto CM et al, J ACC 1986:7:509; Oh JK et al, JACC 1988;11:1227

3. Aortic Valve Area (AVA)

Continuity Equation:

Concept: LVOT flow (SV) = AV flow (SV)

$AVA \times VTI_{AV} = CSA_{LVOT} \times VTI_{LVOT}$

A2 x V2 = A1 x V1

$$AVA\ (A2) = \frac{CSA_{LVOT} \times V1}{V2}$$

Dimensionless Index (DI)
or Velocity Ratio (VR)

Severe AS: AVA < 1cm²

AVA < 0.6 cm²/m²

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Natural History of Aortic Stenosis

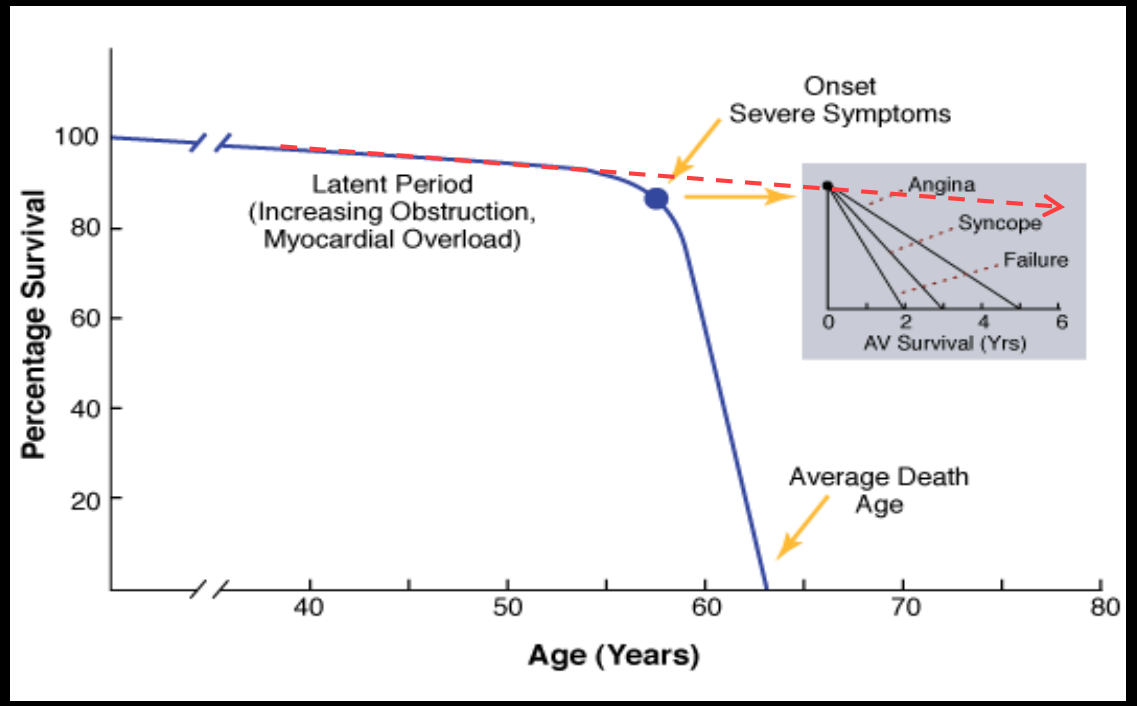
Stages:

A. At risk of AS

B. Progressive AS

C. Asymptomatic severe AS

D. Symptomatic severe AS



Ross J Jr. and Braunwald E, 1968; Circ 38; Suppl 5:61

2014 ASE/ACC Valvular Heart Disease Guideline, Nishimura et al, JACC 63:e57

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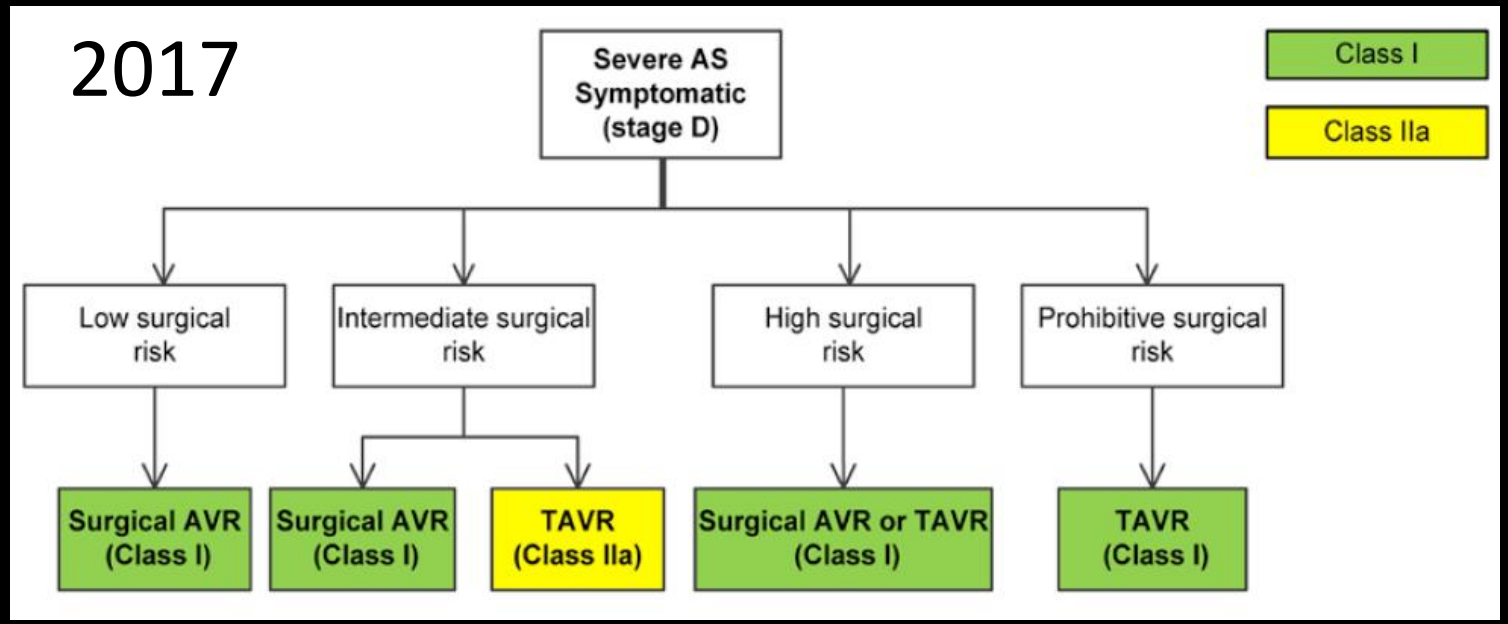
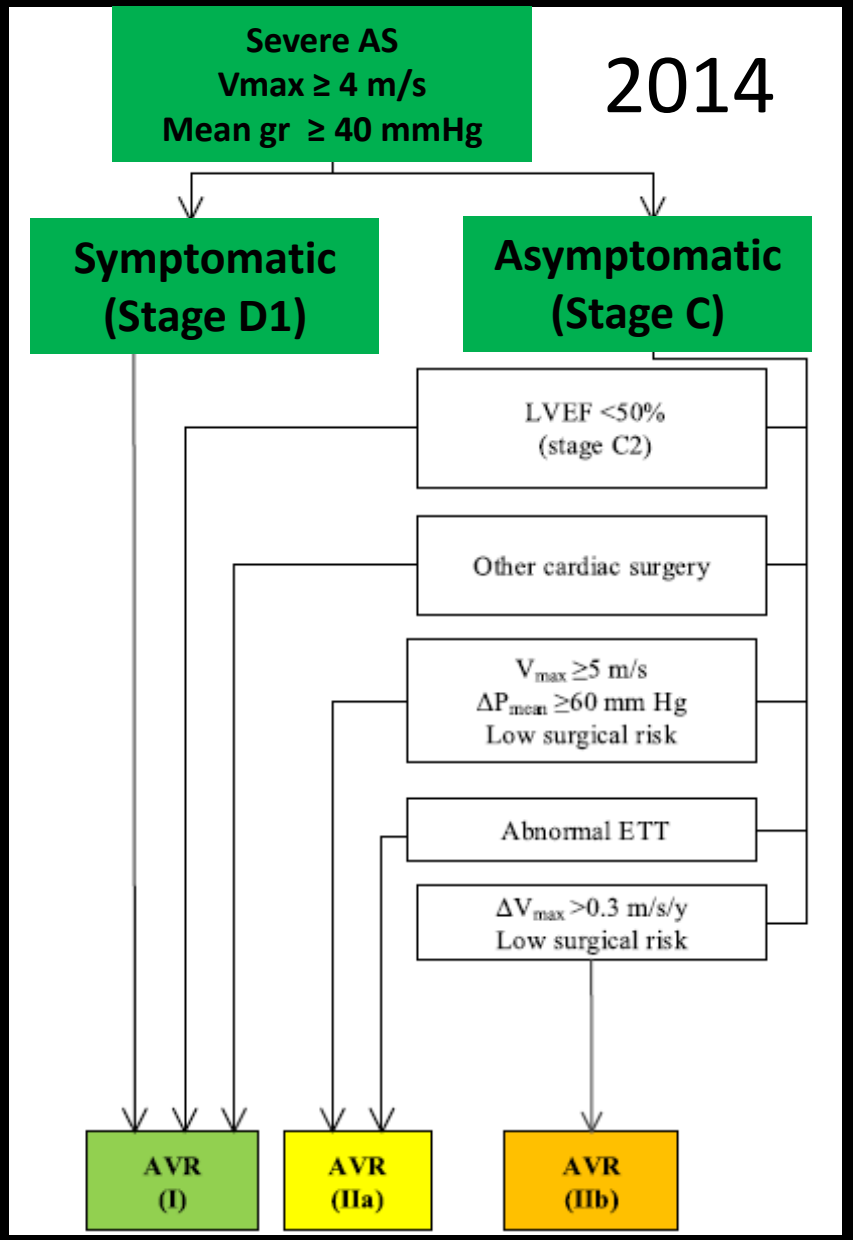
Severe AS - Treatment

- Operate at the ONSET of symptoms



Circulation. 2017;135:e1159–e1195



2014 ASE/ACC Valvular Heart Disease Guideline, Nishimura et al, JACC 63:e57

Take home points

1. Severe AS can be recognized on physical exam (soft S2)

2. AS severity should be assessed by Transthoracic Echocardiography:

AV max velocity	≥ 4.0 m/sec
AV mean gradient	≥ 40 mmHg
Dimensionless index/VR	≤ 0.25
AVA	< 1.0 cm^2 (<0.6 cm^2/m^2)
LV hypertrophy	+/-

3. Operate at the ONSET of symptoms, irrespective of LV function (surgical AVR or TAVR)

4. Severe asymptomatic AS remains a clinical challenge

Consult #2

Consult #2 - Objectives

At the conclusion of this activity, participants will be able to:

