Pericardial Disease
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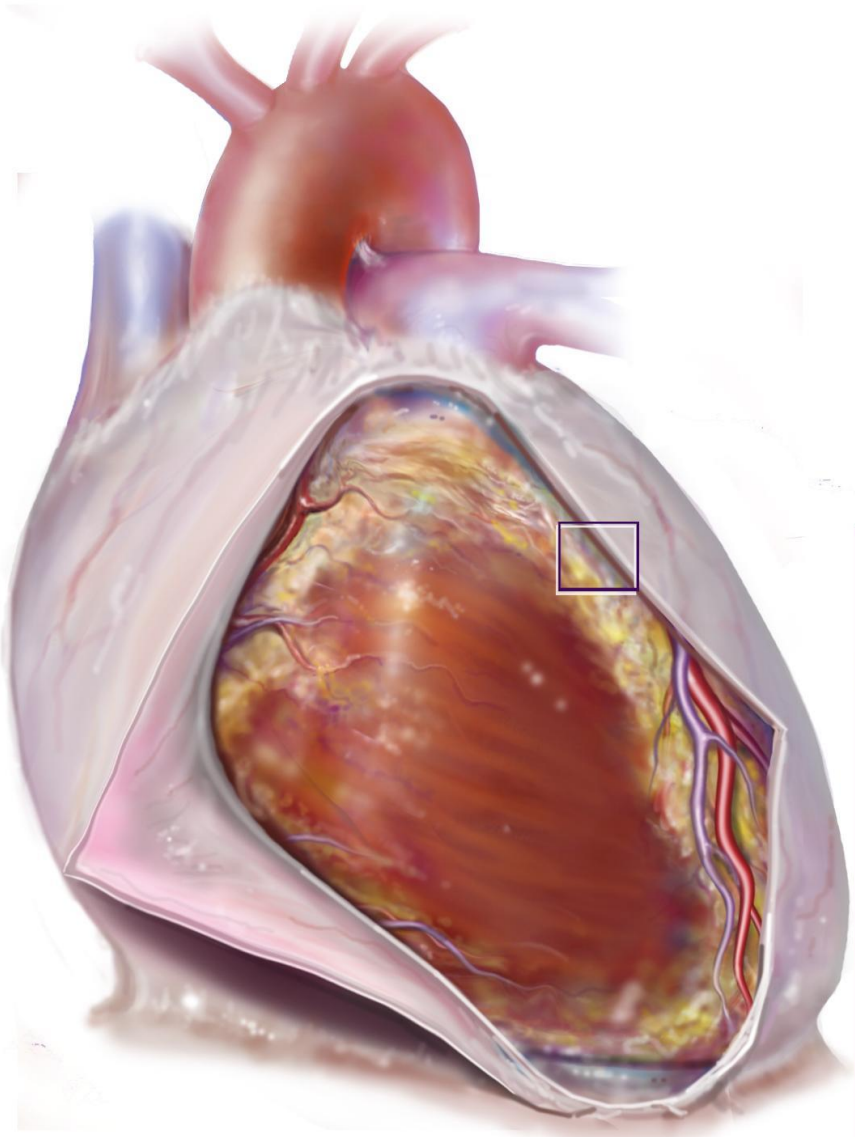
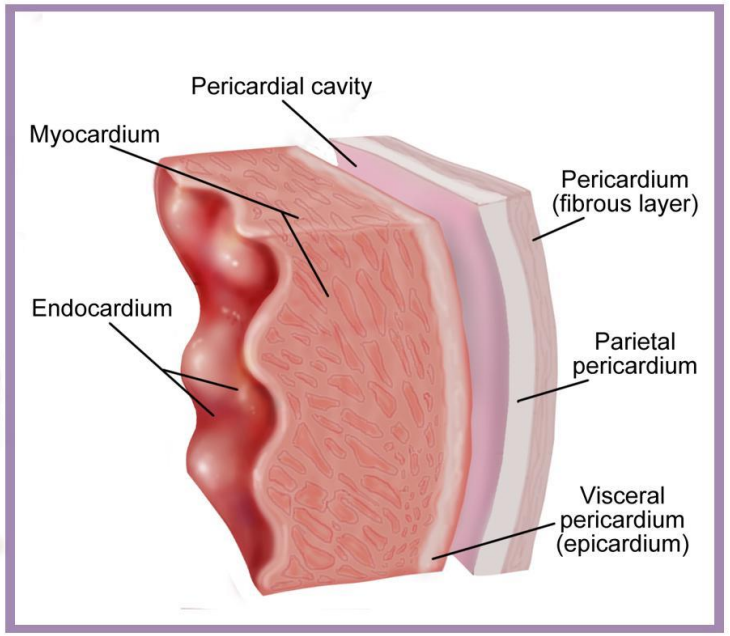
PGY-4

Structures of the Pericardium





Basics

- In healthy individuals, the pericardial cavity contains 15 to 50 mL of an ultrafiltrate of plasma
- Disease of the pericardium can be isolated or due to an underlying systemic disorder.
- Disease can manifest as fluid accumulation, fibrosis/scarring and/or calcification.
- At baseline, the pericardium behaves like rubber, however if it is stretched beyond its normal threshold it can quickly become stiff and non compliant.

Etiologies of Pericardial disease

- **Infectious**
  - **Viral**, echovirus, coxsackie, also HIV
  - Bacterial and fungal, usually with purulent effusion
  - Others (Rickettsia, Chlamydia, Borrelia, Mycoplasma, Treponema, Ureaplasma, Nocardia, Tropheryma)
- **Radiation**
- **Post cardiac injury syndrome**
  - Post-myocardial infarction
  - Post-pericardiotomy
  - Post-traumatic (including iatrogenic)
- Drugs and toxins
- Metabolic (uremia, dialysis-associated, myxedema, ovarian hyperstimulation syndrome)
- Malignancy (especially lung and breast cancer, Hodgkin lymphoma, and mesothelioma), usually have sanguinous effusion
- **Rheumatologic** (SLE, rheumatoid, scleroderma)
- Collagen vascular disease
- Idiopathic

