# Valvular Heart Disease
# Other Cardiac Issues

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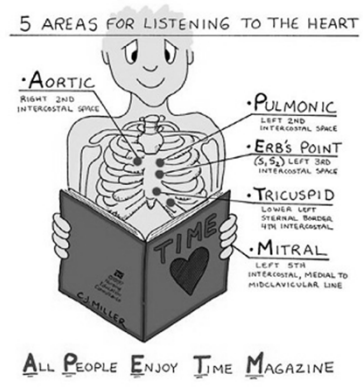
## CMC-CSC
## Test Blueprint

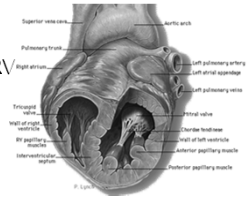
A. Acute Coronary Syndrome
B. Dysrhythmias
C. Heart Failure
D. **Other Cardiac Issues**
   - **Papillary Muscle Rupture**
   - **Ventricular Septal Rupture**
   - **Ventricular Aneurysm**
   - **Valvular Heart Disease**
   - **Acute Inflammatory Disease**
E. Vascular Issues

**B. Surgical Treatment of:**
1. Non-congenital heart defect repair in adults (e.g., ventricular septal defect [VSD], left ventricular aneurysm repair)
2. Cardiac rhythm disorders (e.g., MAZE procedure/modified MAZE)
3. Congenital heart defect repair in adults
4. Coronary heart disease
5. Hypertrophic obstructive cardiomyopathy (HOCM)
6. Valvular disease
   a. repair
   b. replacement

CMC | CSC




## Papillary Muscles

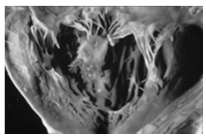


- Located in the ventricles
- Three papillary muscles in RV
  - Anterior -- largest
  - Posterior
  - Septal
- Two papillary muscles in LV
  - Anterior -- largest
  - Posterior
- Attached to the AV valves (mitral & tricuspid)
- Contact to prevent inversion or prolapse of the mitral or triscuspid valves
- Prevents regurgitation

## Papillary Muscle Rupture
### Acute Mitral Regurgitation

Cause

- Complication of AMI
- Occurs within first week
- More with inferior MI or subendocardial MI



## Papillary Muscle Rupture
### Acute Mitral Regurgitation

Clinical Manifestations

- Hypotension
- Tachycardia
- Dyspnea
- Crackles
- S3
- New **holosystolic** at the apex

- ↑ RAP, PAP, PAOP
- Large *v* waves on PAOP waveform
- ↓ CO/CI
- ↓ $SpO_2$, $SvO_2$

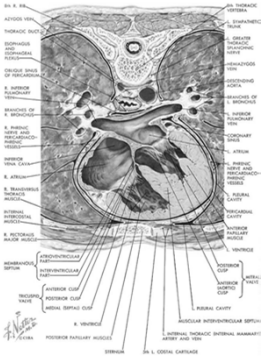
Symptoms of Acute Cardiogenic Shock & Pulmonary Congestion
New **holosystolic** at the apex

## Papillary Muscle Rupture

Treatment

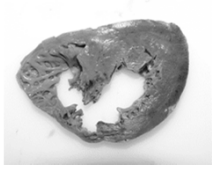
- Prepare for emergency mitral valve replacement
- Treat the symptoms of the mitral regurgitation, cardiogenic shock and pulmonary congestion
  - Supportive management until surgery
  - Afterload reduction with nitrates and nitroprusside
  - Diuretics
  - IABP
  - Oxygen/ventilator

## Ventricular Septal Rupture



Cause

- Complication AMI
- Occurs 3 and 5 – 7 days post AMI
- Complication of endocarditis



## Ventricular Septal Rupture

### Symptoms

- Hypotension
- Tachycardia
- Tachypnea
- New **holosystolic murmur at lower left sternal border**
- Thrill
- Pulmonary congestion
- Syncope
- ↑ RAP, PAP
- Large *v* on PAOP waveform
- ↑ $SvO_2$
- ↑ oxygen gradient (left to right shunt)
- Inaccurate ↑ CO/CI

Sudden hemodynamic compromise and pulmonary congestion
New **holosystolic murmur at lower left sternal border**

## Ventricular Septal Rupture

Treatment

- Emergent surgical repair
- Supportive care of cardiogenic shock while preparing for surgery
  - Vasodilators
  - Inotropes
  - Diuretics
  - IABP

## Ventricular septal defects

- Opening of the septum between the right and left ventricle
- Oxygenated blood in the LV returns to the RV rather than the oxygenated blood continuing forward to deliver oxygen to the cells.
- Results in an increase in ventricular workload
- Leads to heart failure