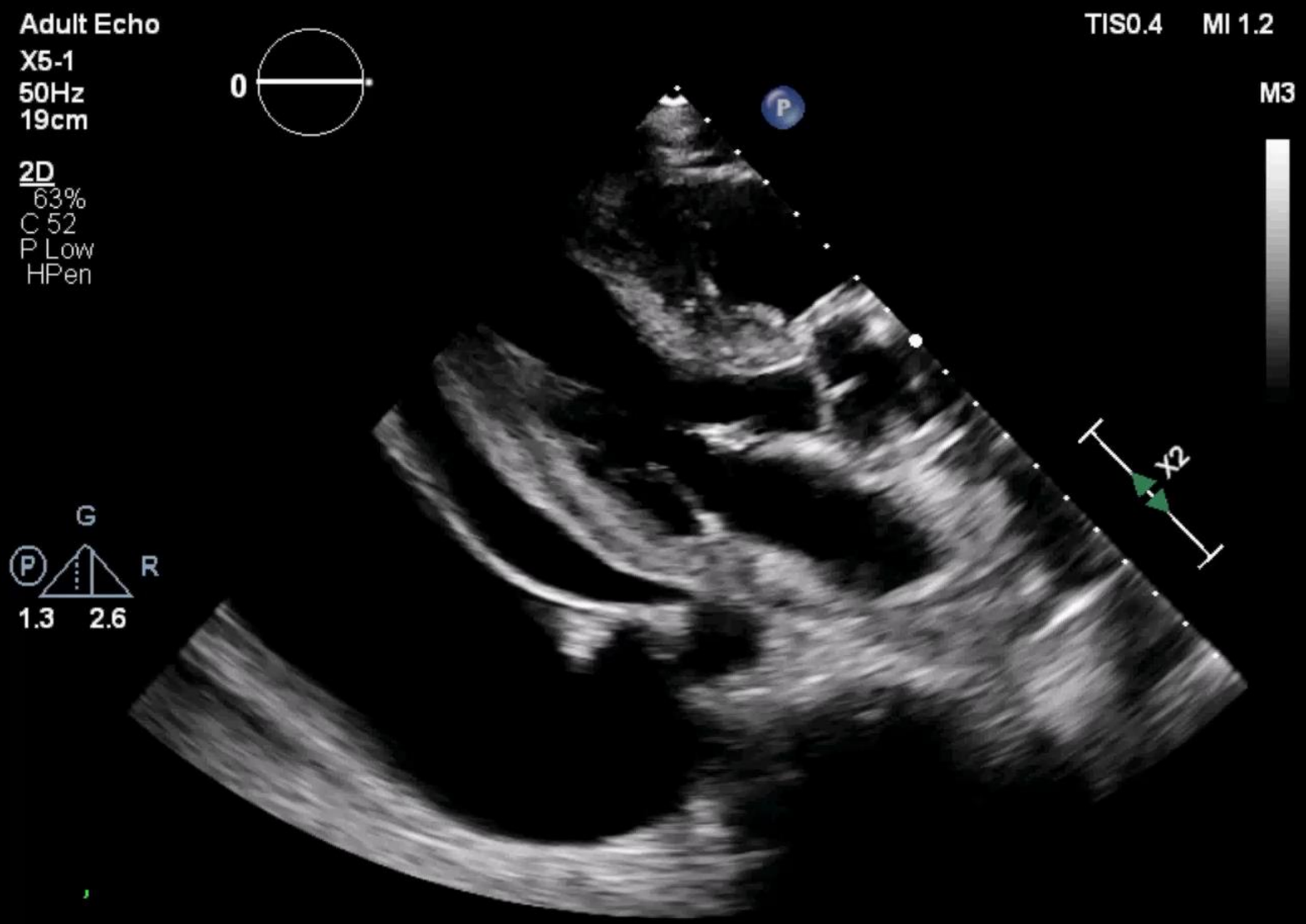
1. Be able to differentiate between pleural and pericardial effusion on transthoracic echocardiogram

2. Understand pathophysiology of cardiac tamponade

3. Recognize echocardiographic features of cardiac tamponade

Curbside Consult

A 62 yo M with SOB and a large pericardial effusion on POCUS. Does he need emergent pericardiocenthesis?



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Questions:

1. Is this pericardial effusion or pleural effusion?

2. Is hemodynamic compromise present?

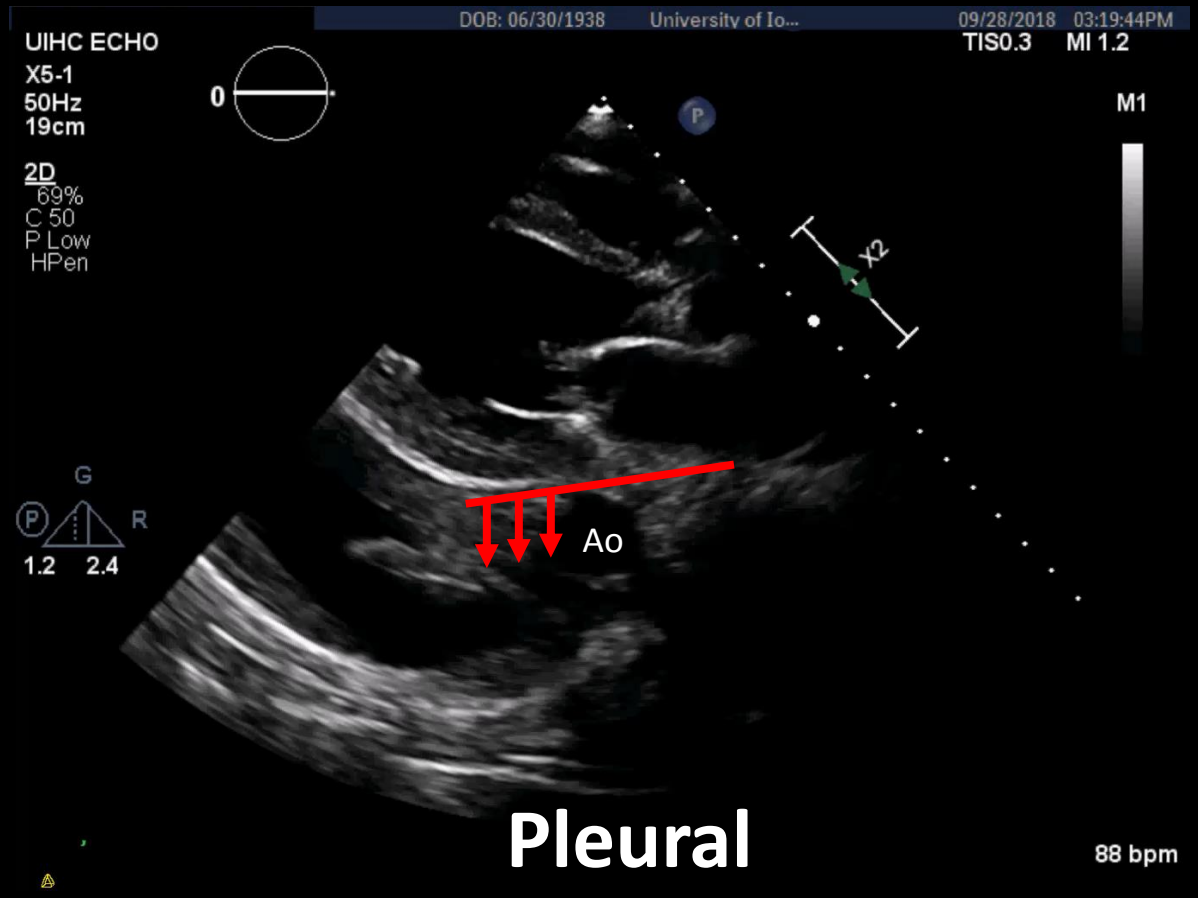
3. Can it be drained percutaneously?

PLEURAL vs PERICRDIAL effusion?