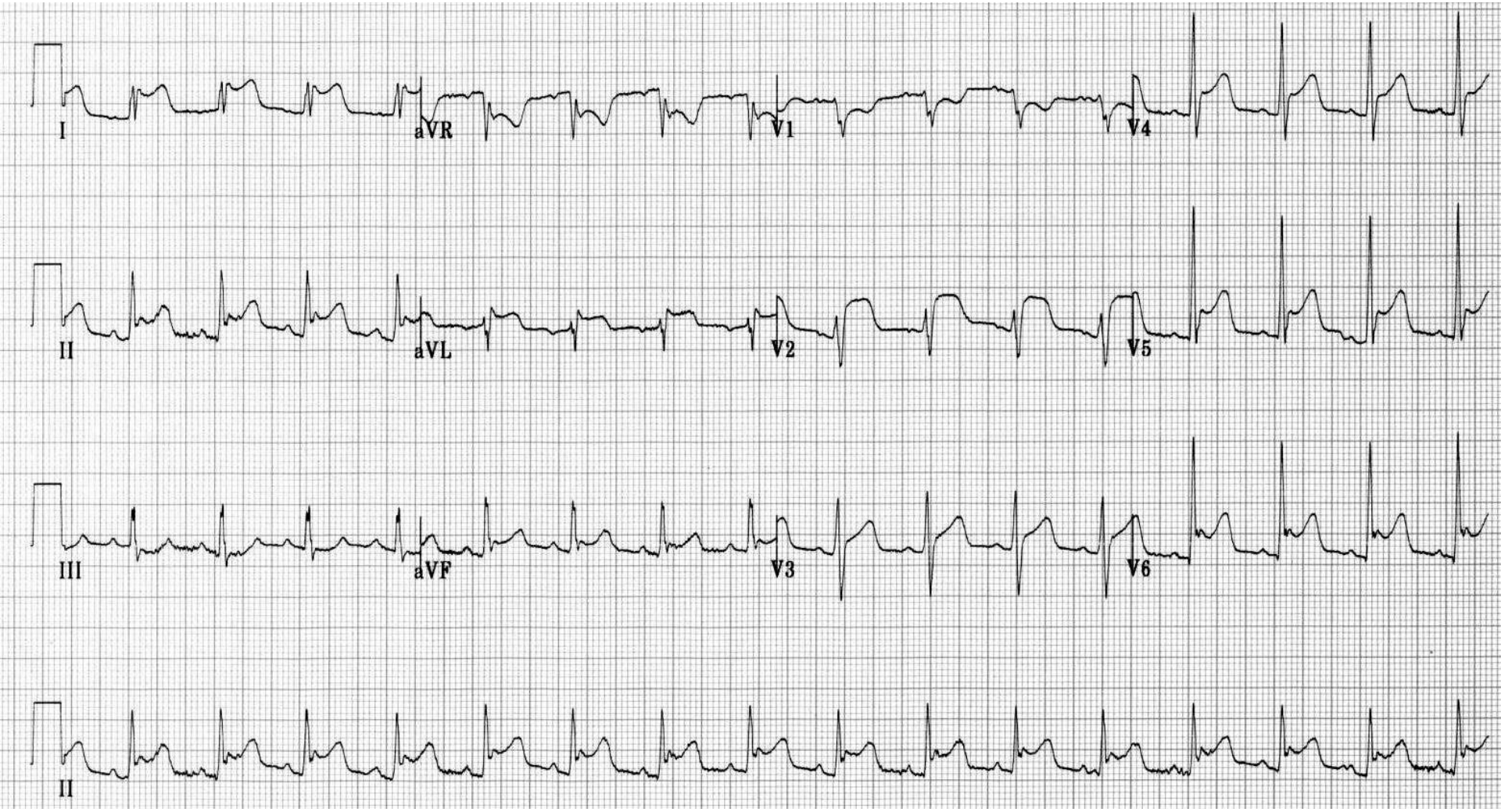
A 63-year-old man is evaluated for pleuritic left-sided anterior chest pain, which has persisted intermittently for 1 week. The pain lasts for hours at a time and is not provoked by exertion or relieved by rest but is worse when supine. He reports transient relief with acetaminophen and codeine and occasionally when leaning forward. He has had a low-grade fever for 3 days, without cough or chills. Medical history is significant for hypertension and tobacco use. His only current medications are acetaminophen and codeine.

On physical examination, temperature is 37.8 ° C (100.0 ° F), blood pressure is 132/78 mm Hg, pulse rate is 98/min, and respiration rate is 16/min. No jugular venous distention is noted. Lung auscultation reveals normal breath sounds with no wheezing. No pedal edema is present.

Electrocardiogram is shown on next page.

Which of the following is the most appropriate management?

A. Azathioprine
B. Chest CT scan
C. Aspirin and colchicine
D. Pericardiectomy
E. Prednisone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Pericarditis

- Presentation:
  - Pleuritic chest pain, dyspnea, +/- fever, chills
  - Post MI can have acute (1-7 days) or late (1-2 weeks to months – Dressler) pericarditis
- Physical exam:
  - Chest pain improved by leaning forward, ***not with nitrates***
  - Pericardial friction rub possible
  - EKG with characteristic diffuse ST Elev, PR depressions
- Labs:
  - Elevated WBC, ESR, CRP, sometimes can have elevated troponin (myocarditis)
- Imaging:
  - CXR usually normal but could have enlarged cardiac silhouette if they have pericardial effusion.
  - Echo: may show thickened pericardium, effusion, but can also be normal
- Differential:
  - Be sure to consider other possible causes of chest pain such as ischemia, GERD, PE, Pneumonia, etc.

Pericarditis

- 4 stages of EKG changes
  - Stage I: Diffuse, concave ST-segment elevation
  - Stage II: ST segments normalize, J point returns to baseline, T-wave amplitude begins to decrease, PR-segment depression begins to appear
  - Stage III: Symmetric, diffuse T-wave inversions
  - Stage IV: Changes normalize or T-wave inversions may become permanent
- Pericardial friction rub
  - It consists of three components corresponding to ventricular systole, early diastolic filling, and atrial contraction and has been likened to the sound made when walking on crunchy snow.

Pericarditis

- Identifying etiology:
  - Most common is idiopathic or viral (coxsackie, echovirus)
  - Most common confirmed diagnoses are
    - Malignancy, TB or autoimmune
  - Post Myocardial infarction, i.e. Dressler
  - Other less common causes usually are rheumatologic (e.g. SLE, Rheumatoid, scleroderma)
- **Diagnosis should be suspected if at least 2 of the following:**
  - Pleuritic chest pain, pericardial friction rub, suggestive EKG, or new/worsening pericardial effusion
- Most cases are low risk, but up to 15-20% can be high risk.
  - High fevers, Pericardial effusion with tamponade, myocarditis, constriction
- Low risk patients can usually be treated as outpatients while high risk patients need further workup and should be admitted.

