Case

A 65 female presents with ongoing shortness of breath and peripheral oedema. GP has trialed frusemide but has progressively developed peripheral oedema with ascites. Weight has increased by 10kg. She has a background of HTN, rheumatoid arthritis for which she is on DMARDS and has a 20-pack year smoking history. In the past she has experienced sharp chest pains. She has never been investigated for coronary artery disease. Her current exercise tolerance is 400metres.

On examination, BP is 112/80. Heart sounds dual. Crackles at base. JVP is prominent with respiration and ascites present. Fast scan in ED suggests preserved LV function.

Right heart catheterization

HAEMODYNAMICS

Baseline :

Right Heart (mmHg)

RA =	a wave = 26	v wave = 27	mean = 25	HR = 85
RV =	50/22			HR = 87
MPA =	47/27		mean = 37	HR = 85
PCW =	a wave = 30	v wave = 29	mean = 25	HR = 85

Trans Pulmonary Gradient = 12 mmHg

Baseline :

Body Surface Area 1.61 m2

Baseline :

SVR	745.58 dsc^(-5)	9.3 (WU)
PVR	116.19 dsc^(-5)	1.5 (WU)
TVR	987.65 dsc^(-5)	
TPR	358.26 dsc^(-5)	
SVRI	1201.28 dsc^(-5)*m2	
PVRI	187.21 dsc^(-5)*m2	
TVRI	1591.31 dsc^(-5)*m2	
TPRI	577.24 dsc^(-5)*m2	
TPR:TVR Ratio	0.36	
PVR:SVR Ratio	0.16	

Baseline :

Thermal Cardiac Output	8.26 L/min
Thermal Cardiac Index	5.13 L/min/m2

Question

Whilst interpreting the results, what is the next best investigation to give you a diagnosis?

- Exercise RHC
- Left heart catheterization
- Transthoracic echo
- Cardiac MRI
- High resolution CT chest

Left & Right cath performed

HAEMODYNAMICS

Baseline :

Right Heart (mmHg)

RA = a wave = 26 v wave = 27 mean = 25 HR = 85
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PCW = a wave = 30 v wave = 29 mean = 25 HR = 85
Trans Pulmonary Gradient = 12 mmHg

Left Heart (mmHg)

LV = 165/31 HR = 90
AO = 144/71 mean = 102 HR = 87

Baseline :

Body Surface Area 1.61 m2

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Baseline :