STEP 1: Obtain Parasternal Long Axis view

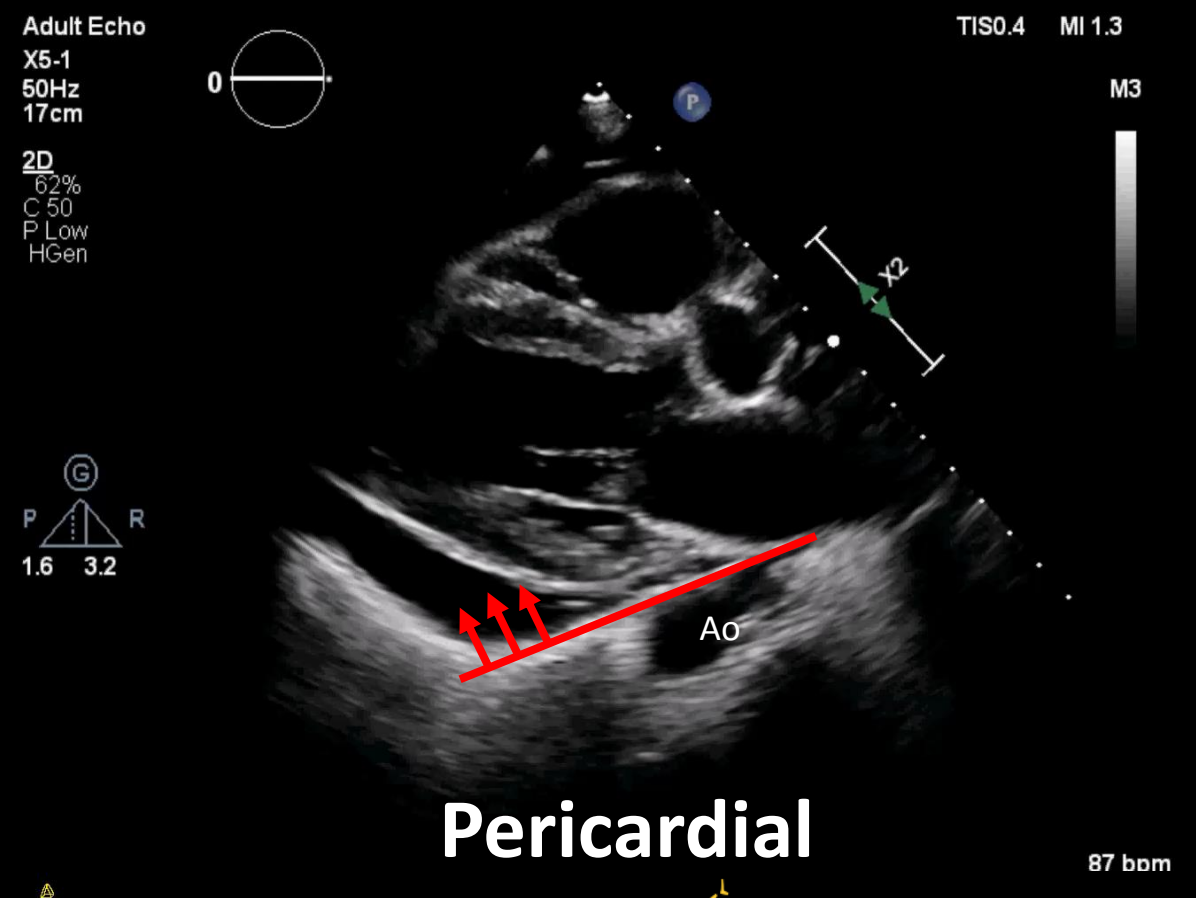
STEP 2: Find descending thoracic Aorta

If effusion is POSTERIOR to Aorta



Pleural

If effusion is ANTERIOR to Aorta



Pericardial

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Curbside Consult

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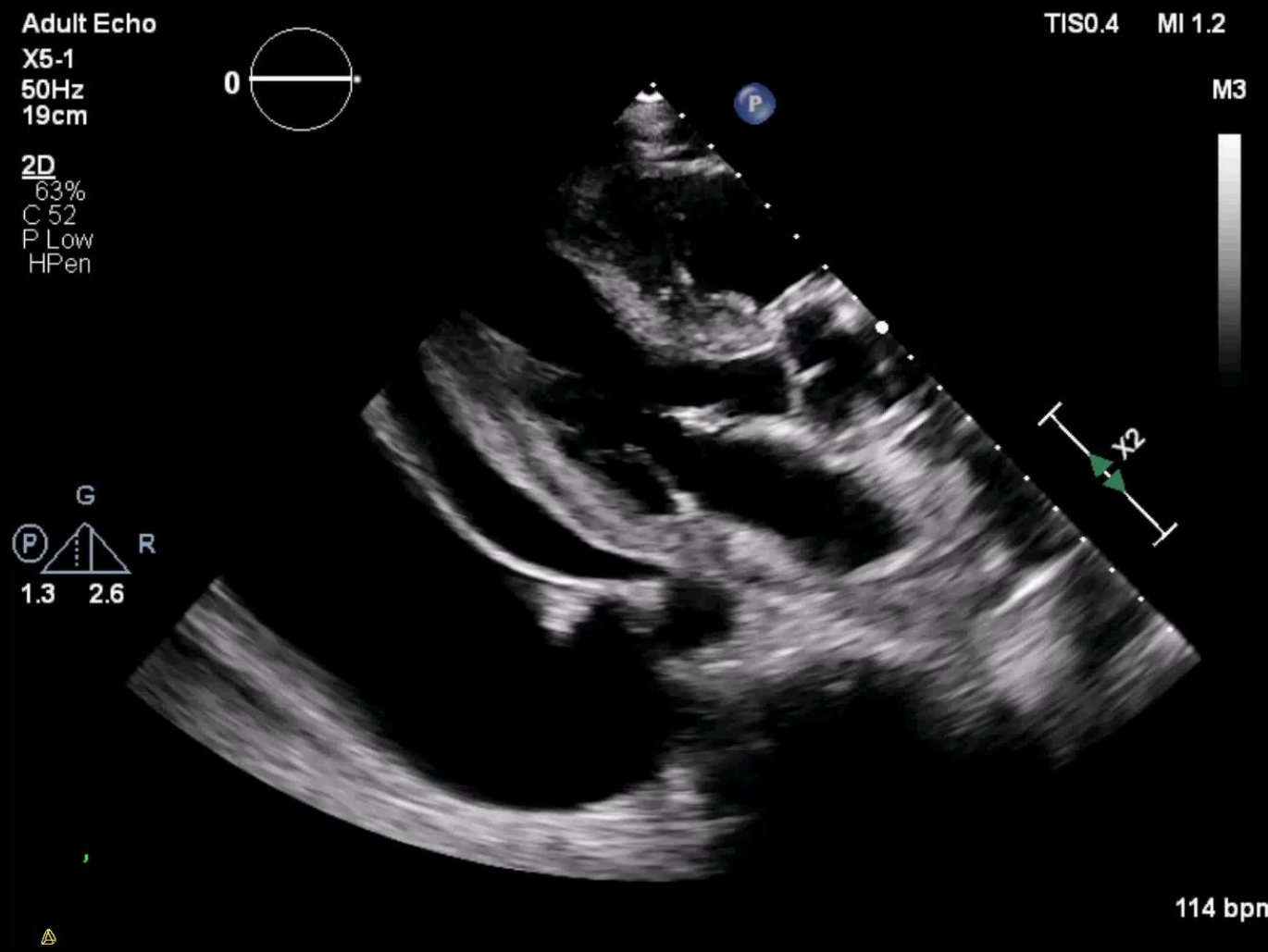

Large pleural effusion, small to moderate pericardial effusion

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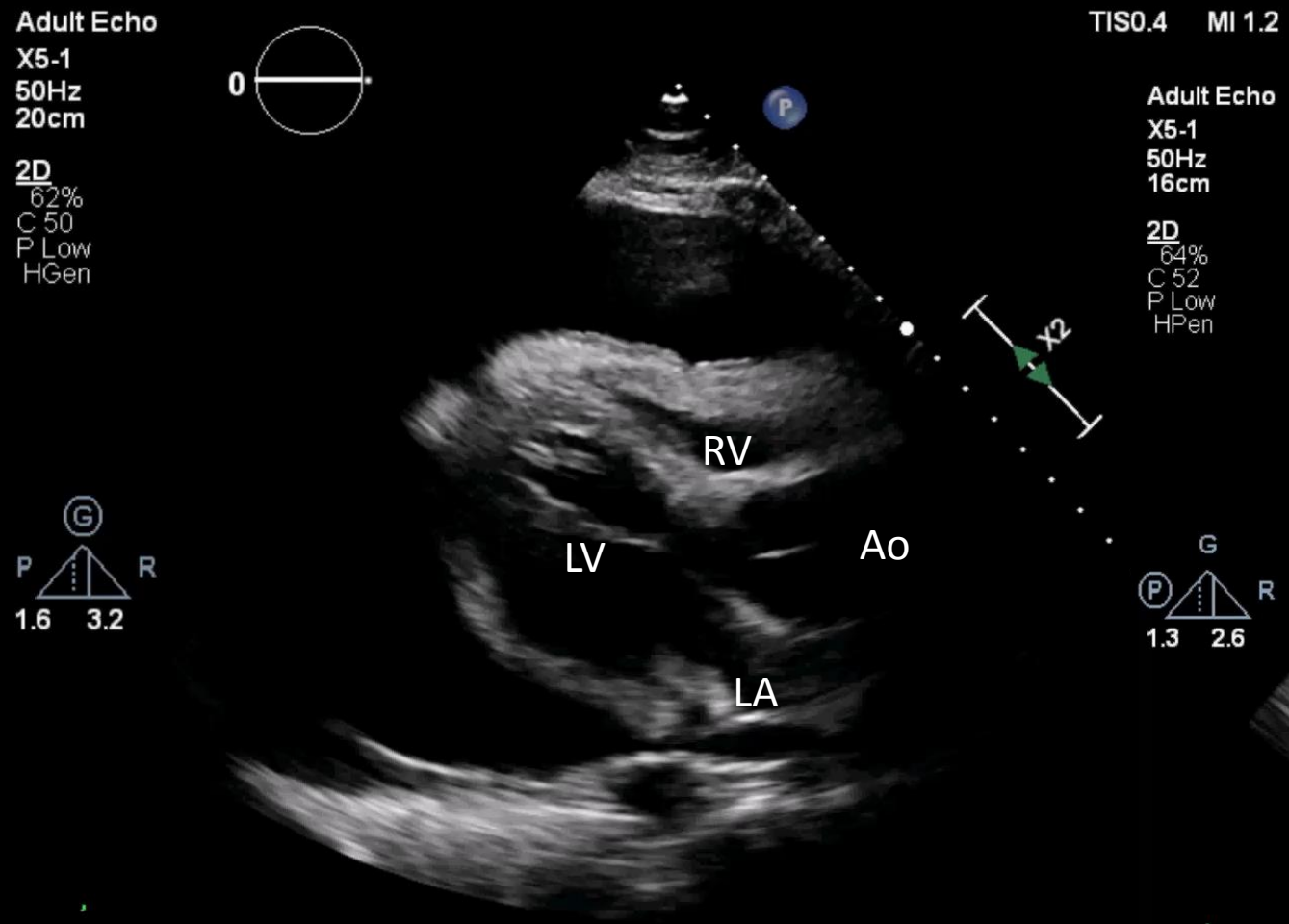
STAT CONSULT

A 59-yo F with Hx of breast cancer presents to the ER with worsening shortness of breath. HR 120 bpm, BP 95/72 mmHg, RR 20/min. On exam her extremities are cool, neck veins are distended, and heart sounds are distant:

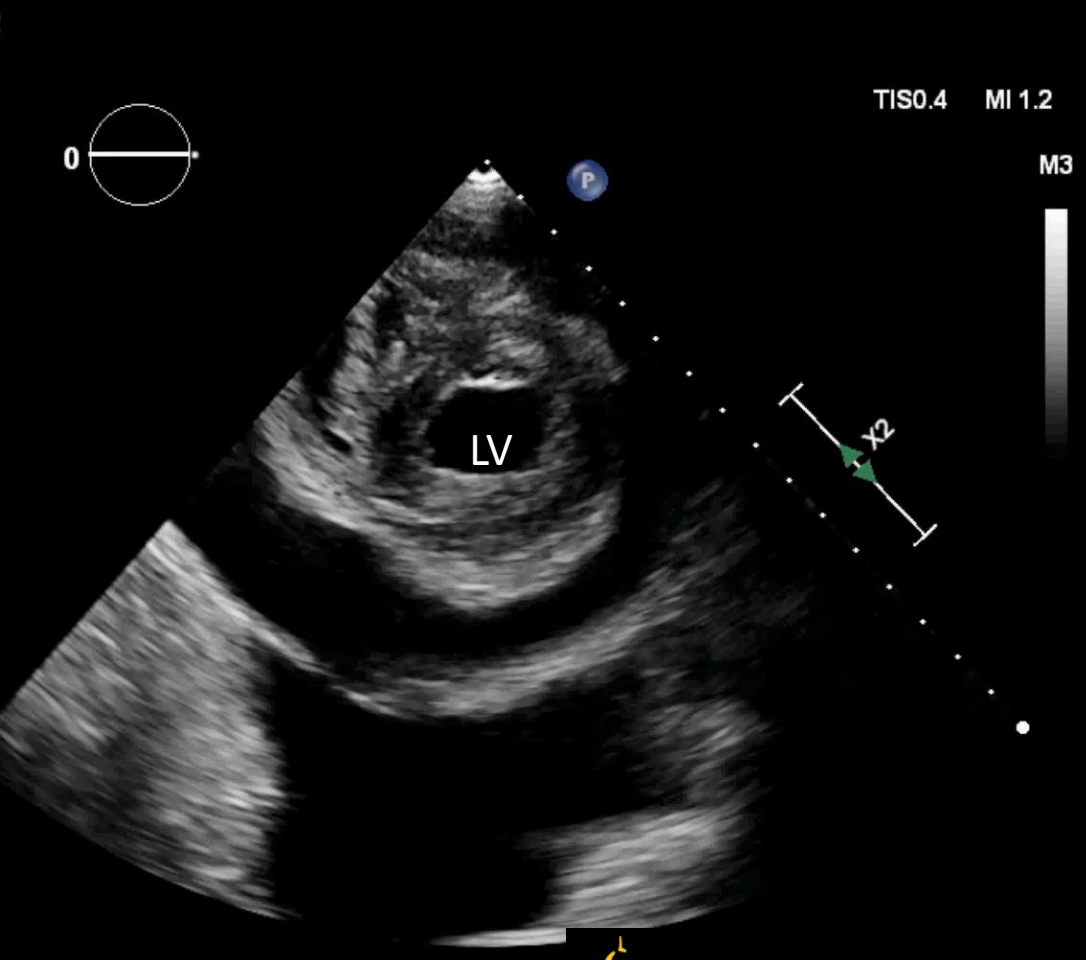
STAT CONSULT

A 59-yo F with Hx of breast cancer presents to the ER with worsening shortness of breath. HR 120 bpm, BP 95/72 mmHg, RR 20/min. On exam her extremities are cool, neck veins are distended, and heart sounds are distant:

Parasternal long axis view



Parasternal short axis view



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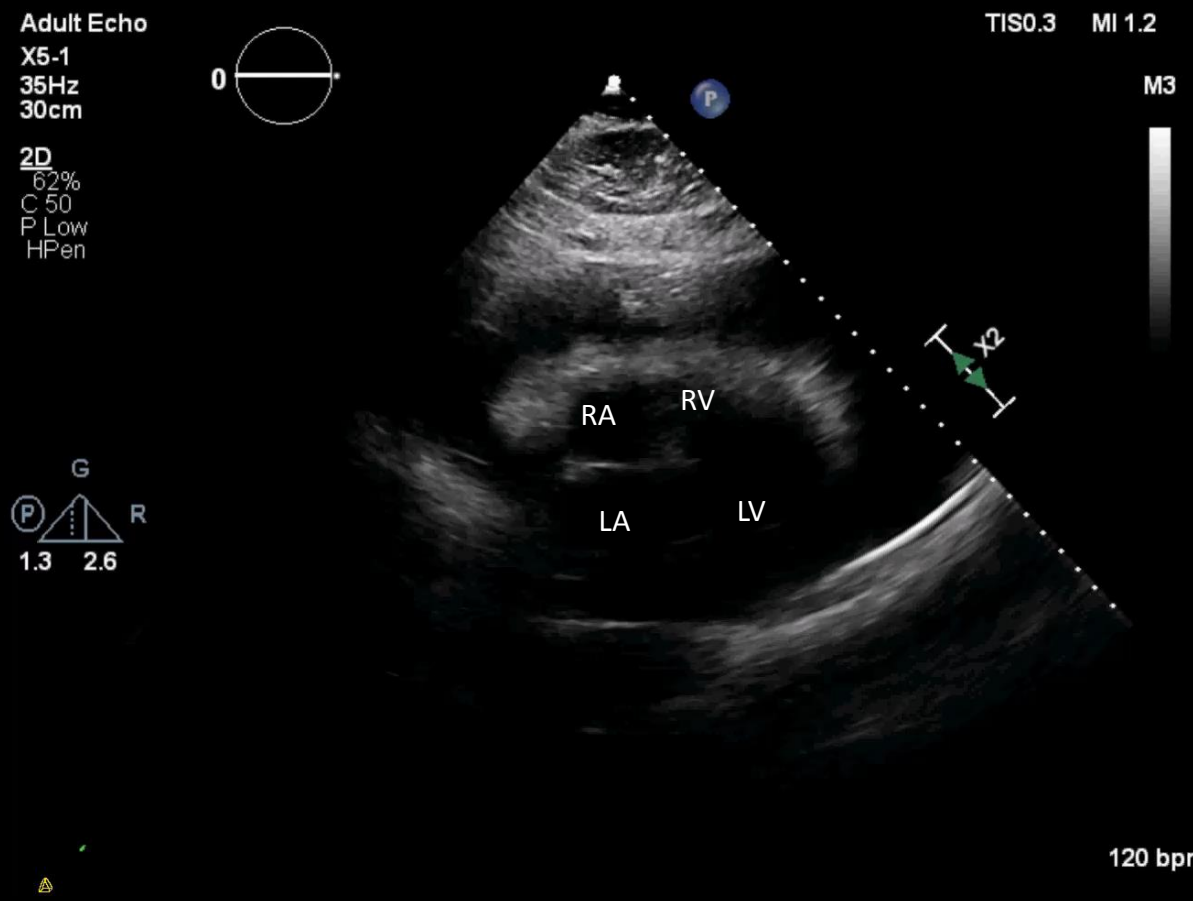
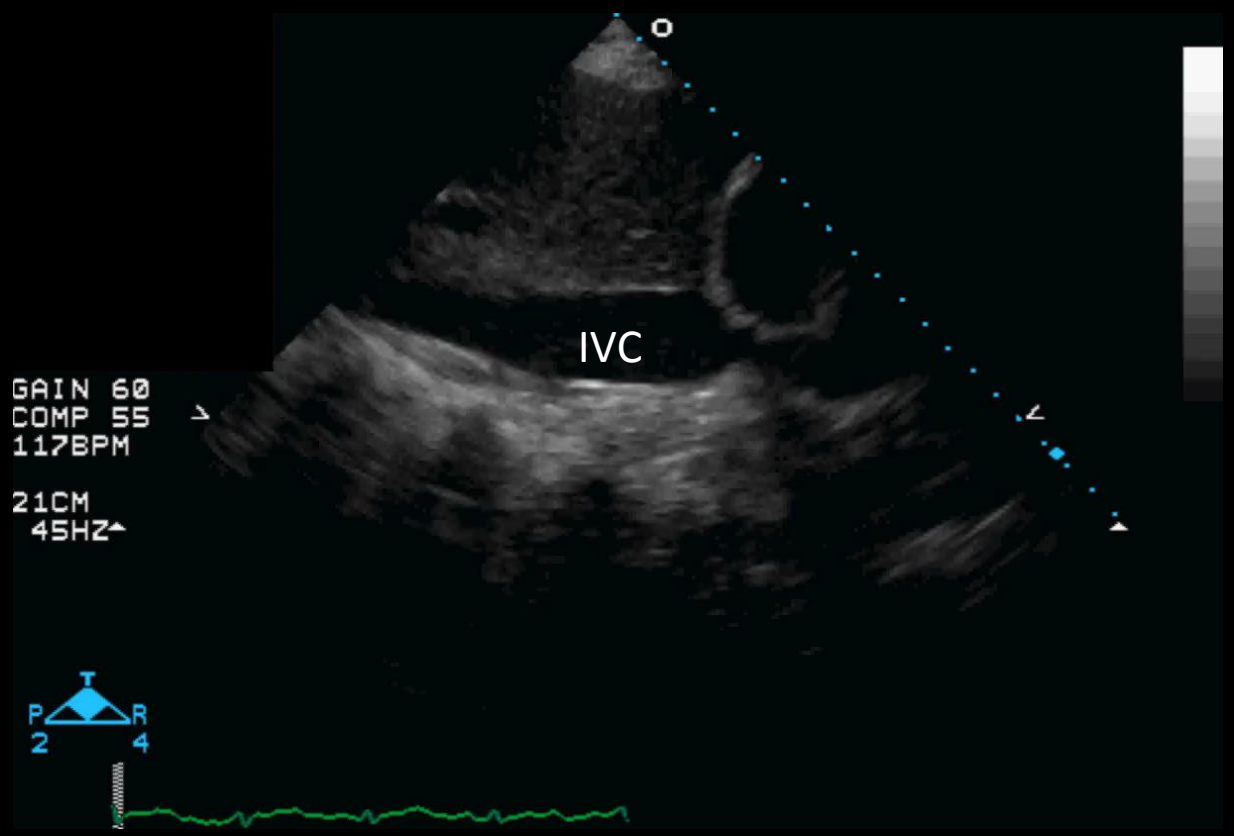
Subcostal view



Subcostal IVC view



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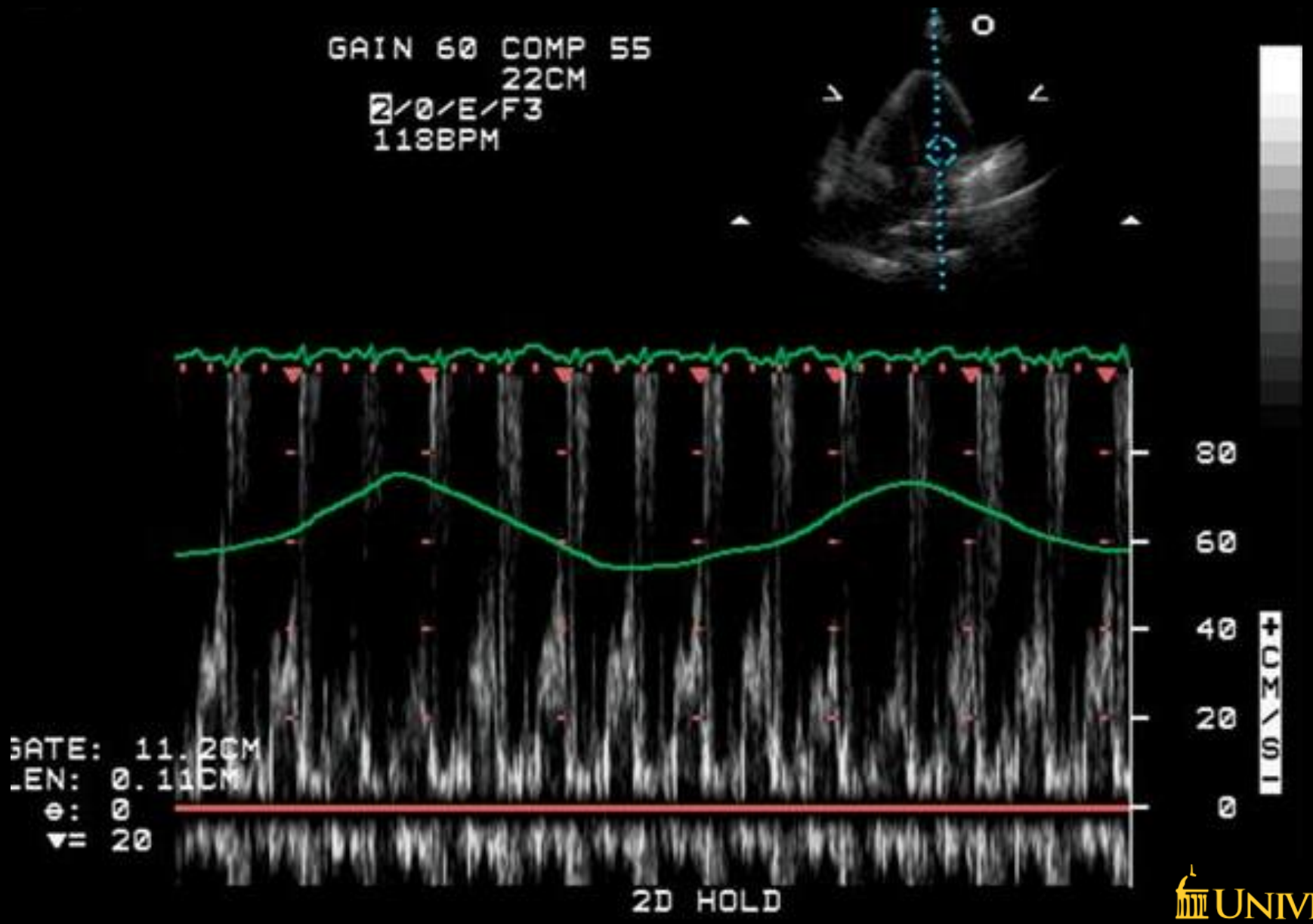
PW Doppler through MV with Respirometer



Questions:

1. Is this pericardial effusion or pleural effusion?

2. Is hemodynamic compromise present?

3. Can it be drained percutaneously?

Cardiac Tamponade

Cardiac Tamponade

- Clinical diagnosis; Not diagnosed by Echo
- Clinical syndrome of :
  Hypotension
  Tachycardia
  Symptoms due to higher intrapericardial pressure than intrathoracic pressure; Pulsus paradoxus:

https://www.youtube.com/watch?v=d4aCDhMvb0M

Cardiac Tamponade

- Symptoms: dyspnea, chest pain, and/or non-specific sense of discomfort

- Physical exam:

  - Pulsus paradoxus (an inspiratory decline >10 mmHg in SBP)
  - Beck's triad: hypotension, muffled heart sounds, and elevated JVP
  - EKG: low voltage and electric alternans
  - CXR: flask-like or "boot-shaped" appearance of cardiac silhouette

Cardiac Tamponade - Pathophysiology

- Pericardial Pressure > Intrathoracic pressure

- Impaired cardiac filling

- As fluid accumulates in the pericardial sac, pericardial pressure rises, and systemic and pulmonary venous pressures must increase to maintain cardiac filling. When compensatory mechanisms are exhausted, preload becomes insufficient to sustain cardiac filling and coronary and systemic perfusion