Pericarditis

- Treatment:
  - Idiopathic/viral: High dose NSAIDs are recommended as 1st line therapy for acute and even recurrent pericarditis. Colchicine is also highly recommended as an adjunct therapy
    - Common NSAIDs are aspirin, ibuprofen or indomethacin
  - Post MI pericarditis therapy is preferentially high dose aspirin with taper.
  - High dose steroids not routinely recommended as they increase risk of recurrence. If absolutely needed, would use low dose with as rapid taper as able. (0.2-0.5mg/kg/day)
  - Can consider monitoring CRP during therapy to guide length of tx.
  - For pericarditis due to co-existing disease, need to treat the underlying condition (i.e. TB, autoimmune, connective tissue disease).
  - Drainage of pericardial effusion if echo findings of hemodynamic compromise.

Pericarditis

- Between 15-30% of pts can have recurrent pericarditis after successful initial therapy.
- Recurrence should be treated the same as initial episode (NSAID+colchicine) but should also raise concern for underlying disorder, further testing is clinically indicated.
- Can consider steroids for continued recurrence or if co-existing rheumatologic disorder

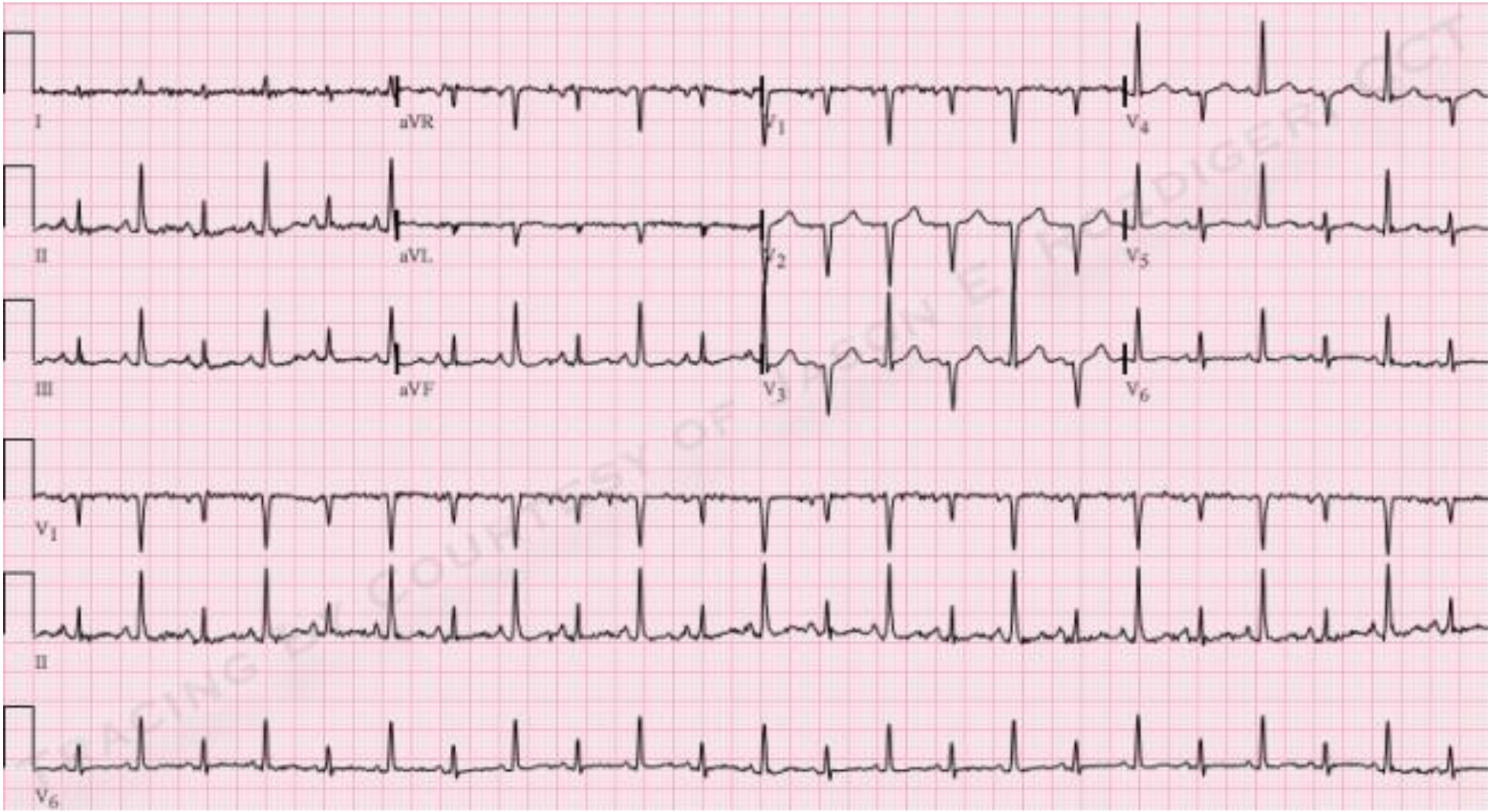
65 year old male with 2 week history of worsening shortness of breath. Initially just with exertion but symptoms are now present at rest. He denies chest pain, palpitations, orthopnea or diaphoresis. Past medical hx only notable for hypertension.

On physical exam, BP is 90/50, P is 120, RR 20 at rest, O2 sat is 93% on RA.

He appears to be in mild distress 2/2 resp. difficulty, lungs are clear to auscultation, RRR with no murmurs but heart sounds are soft, there is JVD to the mandible. There is no peripheral edema.

Electocardiogram is shown on the next page.

- What is the most appropriate 1st step in management?
  A. CT scan of the chest
  B. 80mg IV lasix
  C. SVNs
  D. IV fluids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Pericardial effusion and Tamponade

- Tamponade: Effusion causing increased intrapericardial pressure which inhibits normal diastolic filling and cardiac output.
- Presentation:
  - Asymptomatic ➔dyspneic, chest pain if co-existing pericarditis
- Physical exam (Beck's triad)
  - Tachycardia, Distant heart sounds
  - Jvd
  - Hypotension
  - Pulsus paradoxus may or may not be present
- Imaging
  - Enlarged cardiac silhouette on CXR, effusion on CT scan
  - Echo may show evidence of RV collapse in diastole which indicates tamponade physiology
- Labs
  - Depends on what is causing the effusion, could be normal or reflect underlying inflammatory process (e.g. co-existing pericarditis)

