Neoplasm (primary rare with mets 30x more common)

- Benign: Atrial Myxoma, Lipoma, Fibroelastoma, Rhabdomyoma, Fibroma, Hemangioma
- Malignant: Sarcoma, Angiosarcoma, Rhabdomyosarcoma, Osteosarcoma
- Mets: direct from lung/breast and venous extension from renal/lung/adrenals
- NB when you see a mass in the heart think of thrombus, vegetation, and neoplasm

### Endocarditis (refer)

## Myocarditis

- Etiology
  - Idiopathic (most common) 0
  - Infectious (similar to pericarditis) + Trypanosomi cruzi aka Chagas' Disease 0
  - 0 **Heavy Metals**
  - Trauma (radiation, physical, etc) 0
  - Stings/Bites 0
  - Drugs (anthracyclines, ethanol, cocaine, etc)
  - Autoimmune Dz (SLE, sarcoidosis, scleroderma, etc)
- S/S
- Pts sometime have a recent flu-like illness and then variable cardiac Sx from asymptomatic pts w/ ECG changes (arrhythmias, heart block, mimic ischemic changes) to mimicking ACS to chronic systolic CHF 2/2 dilated CM from muscle cell apoptosis to fulminant CHF to sudden cardiac death
- EKG: low voltage, STT changes, prolonged QT, premature beats, etc
- Echo: chamber dilation, wall motion abnormalities, pericardial effusion, etc
- Labs: increased APRs, leukocytosis, increased cardiac biomarkers, autoimmune antibodies, etc 0
- b/c S/S are not specific definitive diagnosis requires endomyocardial Bx (lymphocyte infiltration ± myocytosis) but even this test lacks sensitivity and specificity
- Studies are looking into the use of PCR in evaluating myocardium for infectious agents
- Tx
- Supportive care for CHF, arrhythmias, etc 0
- Avoid digoxin b/c increases harmful cytokines
- There is little evidence to support the use of immunosuppression/IVIG unless due to an autoimmune process
- Studies are underway evaluating the utility of antiviral IFN-alpha in pts w/ +enterovirus myocarditis
- Avoid exercise b/c of increased r/o sudden death

## **Acute Pericarditis**

Etiology

0

- Idiopathic (30% after extensive work-up)
  - likely postviral b/c often there is a preceding flu-like illness therefore always ask
- O Infectious
  - Viral: Coxsackie, Echo, Adeno, Entero, Influenza, HIV, etc
    - Bacterial: TB, Lyme, Strept/Staph (from pneumonia, cardiac surgery, endocarditis), etc
  - - **Fungal** Parasitic
- Neoplasm (refer above)
- Adjacent Dz
  - Acute vs Chronic Post-MI
  - Pneumonia
  - **Pulmonary Embolus**
- Autoimmune
  - Connective Tissue Dz: SLE, Drug-Induced Lupus, Scleroderma, RA, Sarcoidosis
  - Vasculitides
  - Drugs: Heparin, Coumadin, Doxorubicin
- Metabolic 0
  - Uremia
- Trauma
  - Post-Pericardiotomy Syndrome (4wks after surgery, seen in 20% of CABG pts)
  - Post-MI (2wks after event)
  - Radiation
  - Injury
- Clinical Features
  - Unique Chest Pain (not pressure sensation like MI)
    - Localized to Retrosternal and Left Precordial Regions Radiating to Trapezius b/c the phrenic nerve innervates both the pericardium and trapezius muscles (not down arm or jaw like MI)