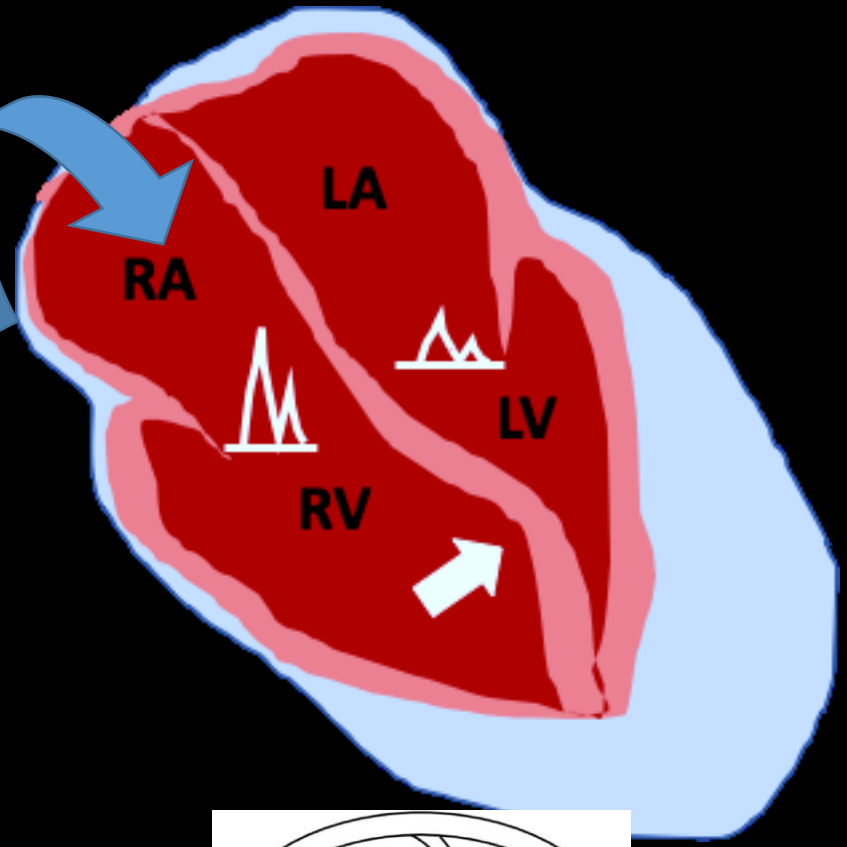
Cardiac Tamponade - Pathophysiology

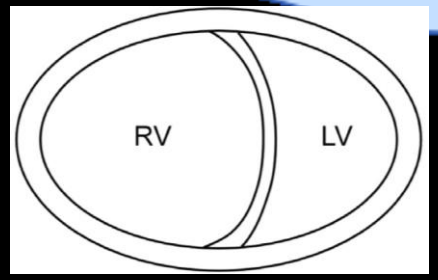
- Lower-pressure chambers (atria) are affected before higher-pressure chambers (ventricles)

- The compressive effect of the pericardial fluid is seen most clearly in the phase of the cardiac cycle when pressure is lowest in that chamber—systole for the atrium, diastole for the ventricles

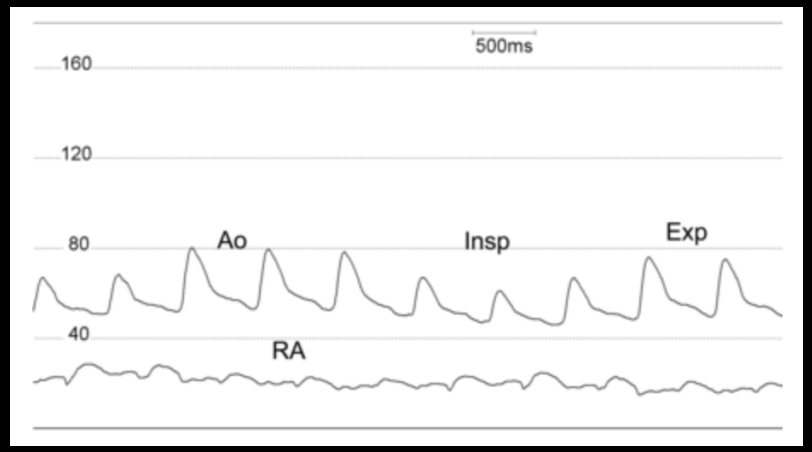
Cardiac Tamponade - Pathophysiology

INSPIRATION



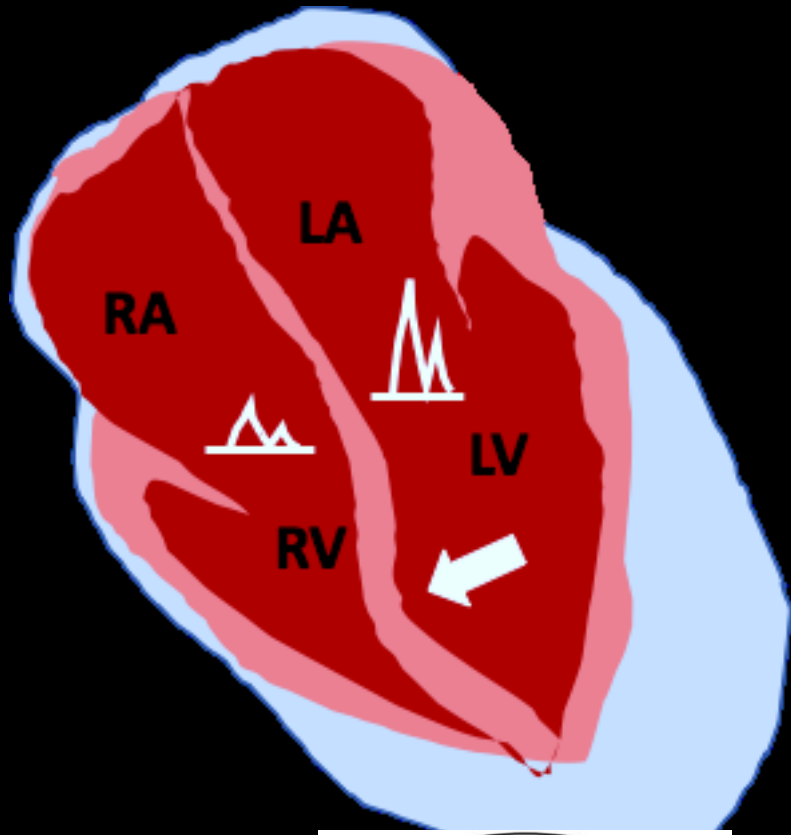


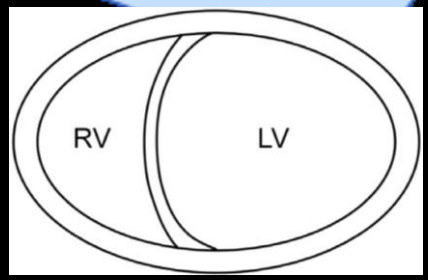
Pulsus paradoxus: SBP drop (>10 mmHg)



Ventricular Interdependence

EXPIRATION





J Am Soc Echocardiogr 2013;26:965-1012

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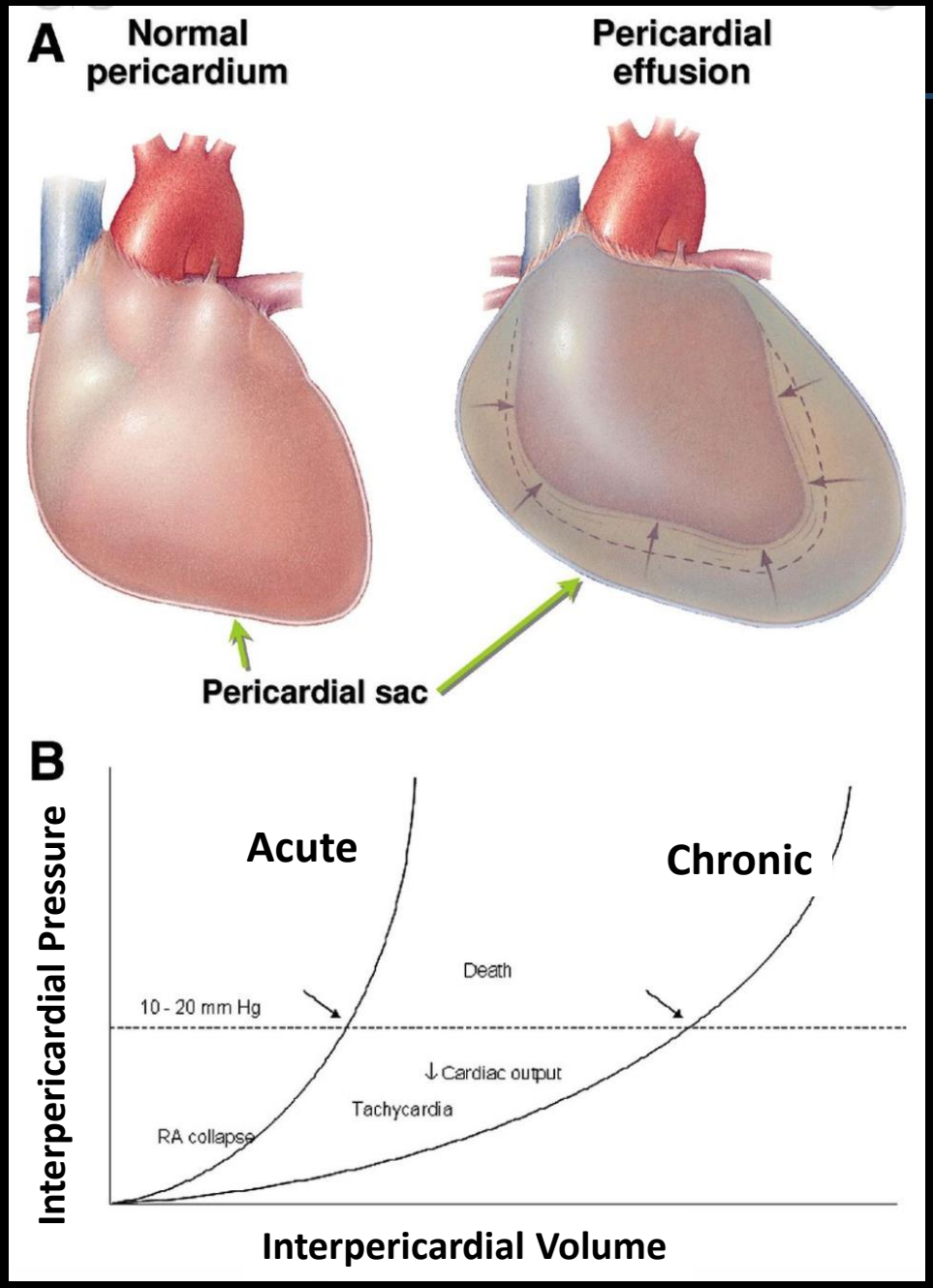
Cardiac Tamponade - Pathophysiology

- The size of the effusion, its rate of accumulation, and any pathology altering the pressure-volume relation of the pericardium determine the extent of hemodynamic compromise.

Pericardium - Pressure Volume Curves



CHRONICALLY
- slowly developing effusions are safer
- pericardium stretches
- tamponade / large volume effusion

ACUTELY
- rapidly developing effusion
- pericardium has no time to stretch
- tamponade may occur with small volume

* The flat initial segments of the curves represent the pericardial reserve volume that once exceeded, causes a steep increase in pressure.