Pericardial Effusion and Tamponade

- Bacterial, Tuberculous and malignant or traumatic effusions more likely to cause tamponade compared to viral etiologies.
  - Consider urgent drainage for these
- Malignancy can cause sanguinous effusions if tumor is involving the pericardium
  - If drained, effusion can recur, window and intrapericardial chemo may be necessary
- Trauma to the pericardium/myocardium can result in rapid accumulation of blood (e.g. MVC, penetrating trauma, post PCI/PPM), **surgical emergency.**

Pericardial effusion and Tamponade

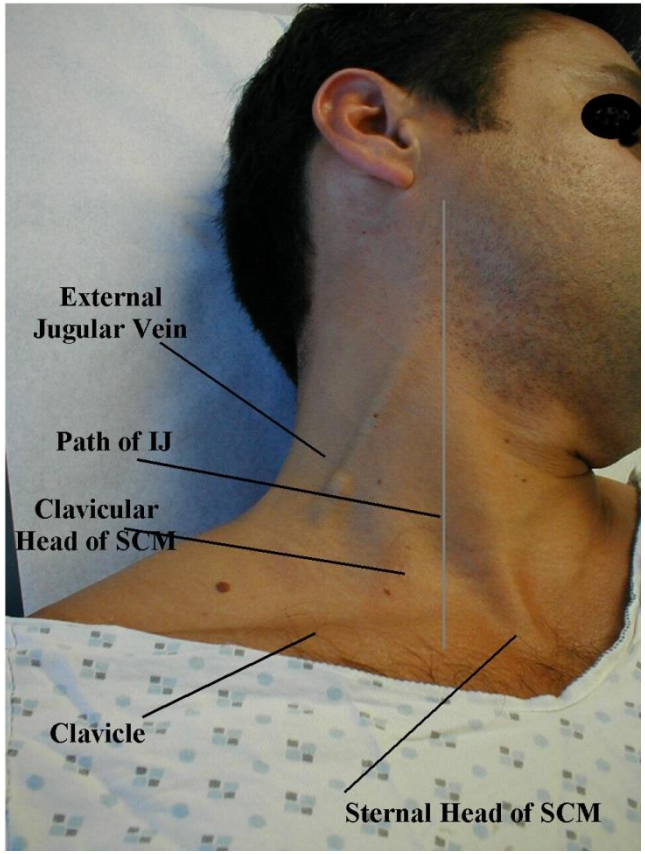
- Rate at which fluid accumulates is important
  - The slower the accumulation, the less likely for tamponade physiology to develop
  - The pericardial sac will slowly enlarge to accommodate extra fluid and pressure will build slowly allowing the myocardium time to adjust.
    - Subacute presentations like this may only have peripheral **edema** as presenting symptoms.
  - Rapid accumulation, even small amounts of fluid, almost always will cause tamponade
- **Diuretics can actually worsen tamponade!**

Pericardial effusion→Tamponade

- JVD
  - Need to examine the right

  internal jugular vein if able. R EJ ok if unable to see IJ.

  - Patient should be supine with HOB Elevated at about 30-45 degrees.
  - Elevated JVD occurs in tamponade because of pressure overload in the intrapericardial space.
  - Intrapericardial pressure becomes equal or greater than ventricular filling pressures which impedes venous return
  - This is why diuretics will worsen tamponade, decreasing CVP allows intrapericardial pressure to dominate.

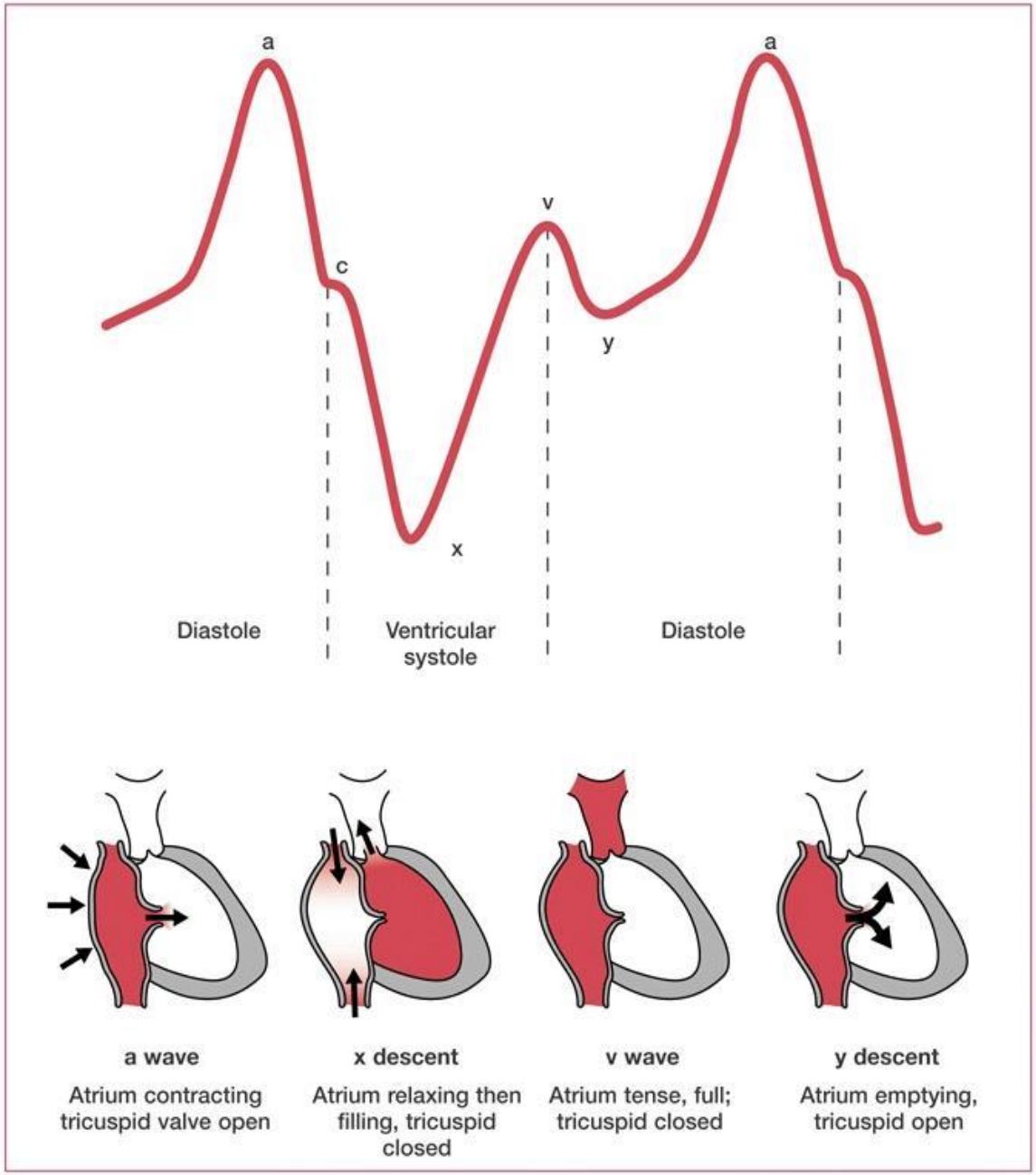


JVP waveform

- Remember that it is directly related to right atrial pressure
- Can be difficult to appreciate in standard patients but can become prominent when right sided pressures increase.
- Easiest way to observe is with invasive monitoring, i.e. RHC.