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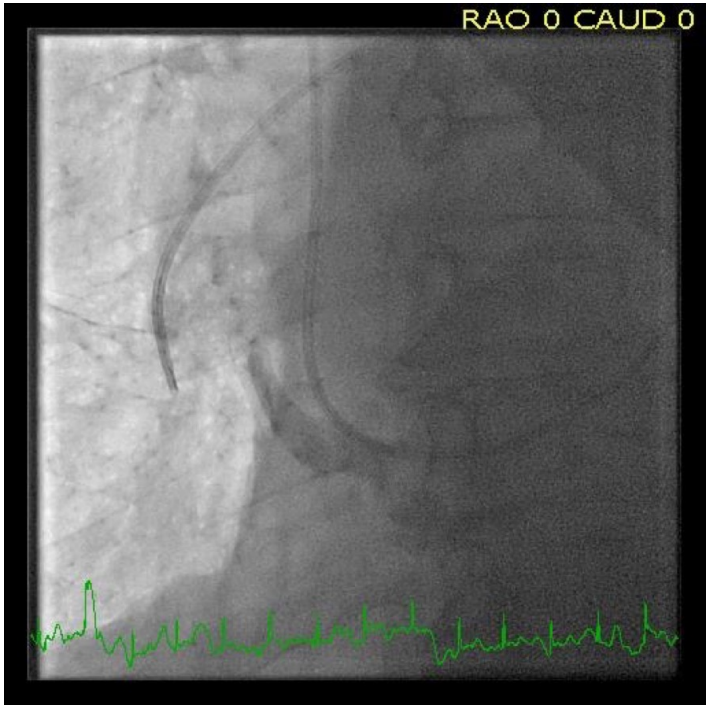
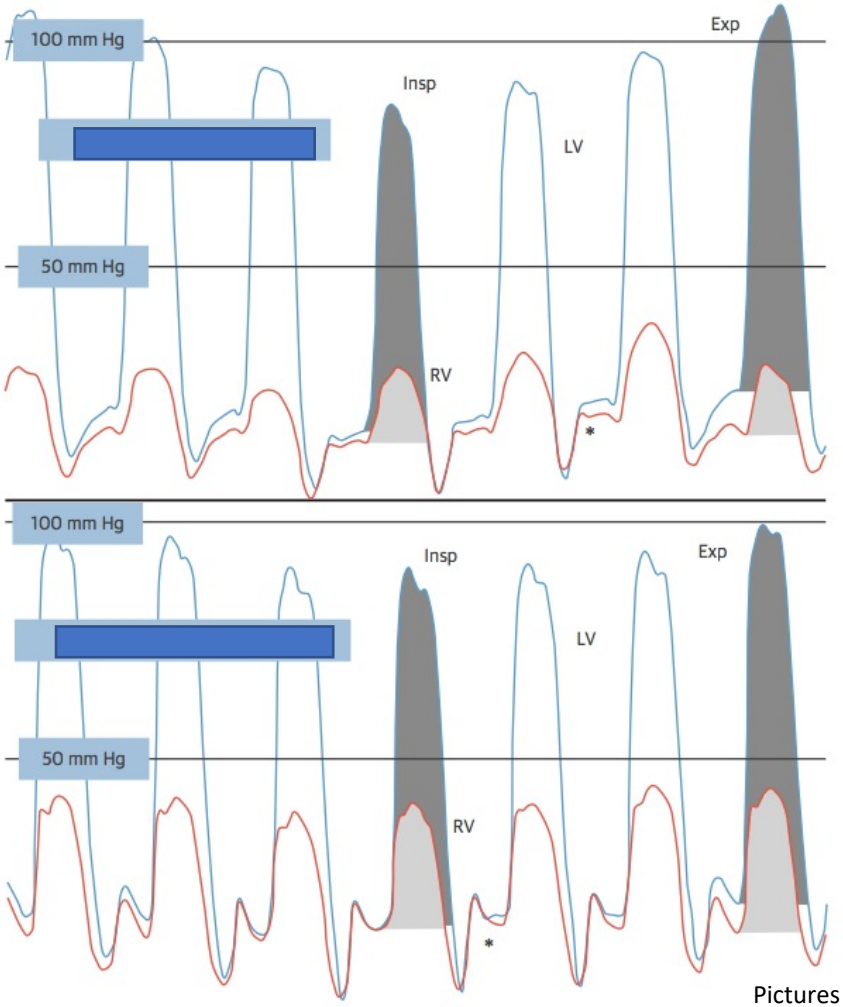


Measurements taken during respiration: What do you expect: A or B?