Iatrogenic Pericardial Effusion and Tamponade in the Percutaneous Intracardiac Intervention Era.
Holmes Jr DR, Nishimura R, Fountain R and Turi ZG. JACC: Cardiovascular Interventions; 2009: Vol 2(8)

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Echocardiographic features of Cardiac Tamponade

Cardiac Tamponade - Echocardiographic features

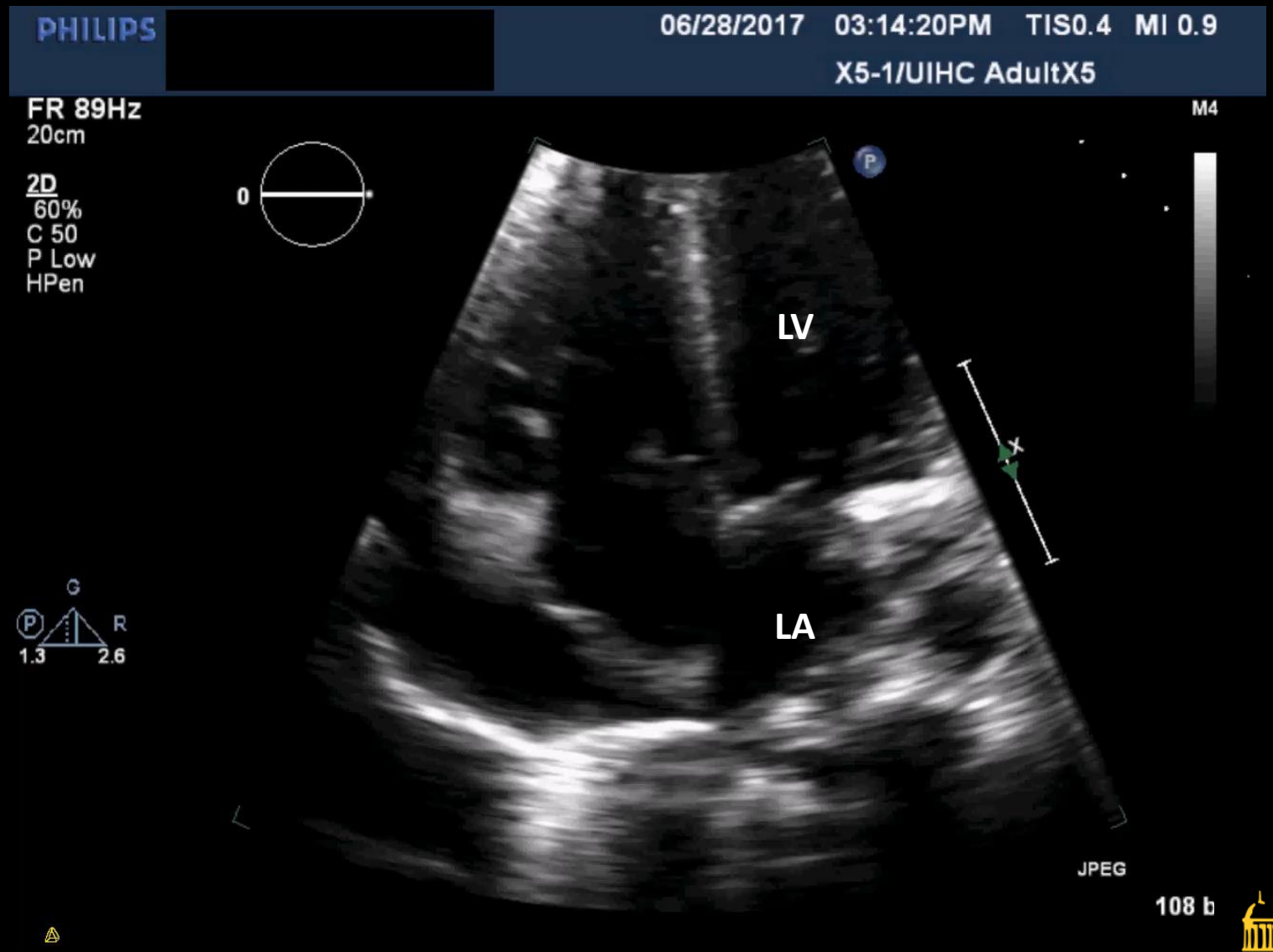
Physiologic changes evident on echocardiographic and Doppler examination include:

1. RA systolic collapse for greater than 1/3 of systole
2. RV diastolic collapse
3. Reciprocal respiratory changes in RV and LV volumes (septal shifting)
4. Reciprocal respiratory changes (>25%) in RV and LV filling
5. Severe dilation of the inferior vena cava/"plethoric IVC"

Otto C. Textbook of Clinical Echocardiography. 6th ed. 2018

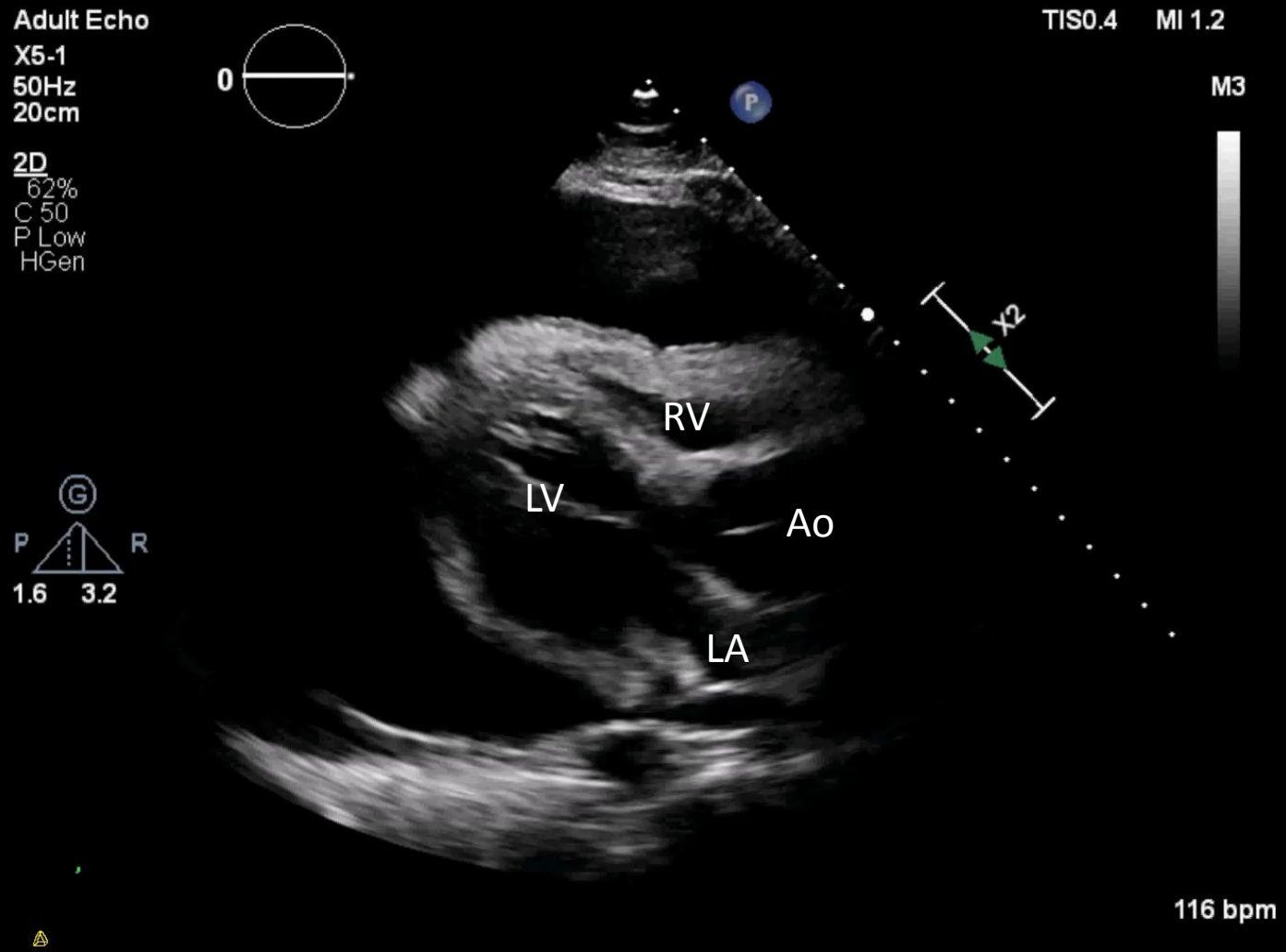
Cardiac Tamponade - Echocardiographic features

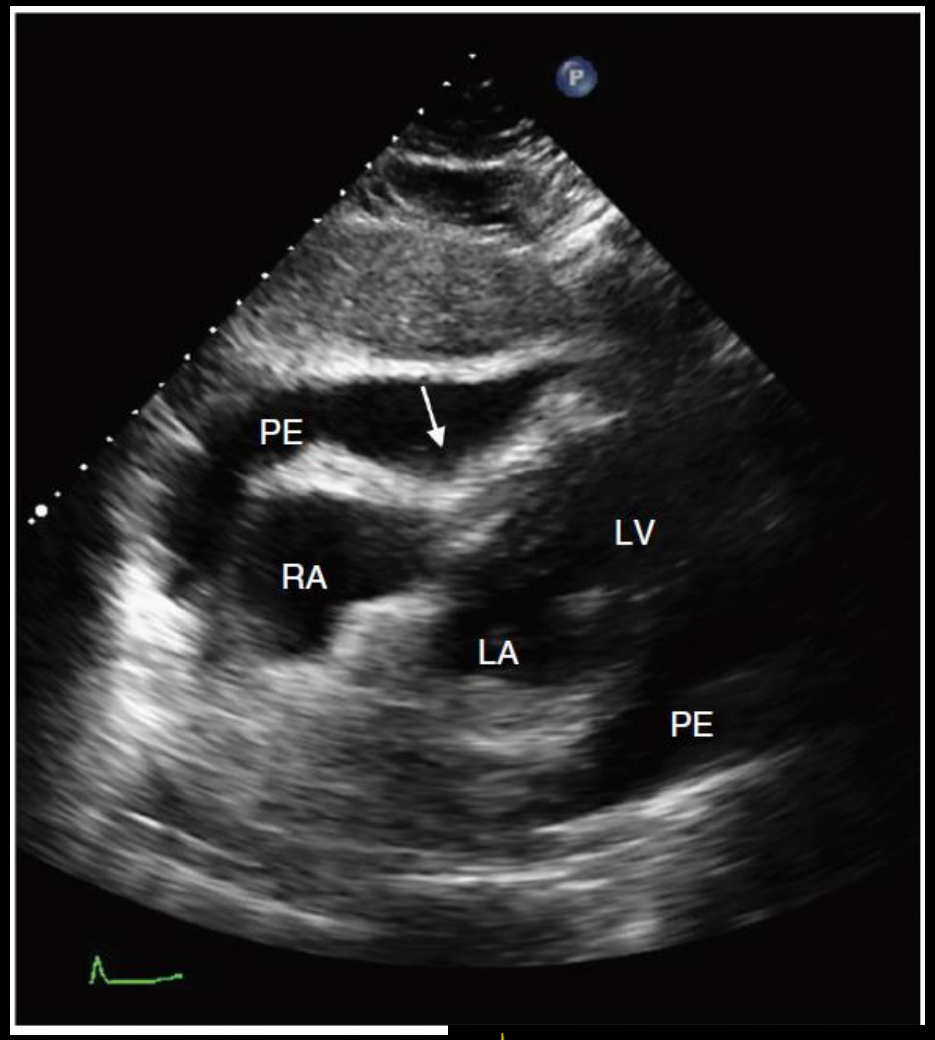
1. RA systolic collapse for greater than 1/3 of systole