Normal Heart & Heart with VSD

## Ventricular septal defects

Clinical features

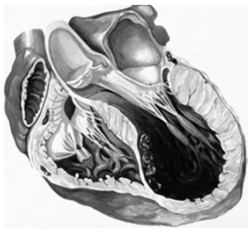
- Harsh and loud holoystolic murmur
- Left atrial dilation
- Left ventricular enlargement
- Pulmonary artery dilation
- Left to right shunt

Normal Heart & Heart with VSD

Holosystolic murmur

## Ventricular Aneurysm

- A ballooning, dyskinetic area of infarcted myocardium
- May be a complication of AMI
- Prone to dysrhythmias, intraventricular thrombus formation, and rupture



## Ventricular Aneurysm

Clinical Presentation

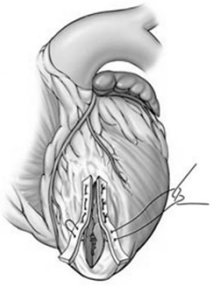
- Diffuse PMI – spread over more than one ICS
- Left ventricular heave
- Atrial Fibrillation or ventricular arrhythmias
- Persistent ST segment elevation
- CXR= left ventricular dilation
- Echo = dyskinesia and left ventricular dilation
- Signs of LVF may be present
- Systemic emboli symptoms may be present
  - Cerebral emboli
  - Peripheral emboli with acute arterial occlusion

## Ventricular Aneurysm

Treatment

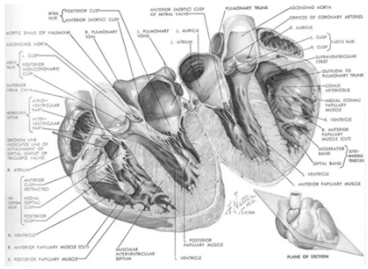
Medical
- Antiarrhythmic medications
- Possible ablation for ventricular arrhythmias
- Anticoagulation medications
- Treatment of HF with ACEI, beta blockers, diuretics, inotropes, and vasodilators

Surgical
- Excision of the weakened area of the myocardium,
- Patch is sewn in the edges of the remaining viable myocardium



## Valvular Heart Disease

- Mitral
- Aortic



## Valvular Heart Disease

- An acquired or congenital disorder of a cardiac valve
- Characterized by
  - Stenosis (obstruction)
  - Regurgitation (backward flow)
- Can occur acutely
- Typically is a chronic progressive disorder
- Causes a significant impact on quality of life
- Medical management delays the inevitable surgery for replacement/repair
- Prosthetic valve creates new problems

## Valvular Heart Disease

Common Causes of Acquired Valvular heart disease

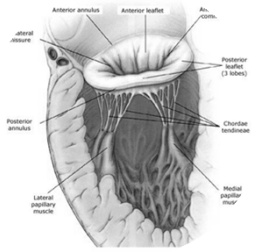
- Rheumatic heart disease
- Degenerative diseases
- Infective endocarditis

## Valvular Heart Disease

Clinical Management

- Cardiac compensatory mechanisms can maintain stability for years before symptoms occur.
- Key is early diagnosis to prevent the long-term consequences
  - Pulmonary hypertension
  - Heart Failure
  - Atrial fibrillation
  - Thromboembolism
- Important to understand
  - The structure and function of the valves
  - The causes and treatments of each disorder

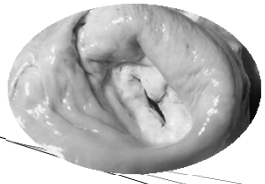
## Mitral Valve




- Large anterior leaflet
- Small posterior leaflet
- Chordae tendineae and papillary muscles prevent the prolapse of valve leaflets into left atrium during systole

## Mitral Stenosis

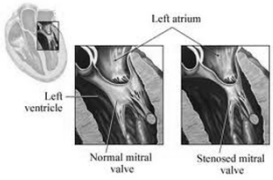
- Mitral valve will not open completely
- Restricts flow of blood from left atrium to LV



- Cause
  - Mostly rheumatic fever
  - Some calcified degenerative disease
- Continuous, slow progressive disease with symptoms appearing 20-40 years after rheumatic fever

## Mitral Stenosis

Pathophysiology




1. Small opening causes ↓ blood flow and ↓ CO
2. ↑ workload in Left Atrium
3. ↑ pressure in LA
4. LA dilation & hypertrophy
5. May lead to A fib
6. LA blood flow stagnant
7. May cause clot formation & thromboembolism
8. ↑ in LA pressure →backflow into pulmonary artery
9. Leads to pulmonary hypertension, congestion, right ventricular hypertrophy and right sided heart failure