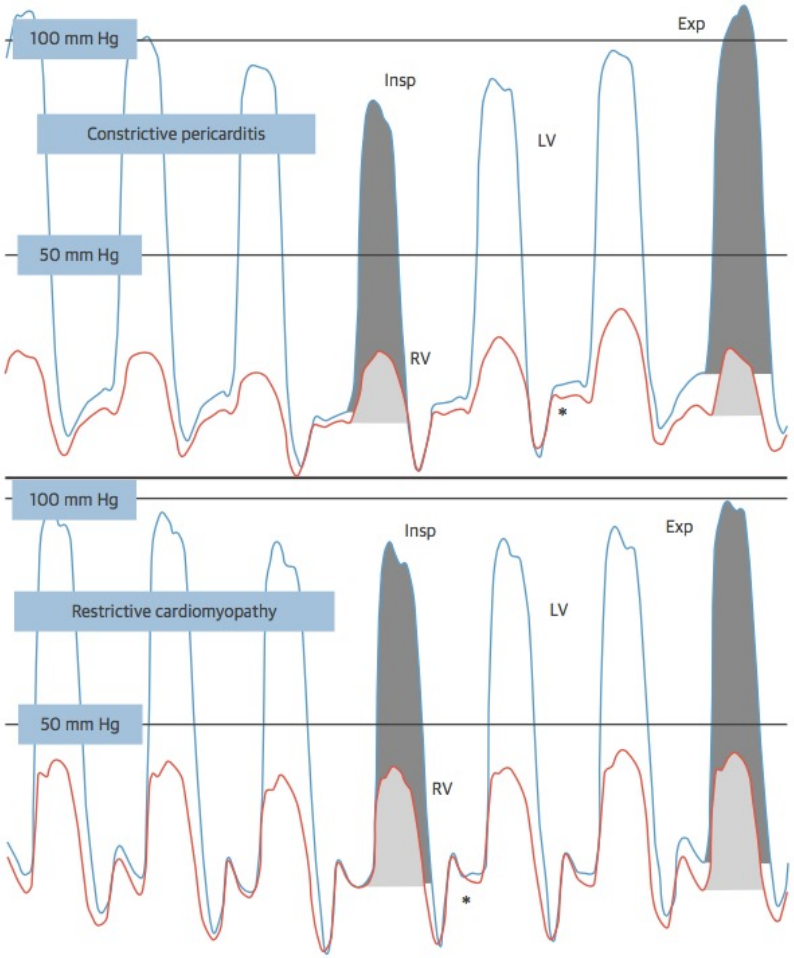
A

B



Pictures taken from : J Am Coll Cardiol 2016;68:2329–47

During Respiration: Expect to see A



Enhanced ventricular interdependence. During respiration

- **Pericardial disease = discordance in LV & RV pressures**
- **Myocardial disease = concordance in pressures.**

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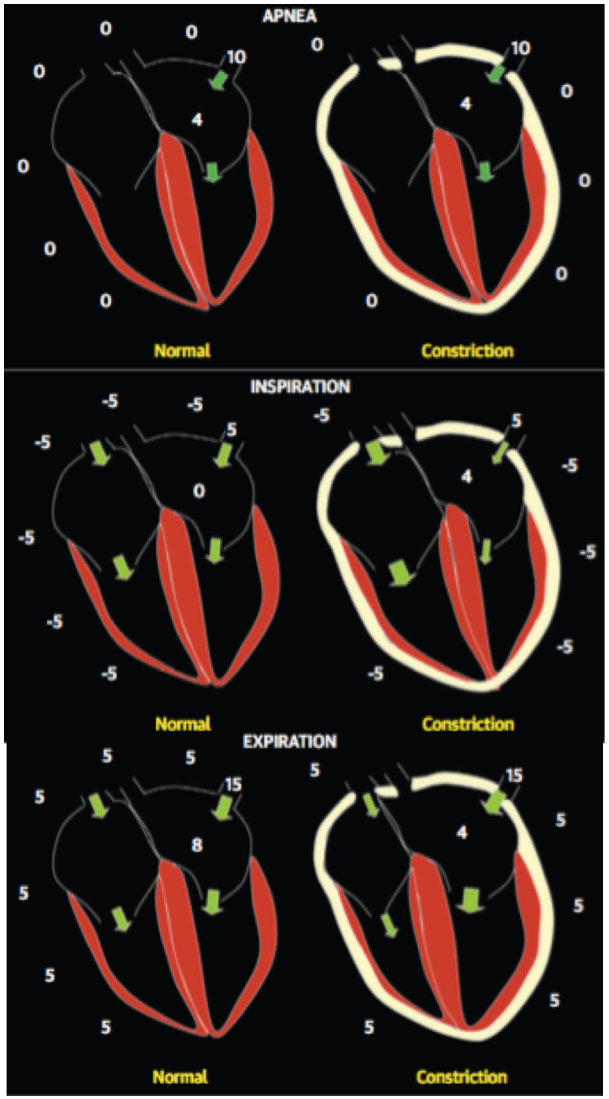
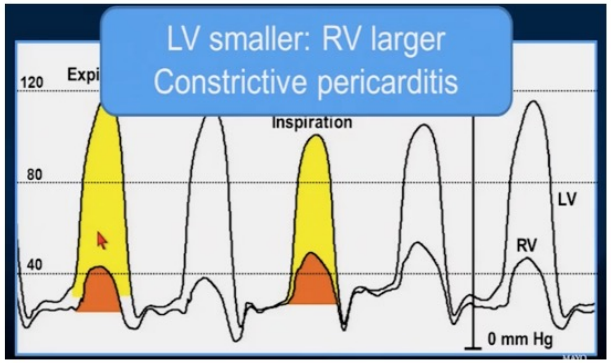
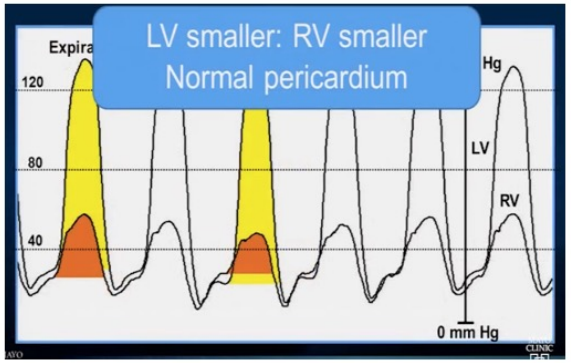
Constrictive pericarditis: Encasement Theory