- insidious onset and can last a long time on the order of hours but sometimes up to days (not acute onset and short duration like MI)
- Positional
  - Worse = lying supine, coughing, swallowing, deep inspiration
  - Better = sitting up and leaning forward, shallow breaths
  - NB in MI no change with respiration or position
- Sometimes pain is absent especially in rheumatoid, neoplastic, radiation, TB, uremic pericarditis
- Does not occur with exertion
- o Pericardial Friction Rub (85% sens)
  - 2/2 rubbing b/t visceral and parietal pericardial surfaces
  - scratching, high pitched three component sound (notoriously variable and evanescent from minute to minute, 50% truly triphasic vs 30% biphasic vs 20% monophasic)
    - pansystolic (most common, ventricular systole)
    - early diastolic (uncommon, ventricular diastole)
    - late diastolic (moderately common, atrial systole)
  - best heard at LLSB with stethoscope placed firmly against chest (diaphragm)
  - pt holds breath after expiration while sitting up
  - different than pleural rub which occurs ONLY when pt breaths and suspends when apneic
- Fever
- o Nonproductive Cough
- Labs: leukocytosis, increased APRs, and sometimes (40%) elevated cardiac biomarkers (if elevated >1wk then
  consider concomitant myocarditis which portends worse prognosis), cardiac auto-antibodies (anti-myolemmal
  and anti-sarcolemmal)
- Diagnosis
  - o Clinical
    - EKG
- 1st: diffuse STE (except in V1 and aVR, upwardly concave ("happy face")) and PRD
  - NB in order to distinguish upward concave STE from early repol calculate the STE to TW height ratio in V6 and when >0.25 then very likely pericarditis and not early repol
  - NB STE reflects epicardial inflammation
  - 2<sup>nd</sup>: ST/PR Segments normalize
  - 3rd: diffuse TWI
  - unlike in MI which occurs during STE in pericarditis they invert AFTER ST segments normalize
- 4<sup>th</sup>: T Wave normalize
- Other: sinus brady, U waves, etc
- NB no conduction abnormalities (if so then myocarditis)
- Echo (not helpful except for occasional stranding)
- o CXR (not helpful)
- Management
  - o Most: can be managed as outpt not requiring Tx b/c mild and self-limited resolving in <1mo
  - o Some: need to be hospitalized requiring Tx
  - o lbuprofen 800mg PO TID-QID tapered over 7d → add colchicines (great also for chronic/recurrent pericarditis) 1.2mg x1d then 0.6mg BID 14d → add prednisone (great for TB etiology) 1mg/kg PO QD tapered over 30d → intrapericardial steroid instillation as last resort → pericardiectomy does NOT prevent relapses but often done as last resort
  - o NB do not use steroids early on b/c it has been found to increase risk of recurrence rather start with NSAIDs
  - o Treat underling cause
  - o Avoid AC b/c high r/o hemopericardium
  - o If infectious then abx
  - o Bx if suspicious for TB/neoplasm
- Complications
  - o Chronic/Relapsing Pericarditis
  - Constrictive/Restrictive Pericarditis
    - Etiology
      - Pericarditis (esp idiopathic, post-viral, radiation, uremia, TB, Coccidio, postpericardiotomy)
    - Clinical Features
      - Diastolic CHF: pericardial scarring results in normal early diastolic filling that is abruptly
        halted during late diastolic filling as the intracardiac volume reaches the limit defined by
        the stiff pericardium (unlike that seen in restrictive cardiomyopathy AND tamponade in
        which there is dysfunction throughout diastole)
        - o Early: Right CHF Symptoms
        - Late: Left CHF Symptoms
      - Pericardial Knock (sound caused by the abrupt cessation of ventricular filling)

- Constriction can affect coronaries and grafts causing ischemic events
- Diagnosis
  - VERY IMPORTANT TO DISTINGUSH FROM R-CM WHICH IS VERY SIMILAR b/c constrictive
    pericarditis can potentially be corrected w/ surgery whereas in R-CM there is no Tx and
    prognosis is poor, in R-CM myocardial relaxation AND compliance is affected whereas in
    constrictive pericarditis relaxation is normal rather there is just impaired compliance w/
    a finite diastolic volume
  - Clinical
  - EKG
- Low Voltages
- NO conduction abnormalities
- CXR
- o Calcifications
- Echo/CT/MRI
  - o Thickened Pericardium but NO Thickened Wall
  - "Septal Bounce" (abrupt displacement of septum during rapid filling in early diastole)
- Cath
  - o Elevated AND Equal Diastolic Pressures in all Four Chambers
  - "Square Root Sign" (ventricular pressure tracing shows rapid y descent w/ decreased pressure at onset of diastole and rapid increased pressure in early plateau)
- Tx
- Can be transient so try NSAIDs, diuretics (cautious as these pts are dependent on higher than normal preload to maintain stroke volume), and sodium restriction for a few months and if no improvement then surgery
  - Surgical Resection of Pericardium (pericardiectomy)
    - Surgical mortality of 10%, 50% effective, it take months for Sx to resolve, post-op there is decreased EF, etc therefore only done for pts in NYHA Class II-III (not I b/c unnecessary and not IV b/c benefit is marginal)
- Pericardial Effusion