LV size and contractility = normal in MS

## Mitral Stenosis

Clinical Presentation

- Mild MS
  - Dyspnea on exertion from pulmonary congestion
- Moderate MS
  - Fatigue
  - Paroxysmal nocturnal dyspnea
  - Atrial fib
- Severe MS
  - Dyspnea on mild exertion or at rest

- Ruddy face (mitral facies)
- Heart Sounds
  - Loud first heart sound S1 (closing snap)
  - Mid-diastolic murmur/rumble at apex
  - Opening snap
  - Right sided S3 & S4
  - Right ventricular heave at sternum
- CXR
  - Left atrial and RV enlargement
  - Pulmonary congestion
- EKG
  - LA enlargement: Notched P waves *P Mitrale*
  - Right ventricular hypertrophy – tall R waves V 1 & V2

Pulmonary congestion & right sided failure signs

## Mitral Stenosis

Clinical manifestations that affect post op care

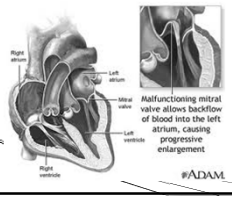
- Nx LV function
- Pulmonary Hypertension
- RV failure
- Tricuspid insufficiency
- High left atrial pressures and pulmonary pressures
- Low CO and pulmonary congestion

## Post op MVR and repair for MS

- Assess for pulmonary hypertension
- Increased PVR leads to RV failure
- Increased CVP = possible RV decompression
- TEE to assess for RV and LV function
- Dobutamine, Milrinone, Norepinephrine to increase contractility of RV and ↓ PVR
- Fluid administration
- PAD does not reflect LA filling pressures related to pulmonary hypertension – Wedge more accurate
- PA catheter may be placed farther in related to dilated pulmonary arteries
- IABP usually not indicated as no LV dysfunction but RV dysfunction

## Mitral Regurgitation

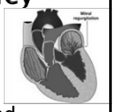
- Mitral valve fails to close completely
- Blood is propelled backward into the LA during systole




- Causes
  - Anything that affects any part of the MV
    - Mitral annulus
    - Valve leaflets
    - Chord tendineae
    - Papillary muscle
  - Mitral Valve Prolapse
  - Rheumatic heart disease
  - Infective endocarditis
  - Cardiomyopathy
  - Ischemic heart disease

## Mitral Regurgitation/Insufficiency

Pathophysiology



1. During systole, a portion of blood is ejected back into the LA
2. ↓ blood in LV → ↓ CO
3. ↑ blood in LA → ↑ LA pressures → pulmonary congestion and ↑ pulmonary pressures → RV hypertrophy
4. During diastole, blood continues to flow into LV→ ↑ LV volume
5. LV hypertrophy

MR = LA enlargement, Left or Ventricular Failure

## Mitral Regurgitation

Clinical Presentation

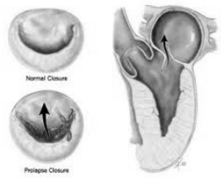
- May be asymptomatic for years
- Initial symptoms
  - Left sided failure
  - Dyspnea on exertion
- Cough
- Peripheral Edema
- Palpitations from new onset of A fib

- Heart Sounds
  - Holosystolic murmur – high pitched, blowing at apex
  - S2 – widely split
  - Displaced PMI laterally
  - Maybe right sided S3 & S4
- CXR
  - Cardiomegaly
  - LA enlargement
  - LV hypertrophy
  - Possible pulmonary congestion
- EKG
  - LA enlargement *P mitrale*
  - Left and/or right ventricular hypertrophy
    - Large QRS complexes

Endocarditis risk is RARE

## Mitral Valve Prolapse

- Mitral valve leaflets prolapse back into the left atrium during systole




- Cause: myxomatous degeneration of valve leaflets causes leaflets to enlarge and prolapse
- Frequently asymptomatic & no treatment needed
- Midsystolic click
- Late systolic murmur

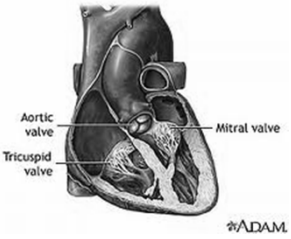
## Post op for Mitral Valve Repair for MR

- Immediate ↑ SVR due to no backflow of blood in LA
- Pulmonary hypertension & Myocardial hibernation take time to reverse
- Inotropes (Milrinone, Dobutamine)
- IABP
- Monitor for RV failure

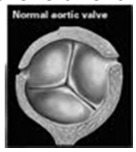
## Mitral Valve Repair vs Mitral Valve Replacement

- Repair preserves native valve
- Repair is favored due to disadvantages of prosthetic valves
  - No anticoagulation needed for repair
- Technically more difficult
  - Depends on degree of regurgitation,
  - Pathophysiology of the regurgitation
  - LV function,
  - Ability of surgeon

## Aortic Valve

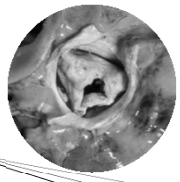



- Has three leaflets or cusps
- Cusps close as the pressure in the aorta becomes *greater than* the pressure in the left ventricle.