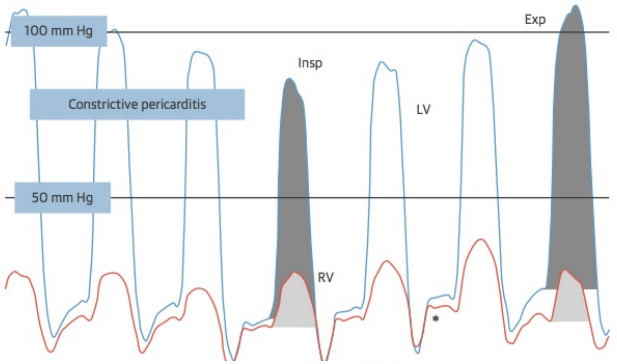


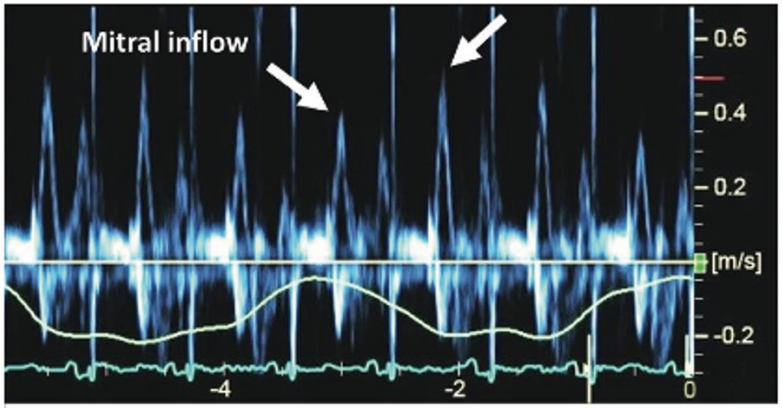
- Inspiration (Opposite true for expiration)
  - Reduced flow between PV and LA
  - Reduced intrathoracic Pi reduces PV Pi
  - As heart is encased: LA Pi does not reduce
    - Reduced driving pressure into LA
    - Reduced LV preload during inspiration.
  - **IVC is outside intrathoracic cavity**
    - Increased RV preload
- Encasement dynamics
  - Ventricular interaction is enhanced
  - LV smaller and RV larger