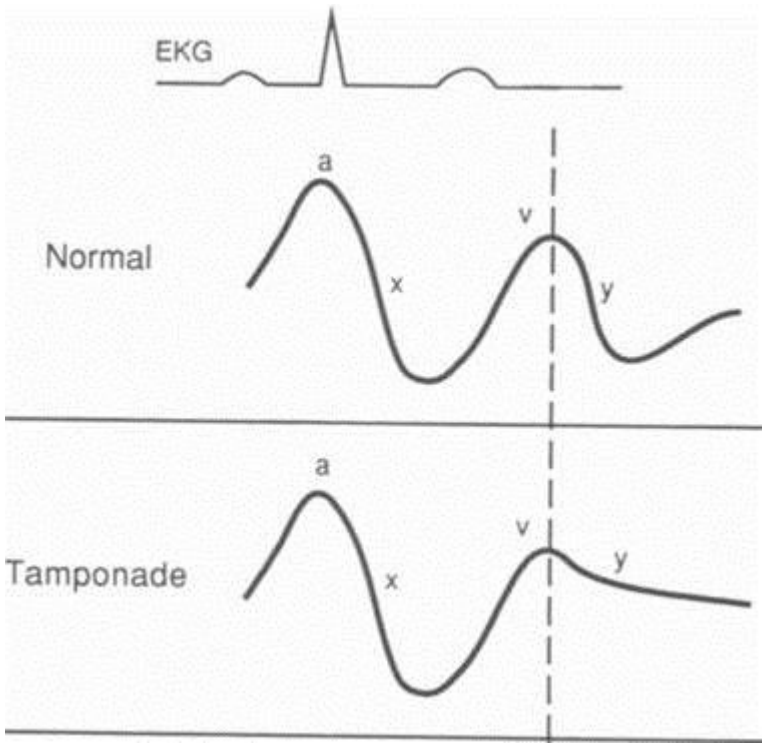
FEATURE	INTERNAL JUGULAR VEIN PULSE	CAROTID ARTERY PULSE
Appearance of pulse	Undulating two troughs and two peaks for every cardiac cycle (biphasic)	Single brisk upstroke (monophasic)
Response to inspiration	Height of column falls and troughs become more prominent	No respiratory change to contour
Palpability	Generally not palpable (except in severe TR)	Palpable
Effect of pressure	Can be obliterated with gentle pressure at base of vein/clavicle	Cannot be obliterated

JVP waveform



-In tamponade, total heart volume is fixed, thus blood can only enter a chamber if blood is leaving downstream.

- During x descent, blood is beginning to leave RV during systole so waveform preserved.
- During y descent, blood is not yet leaving RV and diastolic filling is impaired due to high intrapericardial pressure.



Pericardial effusion→Tamponade

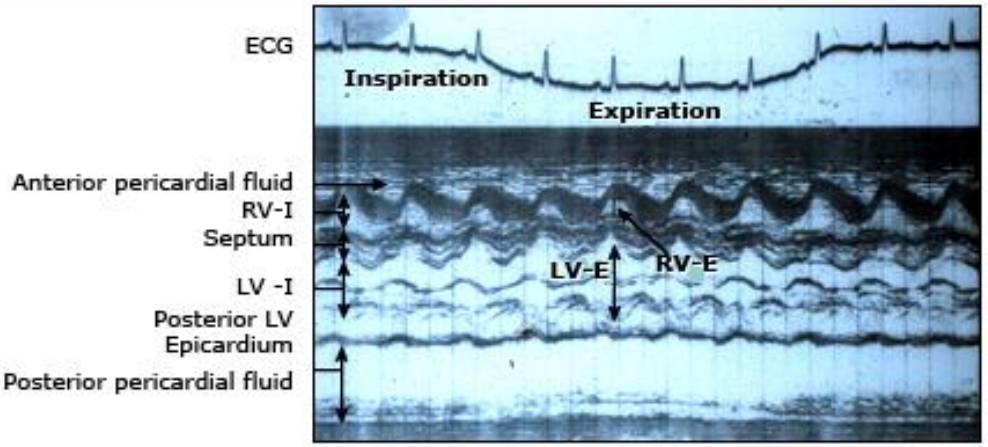
- Pulsus paradoxus –

Sometimes may be seen in tamponade.

– Drop in systolic (>10mmHg) BP with Inspiration.

- Occurs because of impaired cardiac output

M-mode echocardiogram in cardiac tamponade



M-mode echocardiogram showing respiratory motion of the interventricular septum in cardiac tamponade such that during inspiration, the right ventricle enlarges, and the left ventricle becomes smaller.

RV-I: right ventricle in inspiration; RV-E: right ventricle in expiration; LV-I: left ventricle in inspiration; LV-E: left ventricle in expiration.

Reproduced by permission of the American Heart Association from Settle, et al. Circulation 1977; 56:951.

Pulsus Paradoxus

How to check?

- Inflate cuff as normal, obliterate radial pulse
- Listen for Korotkoff sounds (during expiration) while slowly deflating cuff. Record pressure at which you first hear them.
- Continue deflating cuff slowly until you hear sounds throughout respiratory cycle, this is the lower pressure
- If the difference value is > 10 then there is a paradoxical pulse

Pulsus Parodoxus

- Can be found in other disease states, usually those that affect pulmonary system.
  - Bronchitis/COPD
  - Pneumothorax
  - Pulmonary embolism
- These all require more than usual inspiratory effort which may be enough to drop intrathoracic pressure to the point that it prevents normal pulmonary vein flow.