## Aortic Stenosis

- Aortic valve will not open completely
- Restricts flow of blood from left ventricle to aorta

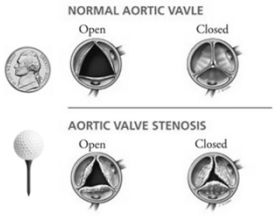


- Most common valve lesion in USA
- Gradual progressive disease – usually remain asymptomatic for decades.

## Aortic Stenosis

Clinical Presentation

- Echocardiogram:
  - Grades:
    - Normal AVA > 2.5 – 3.5 cm2
    - Mild AVA > 1.5 cm2
    - Moderate AVA = 1.0 – 1.5 cm2
    - **Severe AVA < 1.0 cm2**
    - Critical AVA < 0.75 cm2
  - Mean gradients: > 40-45 mmHg
  - Peak velocity over 4 m/s

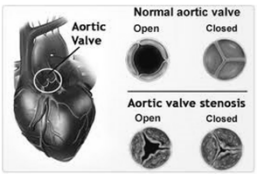



Syncope --- Systolic ejection harsh murmur

## Aortic Stenosis

Pathophysiology

1. Small opening causes ↓ blood flow and ↓ CO
2. ↑ Afterload
3. ↑ workload in Left ventricle
4. ↑ pressure in LV
5. LV hypertrophy
6. LV becomes stiff
7. High LV pressures leads to LV failure and left atrial enlargement
8. High pressures transmitted to the lungs leads to pulmonary hypertension, pulmonary edema and right sided failure




LV size and contractility = abnormal in AS

## Aortic Stenosis

Clinical Presentation

- Syncope, Dyspnea, Angina, – especially with exertion
- Angina more common if CAD as comorbidity
- HF with severe AS

- Heart Sounds
  - Systolic ejection murmur (harsh) at the right sternal border radiating to neck
- CXR
  - May be normal
- EKG
  - Left ventricular hypertrophy with severe AS
  - Left Atrial enlargement, Left axis deviation, Left BBB

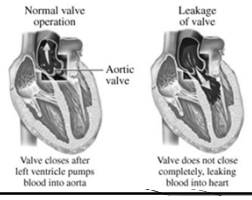
Syncope --- Systolic ejection harsh murmur

## Post op AS

- Inotropes rarely needed
  - Except in patient with impaired LV function – may be caused by myocardial stunning
- Avoid ↑ BP in immediate post-op period
  - "thin" aorta in bicuspid valve patients- more likely to rupture under high pressure
- Maintain AV Synchrony
- Maintain adequate preload
  - PCWP > 15 mm HG
- Avoid hypovolemia and inotropes

## Aortic Regurgitation/Insufficiency

- Aortic valve fails to close completely
- Backflow of blood into the left ventricle during diastole




- Causes
  - Pathological conditions of the aortic valve cusps or aortic root.
  - Valve cusps
    - Rheumatic heat disease
    - Infective endocarditis
  - Aortic Root
    - Hypertension
    - Aortic dissection
    - Marfan's Syndrome
    - Syphilis
  - Severe AI – most frequently caused by bicuspid valve

## Aortic Regurgitation/Insufficiency

Pathophysiology

1. Volume overload leads to compensatory mechanisms
   - Left ventricular hypertrophy
   - ↑ End-diastolic volume which allows normal EF despite ↑ afterload.
2. ↑ LV afterload as the ↑ volume ejected into the high pressured aorta.
3. Ventricular wall thickening with dilatation to accommodate volume overload.
4. Ventricular wall thickens without enlargement but with diminished capacity to accommodate pressure overload.
5. The balance between afterload excess, preload reserve, and hypertrophy may continue for decades
6. Symptoms occur when this balance can't be maintained and there is a reduction in EF or LV dysfunction