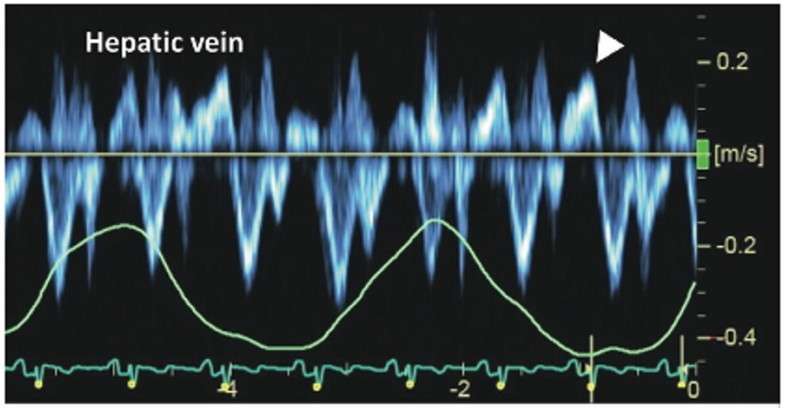


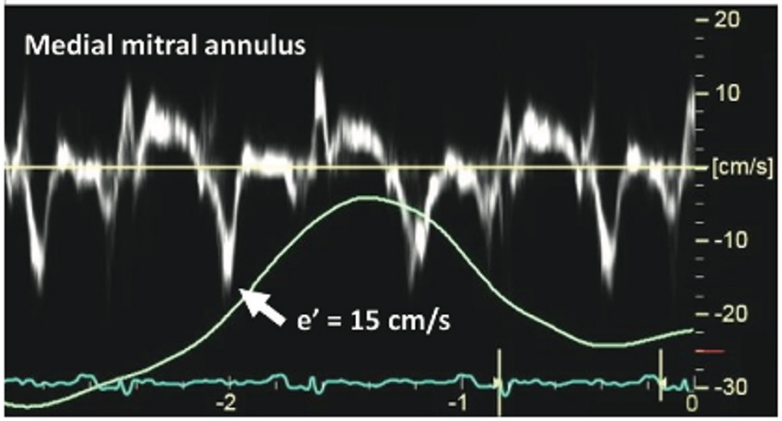


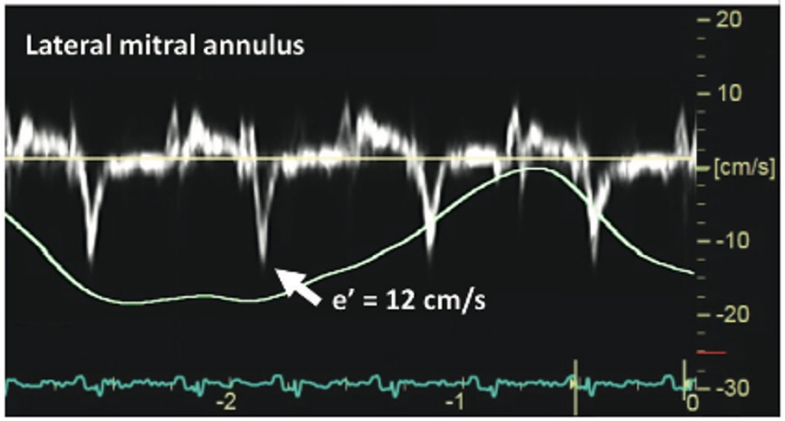
TTE: Constrictive











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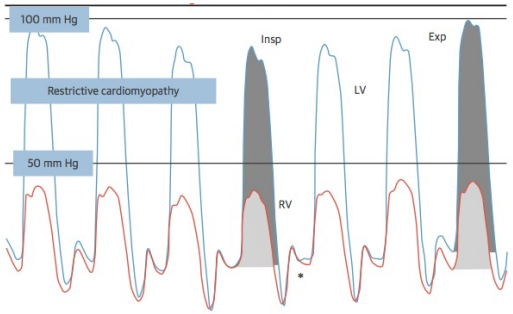