  Definition
  - slow exudation of any volume into pericardial space (pericardium can stretch over time to accommodate any volume if given enough time therefore volume does not matter)
  - Etiology
    - water retention states (CHF, nephrotic syndrome, cirrhosis, et al) (most common)
    - pericarditis (esp TB, neoplasm esp lung/breast/melanoma/lymphoma, pyogenic)
    - chronic hypoTH
    - amyloidosis
  - Clinical Features
    - Muffled/Distant Heart Sounds
      - Dullness over left posterior lung field 2/2 compressive atelectasis (Ewart's Sign)
      - Even though there is an effusion often a rub is STILL heard which raises the question on the theory that the rub is 2/2 to the rubbing of pericardial surfaces
    - Diagnosis
      - Clinical
      - EKG
        - Low Voltages
          - Electrical Alternans (alternate beat variation in the direction of the ECG waveforms due to pendular swinging of the heart within the pericardial space aka motion artifact)
      - Echo (best diagnostic tool)
        - sensitive down to 20mL effusions (normally there is ~10-50cc of clear plasma ultrafiltrate fluid in space)
        - sometimes difficult to differentiate from epicardial fat therefore order an MRI
      - CXR
- o sensitive down to 250mL effusions
- o "water bottle shaped" cardiac silhouette
- Cath
  - o Elevated AND Equal Diastolic Pressures in all Four Chambers
- Pericardiocentesis w/ Bx
  - o Only if you suspect cancer, systemic dz, or bacterial infection

- Check cell count w/ diff, cytology, GS & Cx, Fungal Cx, AFB, ADA and PCR for 0
- 0 Although often ordered there are no accepted criteria which link the following (total protein, glucose, pH, LDH) to a diagnosis
- If negative very likely cancer
- In general when the cause is unknown pericardiocentesis w/ Bx only provided a dx in 5% of cases!!!
- Management
  - treat underlying cause
  - Most: no direct Tx necessary b/c mild and self-limited resolving in <1mo but follow with serial echo in 3mos
  - Some: require Tx (bedside pericardiocentesis or surgical pericardial window) b/c severe (beginning to tamponade), lasting >3mo, or likely secondary cause

### **Pericardial Tamponade**

- Definition
  - quick exudation of any volume into pericardial space
- Etiology

  - iatrogenic (central line placement, pacemaker insertion, pericardiocentesis)
  - post MI free-wall rupture
  - proximal aortic dissection w/ rupture
  - pericarditis (esp idiopathic, neoplasm, uremia, TB, bacterial)
- Clinical Features
  - Muffled Heart Sounds w/ Soft PMI
    - Hemodynamic Instability

Beck's Triad

cardiogenic shock w/o pulmonary edema... VERY IMPORTANT TO KNOW increased intrapericardial pressure resulting in compression of heart chambers resulting in decreased venous return resulting in decreased CO and thus hypotension/tachycardia

JVD and Kussmaul's Sign

prominent x descent (rapid filling during ventricular systole)

absent y descent (absent filling during diastole)

Both R/LV are squished by blood in pericardial sac  $\rightarrow$  decreased capacity  $\rightarrow$ during inspiration a lot of blood returns to RV furthering squishing the LV → less blood ejected by LV → exaggerated decrease in SBP (>10mmHg) or exaggerated decrease in amplitude of the femoral/carotid pulse during inspiration

In RV infarct the increased blood does not squish the LV rather the pericardial sac accommodates it

Diagnosis

# EKG (same as effusion)

- Echo (best diagnostic tool)
  - same as effusion + septal shift with inspiration, R vent/atrial free wall invagination or diastolic collapse, IVC plethora, respirophasic changes in transvalvular velocities (increased across TV and decrease across MV w/ inspiration)
- CXR (same as effusion)
- Cath
- same as effusion + inspiratory increase in R sided pressures and concomitant decrease in L sided pressures
- Management
  - Pressors, fluids, emergent pericardiocentesis except if you suspect hemorrhagic 2/2 free wall rupture, aortic dissection, etc in which case pericardiocentesis is deadly