Pericardial Effusion

Cardiac tamponade can occur without the development of pulsus paradoxus in the following circumstances :

- Coexisting disease that significantly elevates left ventricular diastolic pressure (eg, heart failure, aortic stenosis) or right ventricular diastolic pressure (eg, pulmonary hypertension with cor pulmonale)

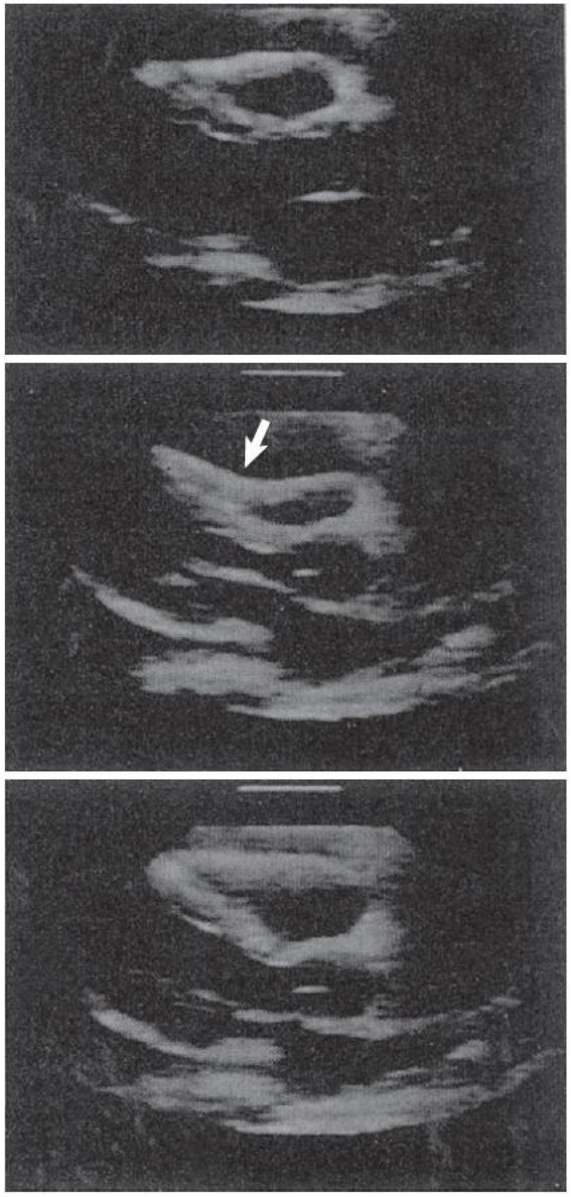
- An intracardiac shunt or significant valvular regurgitation (eg, aortic regurgitation)

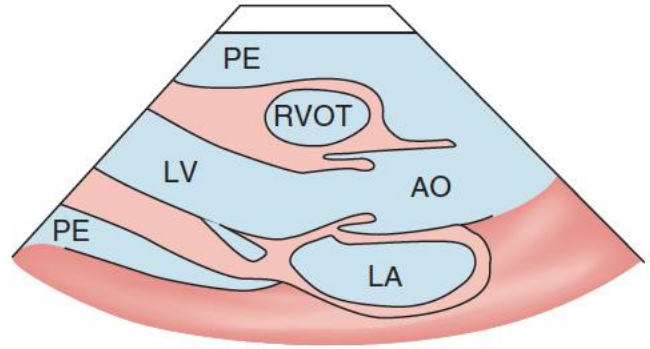
- Aortic dissection with resulting pericardial effusion and cardiac tamponade

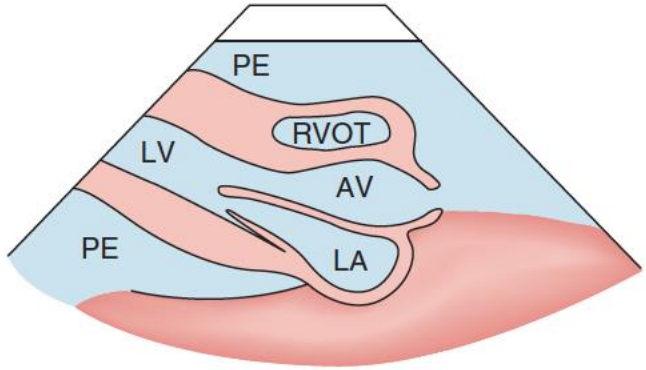
- "Low pressure" tamponade, as in the presence of dehydration and hypovolemia, where a pericardial effusion that would not otherwise cause cardiac compression can affect cardiac function

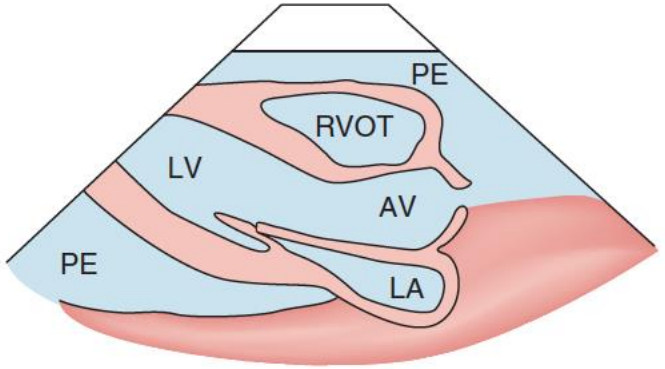
- Positive pressure ventilation

- Echo findings raising suspicion of tamponade
  - Collapse of RV free wall during diastole
  - Variation of flow with inspiration/expiration









Pericardial effusion→Tamponade

- Everyone should have echocardiography if clinical suspicion is high.
- If physical exam and imaging is suggestive of tamponade, patient should be started on IVF and urgently referred for pericardiocentesis or pericardial window. ***Avoid diuretics!***
- If clinically indicated, effusion should be sent for pathology/culture.
- If pt stable and asymptomatic, effusion can be followed with frequent echocardiograms to evaluate for interval change.
  - Can also try to empirically treat for pericarditis, occasionally will be subacute case causing small effusion.

66 year old man is admitted to the hospital with a 2 month history of progressive dyspnea and worsening pedal edema. Medical history is significant for coronary artery bypass surgery 3 years ago. He has a 10 year history of hypertension. Medications are metoprolol, atorvastatin, and aspirin.

On physical exam, temperature is normal, blood pressure is 118/64 mmHg, pulse rate is 120/min, and respiration rate is 26/min. Jugular venous distention that increases with inspiration is noted. Cardiac exam reveals no murmur, rub or gallop. Pulsus paradoxus of 5mmHg is present. Lungs are clear. Hepatojugular reflex is present. Pedal edema of 2+ is noted.