Cardiac Tamponade - Echocardiographic features

2. RV Diastolic Collapse





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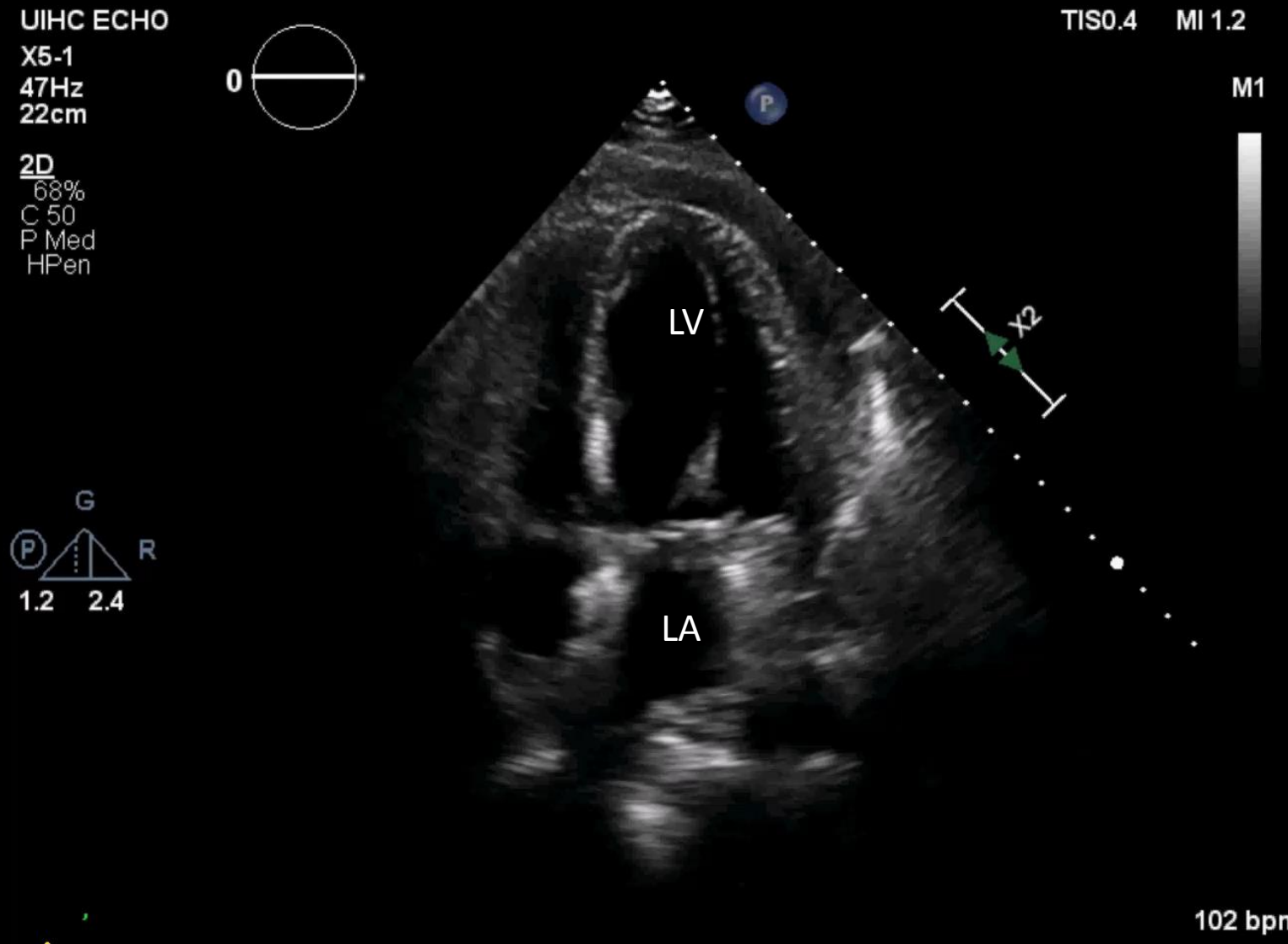
Cardiac Tamponade - Echocardiographic features

Patients with pulmonary hypertension may NOT have Right sided chambers compression

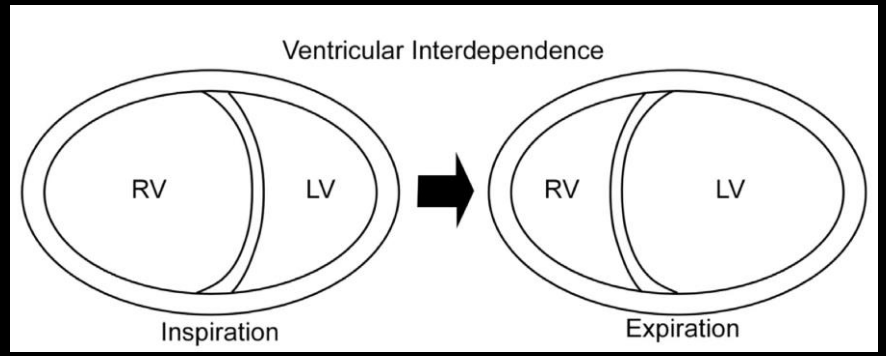
Cardiac Tamponade - Echocardiographic features

3. Reciprocal respiratory changes in RV and LV volumes (septal shifting)



Inspiratory septal bulge / "bounce"

- Cardiac tamponade
- COPD
- Pulmonary embolism



J Am Soc Echocardiogr 2013;26:965-1012

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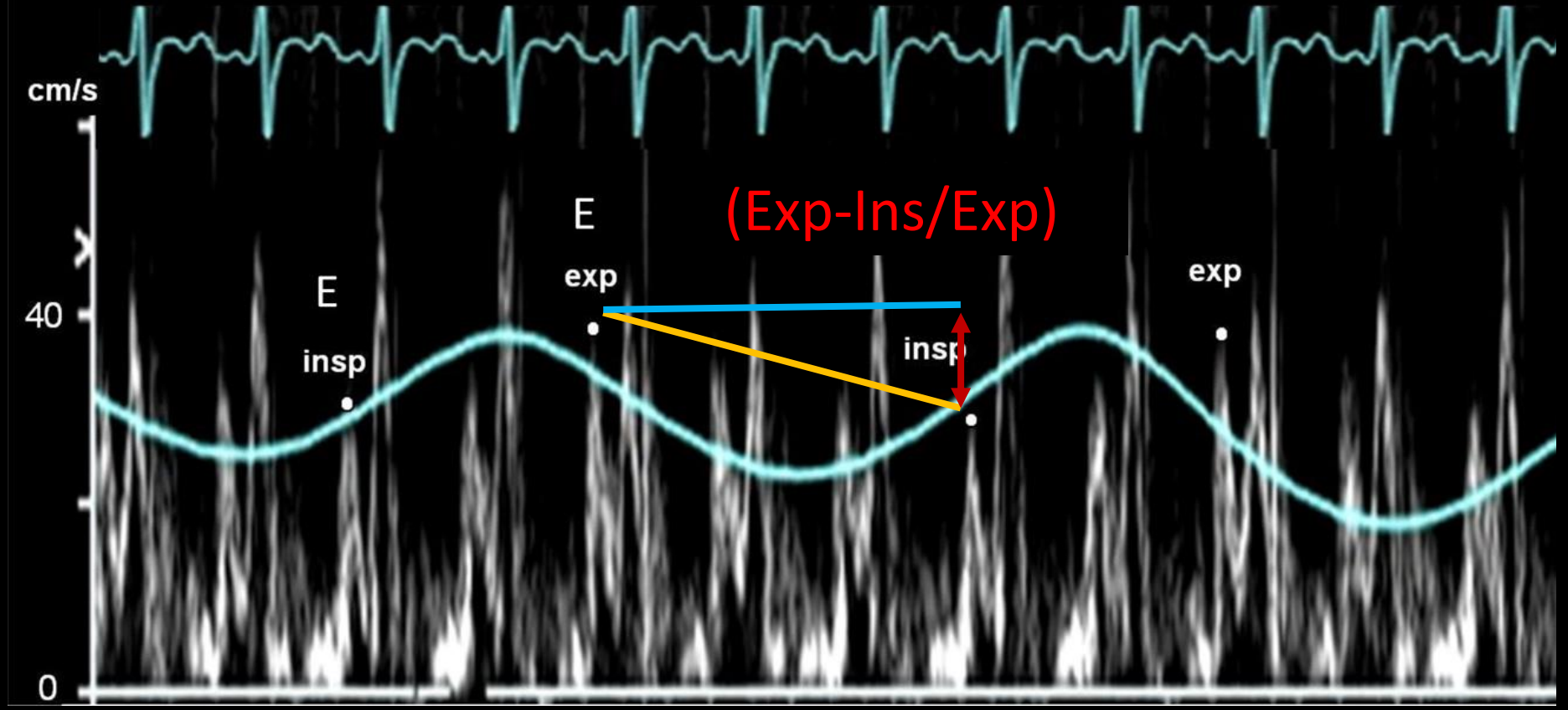
Cardiac Tamponade - Echocardiographic features

4. Reciprocal respiratory changes (>25%) in RV and LV filling

PW Doppler of TV and MV inflow with a respirometer during cardiac tamponade



ASE Clinical Recommendations for Multimodality Cardiovascular Imaging of Patients with Pericardial Disease.
J Am Soc Echocardiogr 2013;26:965-1012

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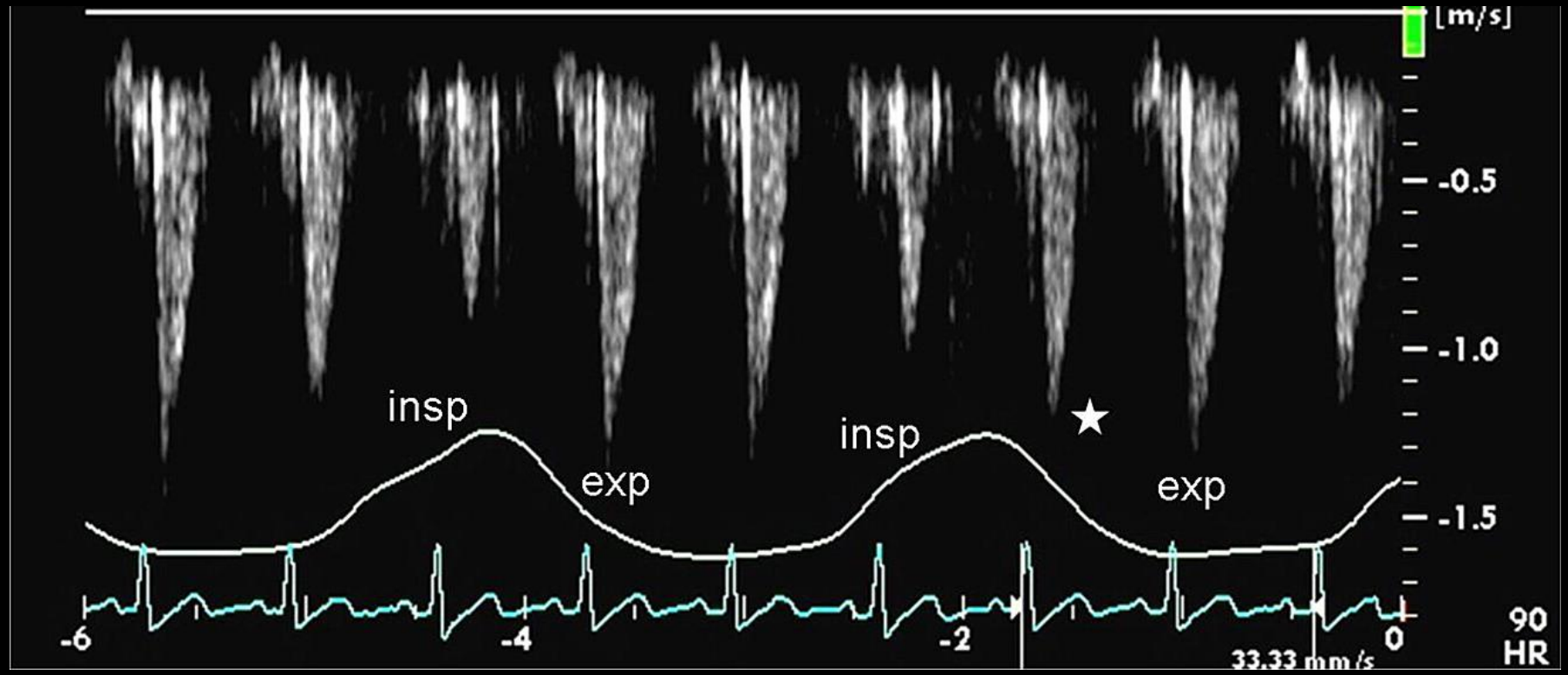
Cardiac Tamponade - Echocardiographic features