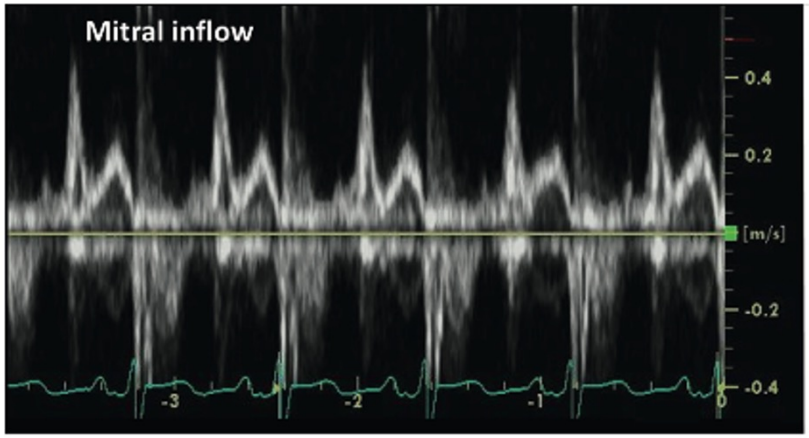
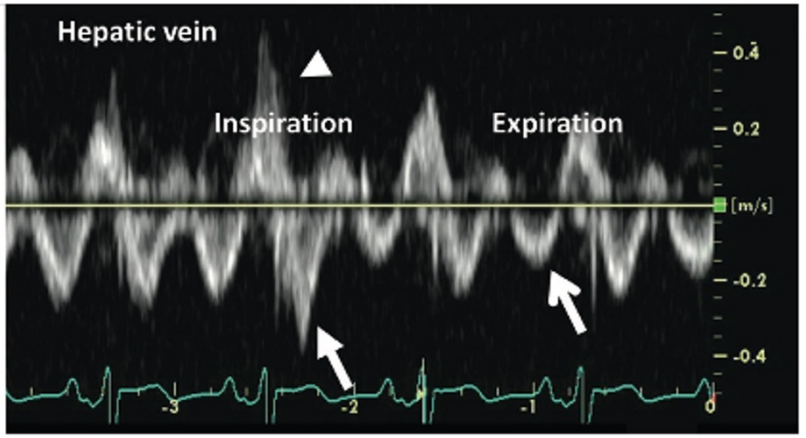
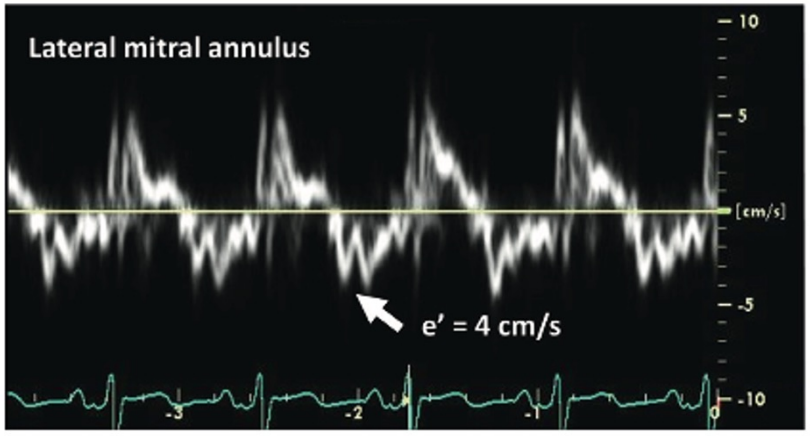
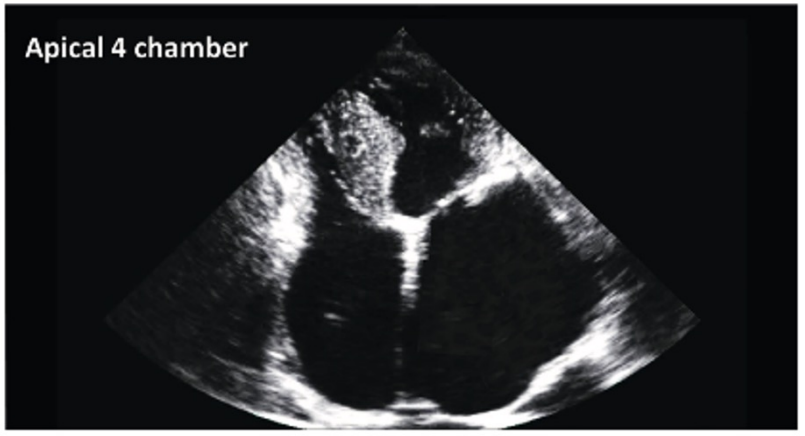
TTE: Restrictive











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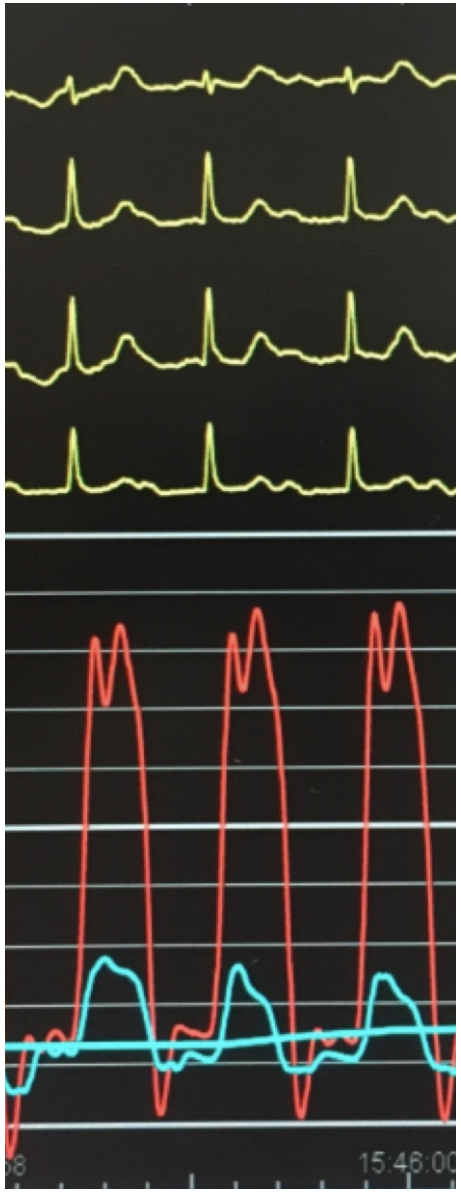