Electrocardiogram shows sinus rhythm with increased voltage in precordial leads. Echo shows LVEF of 70%, restrictive LV filling, increase ventricular thickness, biatrial enlargement, a small pericardial effusion, and abnormal diastolic to-and-fro ventricular septal motion consistent with ventricular interdependence. Chest radiograph is shown on following page.

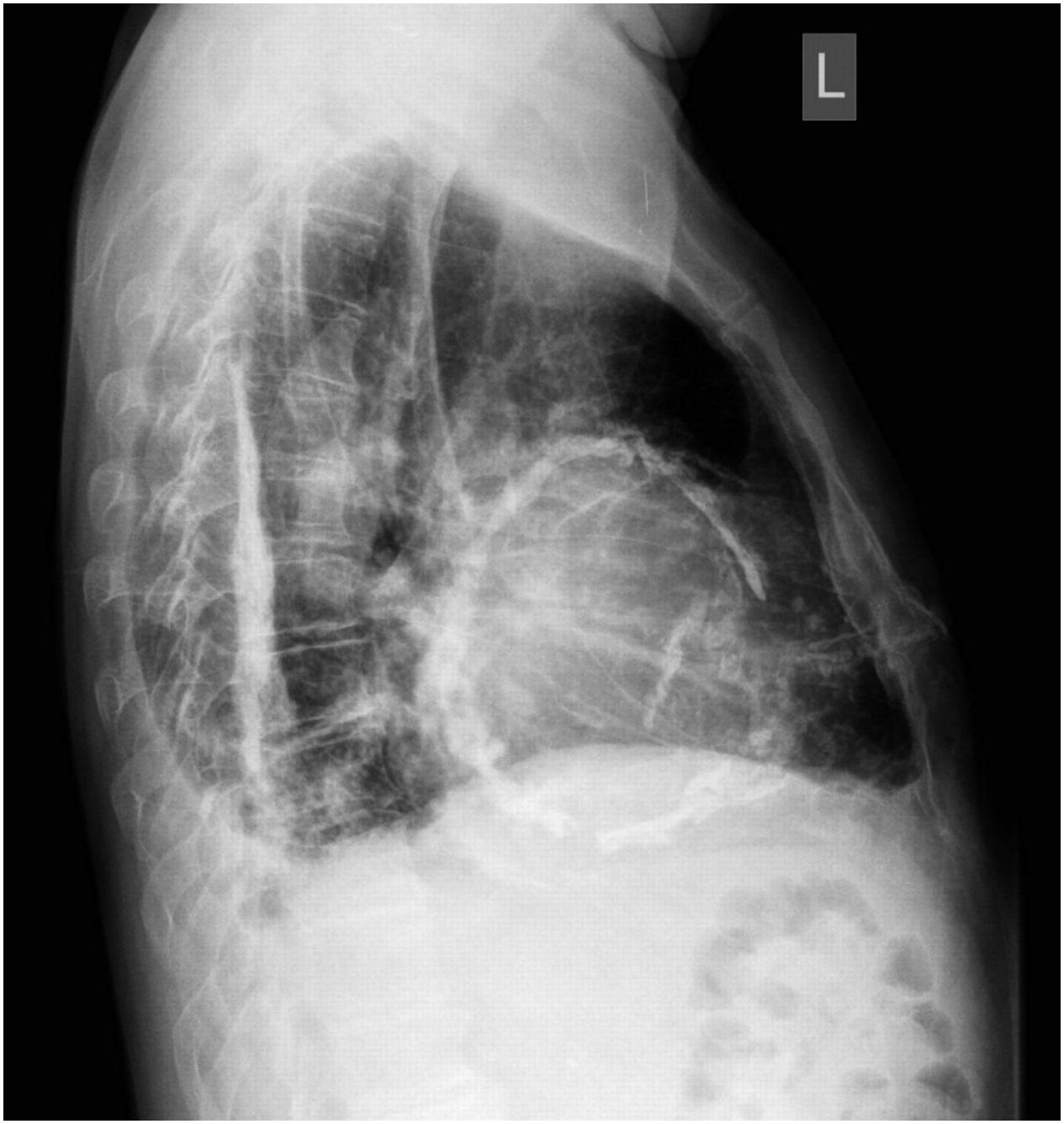
Which is the most likely diagnosis?

A.Cardiac amyloidosis

B.Cardiac tamponade

C.Constrictive pericarditis

D.Severe tricuspid regurgitation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Constrictive Pericarditis

- Decreased elasticity/compliance of pericardium due to thickening, fibrosis, and/or calcification.
- Results in equalization of chamber pressures, rapid early diastolic filling, end diastolic dysfunction, and "**ventricular interdependence**."
- Presentation:
  - Dyspnea, chest tightness, peripheral edema, ascites
- Labs
  - Possibly elevated BNP, troponin, inflammatory markers but can also be normal
- Imaging
  - Can show pericardial thickening, enlarged cardiac silhouette or could be normal.

Constrictive Pericarditis

- Idiopathic or viral – 42 to 61 percent
- Post-cardiac surgery – 11 to 37 percent
- Post-radiation therapy – 2 to 31 percent, primarily after Hodgkin disease or breast cancer
- Connective tissue disorder – 3 to 7 percent
- Postinfectious (tuberculous or purulent pericarditis) – 3 to 15 percent
- Miscellaneous causes (malignancy, trauma, drug-induced, asbestosis, sarcoidosis, uremic pericarditis) – 1 to 10 percent

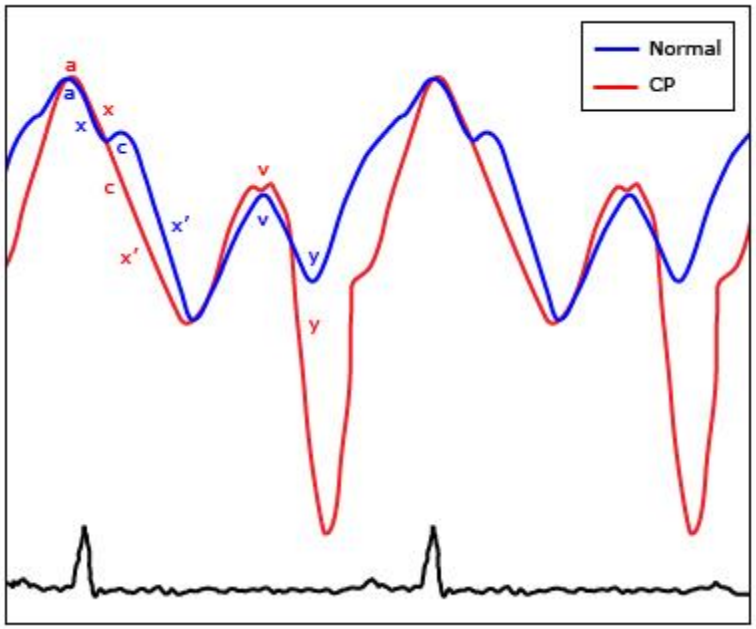
Constrictive Pericarditis

- Physical exam:
  - JVD that worsens with inspiration (**Kussmaul's sign**)
  - Friction rub
  - Pulsus paradoxus may be present
  - Pericardial "knock", early diastolic sound caused by abrupt halt in diastolic ventricular filling
  - Can also have systolic murmur of TR