PW Doppler recording of LV outflow tract velocity and respirometer in a patient with cardiac tamponade and pulsus paradoxus



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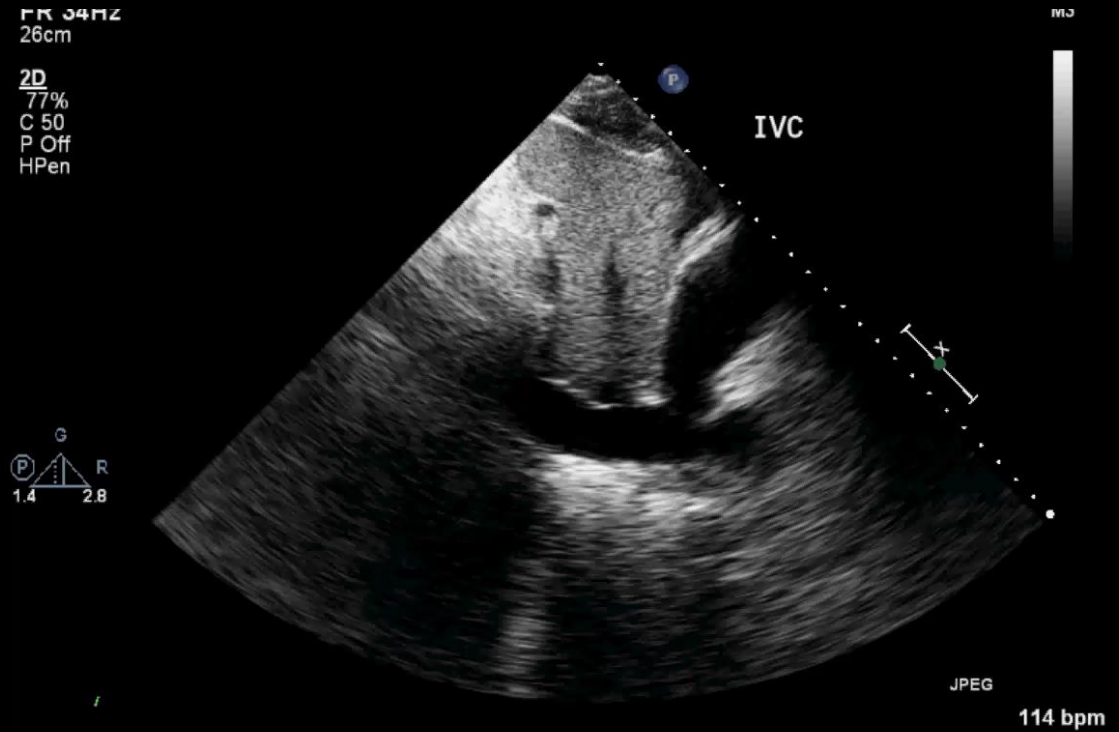
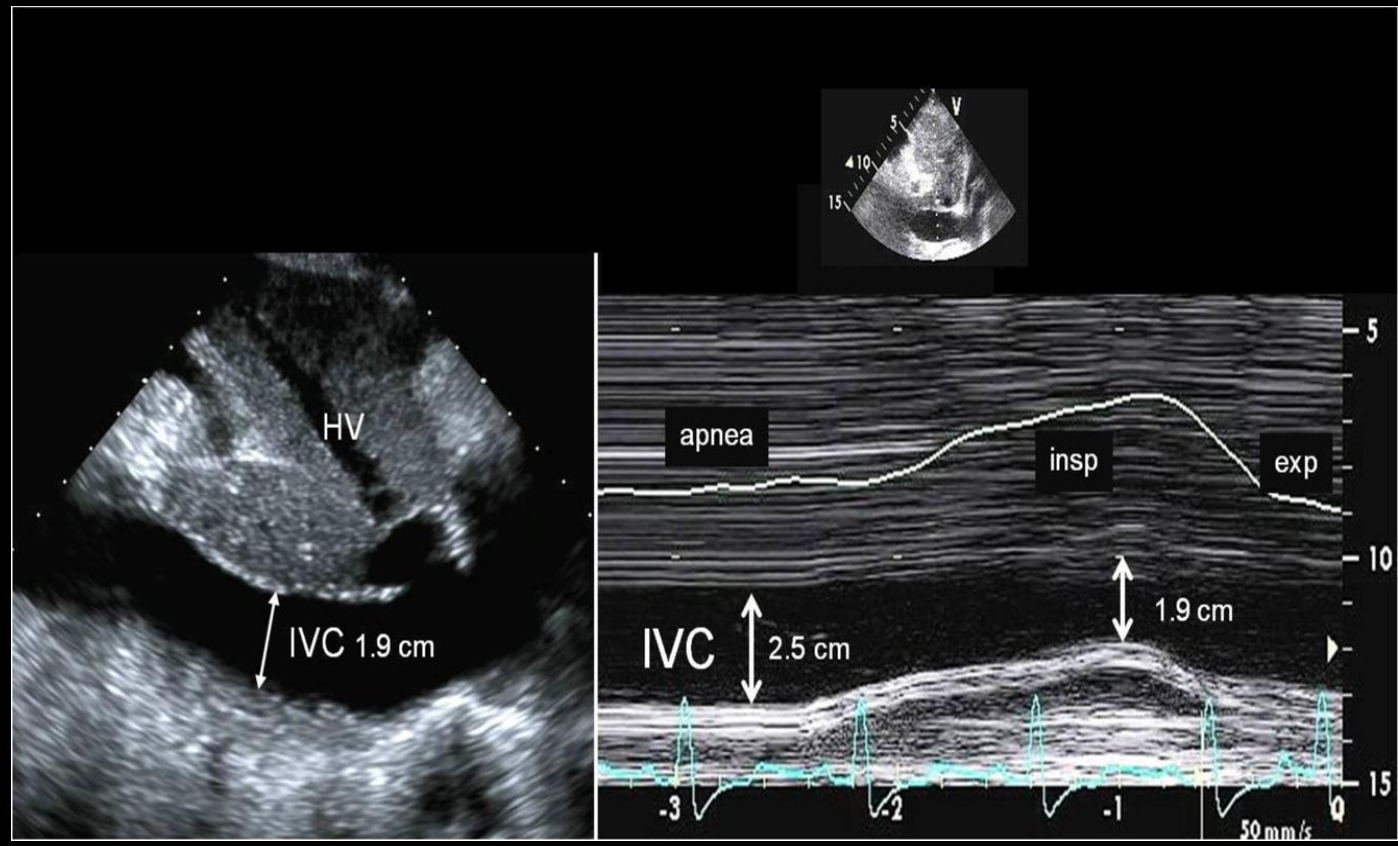
Cardiac Tamponade - Echocardiographic features

5. Severe dilation of the inferior vena cava/"plethoric IVC"





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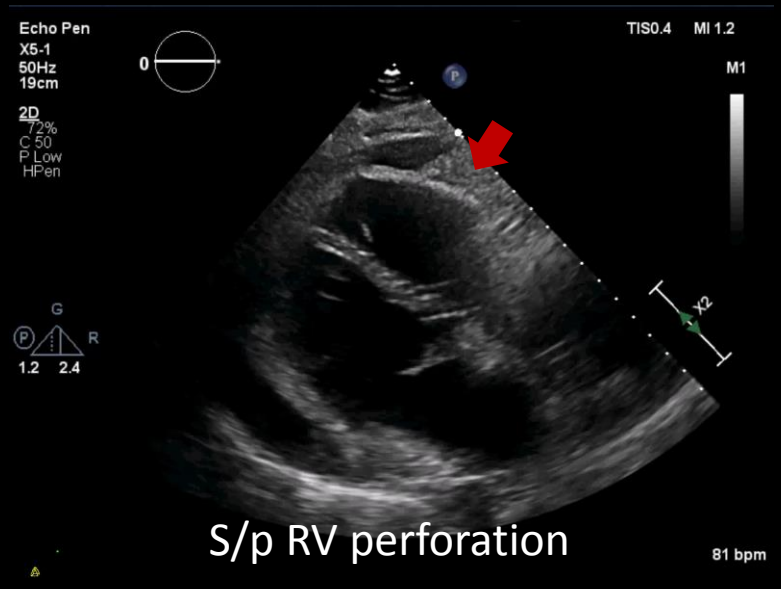
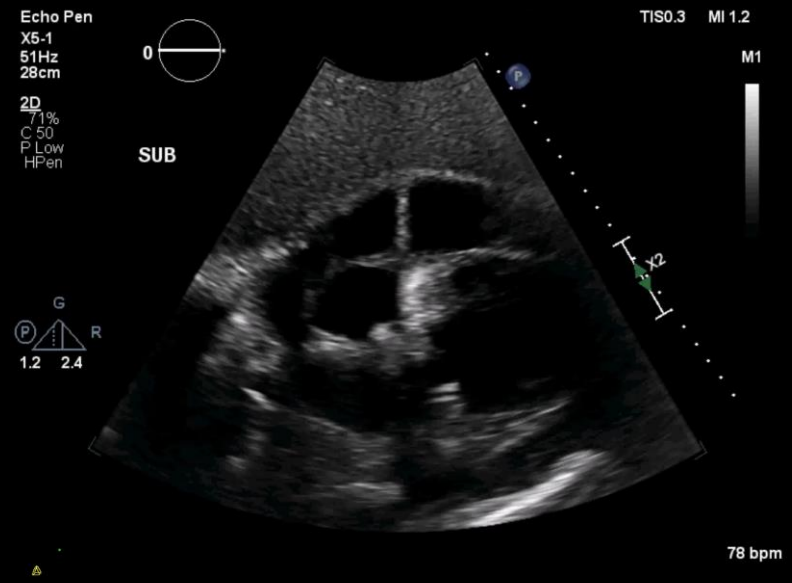
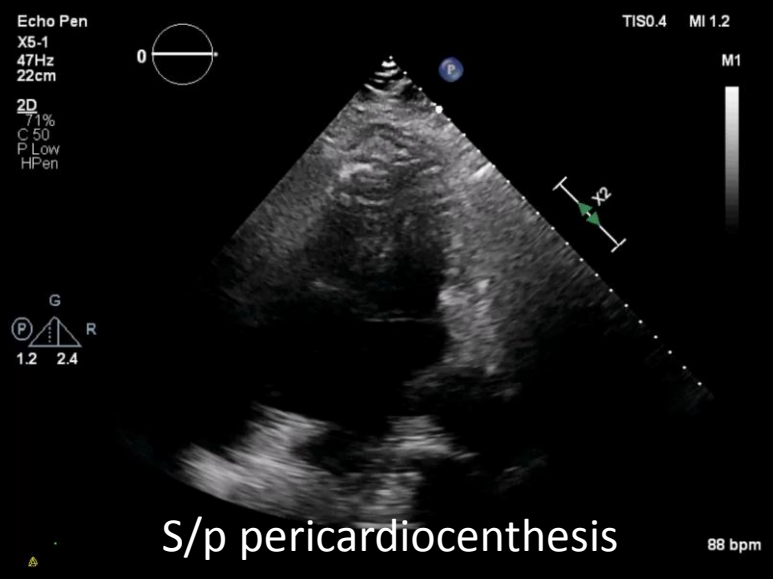
Pericardial effusion - Etiology

- **Bloody coagulum = EMERGENCY: acute MI, aortic dissection, catheter manipulation, pacemaker, cardiac surgery**



S/p RV perforation





S/p pericardiocenthesis

- **Neoplastic**
- **Inflammation (infection, autoimmune, radiation)**
- **Metabolic (hypothyroid, renal failure)**
- **CHF**

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Take home points

1. Cardiac tamponade is a clinical diagnosis and “echocardiographic features of tamponade” are not by themselves an indication for pericardiocentesis

2. Coagulum = Emergency

Thank you!

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