Including these pictures what have we seen?

Equalization: < 5mmHg

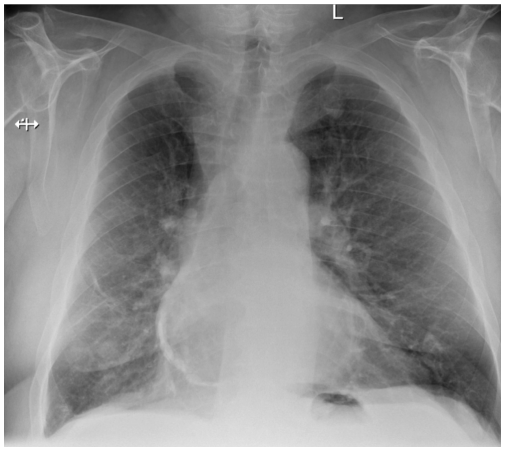
Square root sign

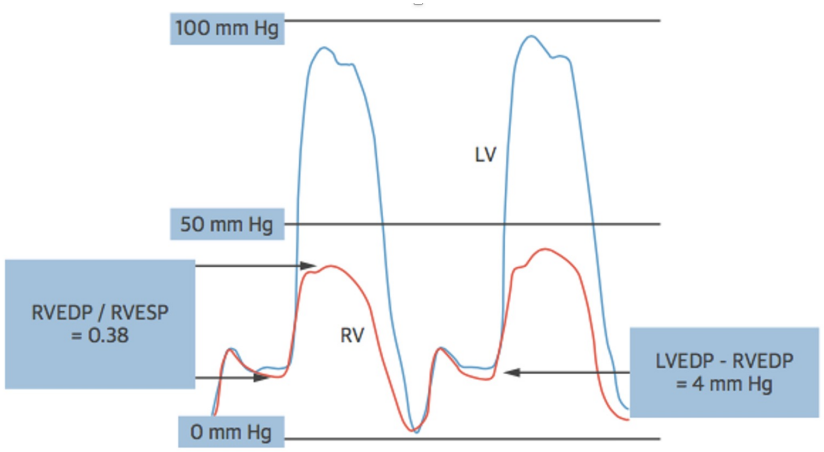
Kussmals: <5mmHg decrease in RA Pi on inspiration

Ventricular discordance

RVEDP: RVESP > 1/3

Pericardial thickening





All signs have been present

- Pericardial thickening and calcification on the CXR
- Equalisation of pressures - **< 5mmHg difference between RA, RV, LA, LV end-diastolic pressures**
- **Square root sign** - dip and plateau – can be in both
  - **Left ventricular rapid filling wave > 7mmHg favours CP**
- Increase in RV end diastolic pressure **> 1/3 RVSP**
- Respiratory haemodynamic leading to **ventricular discordance**
  - By encasing the heart, the changing pulmonary pressures are not transmitted too intra-cardiac chambers
  - inspiration - Pulmonary vein pressure falls but LA does not so LV filling reduces
  - expiration - pulmonary vein pressure increases LA does not increasing LV fill
- Kussmauls
  - <5mmHG decrease in RA on inspiration

Constrictive pericarditis

- Constrictive pericarditis is the encasement of the heart by a thickened, fibrous, and sometimes calcified pericardium.
- In the presence of preserved LV function, history of heart failure with an examination of predominantly right sided features of heart failure, may make the astute clinician think of this diagnosis.
- Right heart catheterization and knowledge of hemodynamic principles is fundamental to its diagnosis.
- Unlike restrictive cardiomyopathy, constrictive pericarditis is potentially reversible.