	TAMPONADE	CONSTRICTION
Paradoxical pulse	Usually present	Present in ≈ ⅓
Equal left/right-sided filling pressure	Present	Present
Systemic venous wave morphology	Absent *y* descent	Prominent *y* descent (M or W shape)
Inspiratory change in systemic venous pressure	Decrease (normal)	Increase or no change (Kussmaul sign)
"Square root" sign in ventricular pressure	Absent	Present

JVP

- In constriction, JVP will show a “steep x and y descent”
- This is caused by the the rapid early diastolic filling seen in constriction.
- After LV systole, a small amount of space is created in pericardial cavity, however after rapid early filling, the myocardium slams into the non compliant pericardial sack and filling abruptly stops.
- This results is steep x and y descents



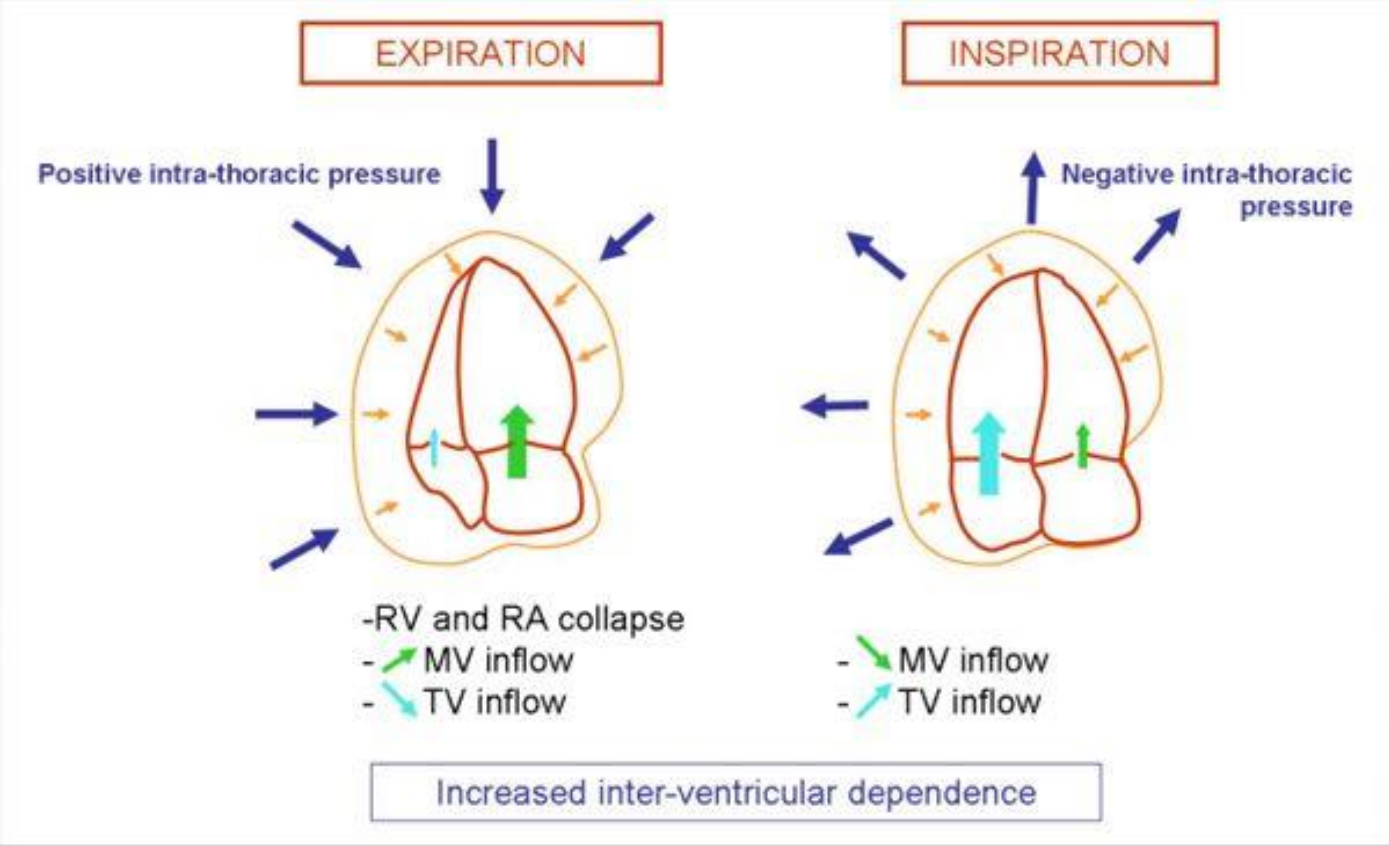
Constrictive Pericarditis

- Echo imaging findings raising suspicion for constriction
  - Septal bounce- abrupt displacement of the IV septum during early diastole due to high filling pressures.
  - Very exaggerated septal shift and flow variation with respiratory cycle
    - Ventricular interdependence

Ventricular Interdependence

- Relationship between LV and RV is reciprocal with regards to filling.
- Pericardium is rigid and fixed and does not expand with inspiration.
- Limited space is available for the heart to fill.
- If RV is filling, septum is displaced to the left, constricting the LV filling and vice versa
  - This relationship can be seen in tamponade as well but is usually less pronounced.

Ventricular Interdepence



Constrictive Pericarditis

- Usually requires invasive hemodynamic measurements for diagnosis if imaging studies are inconclusive.
- Treatment is usually surgical with pericardiectomy.
- Coronary angiography should be performed prior to surgery to evaluate anatomy
- Sometimes an epicardial vessel can be adherent or pinched to/by the fibrotic/stiff pericardium

Effusive-Constrictive Pericarditis

- Rare
- An inflammatory effusion typically dominates early, with constriction being prominent later
- Usually not discovered until after effusion is drained and patient remains symptomatic
- Treatment usually dependent on the cause of inflammation, pericardiectomy may become necessary in refractory cases.

Questions?