AI – unique with both volume & pressure overload

## Post op AVR for AR

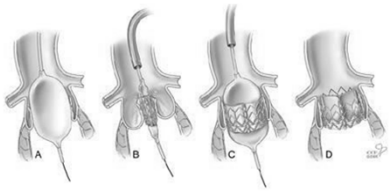
- Due to dilated ventricle use
  - IV vasodilators
  - Inotropic support to promote ventricular empting
    - Milrinone
    - Dobutamine
  - IABP
- Maintain AV synchrony

## Surgical Treatment for Aortic Valve Disease

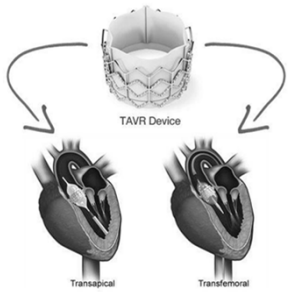
- Aortic Valve Replacement (mainstay)
- Aortic Valve Repair (not mainstream)
- Transcatheter Aortic Valve Replacement (TVAR)

## Transcatheter Aortic Valve Replacement (TVAR)

- Trileaflet bioprosthesis mounted on a balloon catheter delivered through the arterial system via a guidewire. Device is inserted into the midpoint of the native valve



## Trans Apical vs Trans Femoral




- Femoral most common

https://www.youtube.com/watch?v=ztl3cc2EOmM

## TAVR

**Pro's**

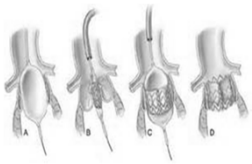
- Less invasive than traditional AVR's
- No sternotomy or cardiopulmonary bypass
- Less ventilation time or extubated in OR
- Shorter ICU length of stay and often discharged within 48 hours postop

**Con's**

- Elderly population with comorbidities
- Higher risk for delirium due to sedation or pain management
- Screening for physical therapy

## TAVI/TAVR Consideration

- Indications
  - Severe symptomatic aortic stenosis not amenable/too high risk for open AVR
    - >50% mortality
    - NYHA class II or greater
    - 1 cardiologist and 2 surgeons
  - Life expectancy greater than 1 year
  - Aortic annulus by CT and echo appropriate size
  - Porcelain aorta
  - Frailty
  - Chest deformity – radiation
  - Open bypass grafts
  - Liver cirrhosis
  - Pulmonary fibrosis
  - Impaired LV function
  - Renal disease



## TAVI/TAVR Exclusions

- Exclusionary Criteria:
  - Bicuspid or unicuspid or noncalcified aortic valve
  - Native aortic annulus size <18 mm (for a native valve), and no greater than 29 mm – size dependent (4 sizes available currently)
  - Severe native aortic regurgitation (>3+)
- Relative Exclusion Criteria:
  - Evidence of MI within 1 month of valve placement
  - Hemodynamic or respiratory instability requiring inotropic support, mechanical ventilation, or mechanical heart assistance within 30 days
  - Hypertrophic cardiomyopathy with or without obstruction
  - Left ventricular ejection fraction <20 percent
  - Severe pulmonary hypertension and right ventricular dysfunction
  - A known contraindication or hypersensitivity to all anticoagulation regimens or inability to be anticoagulated for the study procedure
    - Dual antiplatelet therapy is required for at least 3 months
  - Renal insufficiency (eg, creatinine >3.0 mg/dL) and/or end-stage renal disease requiring chronic dialysis
  - Echocardiographic evidence of intracardiac mass, thrombus, or vegetation
  - Magnetic resonance imaging-confirmed stroke or transient ischemic attack within six months (180 days) of the procedure.
  - Severe incapacitating dementia
  - Estimated life expectancy <12 months due to noncardiac comorbid conditions
  - Severe mitral regurgitation
  - Significant aortic disease, including the following abnormalities may preclude a transfemoral approach:
    - Could be done transaortic or transapical

## Post Op TVAR Femoral

- Usually extubated in OR, if not within 2-4 hrs postop
- Monitor bilateral puncture sites – hold pressure if oozing or bleeding
- Monitor pulses distal to insertion site due to the large catheters and embolization risk
- Monitor neuro assessment due to high risk for strokes
- Maintain SBP between 100mmHg – 130mmHg
  - May use beta blockers or other vasodilators for hypertension
- Discontinue Arterial line after extubation and venous sheath when ACT < 180
- Internal Jugular discontinued on POD 1 and transferred to Telemetry
- All patients assessed for rehab upon transfer from ICU

## Post Op TVAR Apical Postop

- Monitor hemodynamics, neuro assessment, urine output, & chest drainage same as an open sternotomy incision
- Wean to extubate within 6 hours of anesthesia end time. Encourage incentive spirometer every hour while awake
- Discontinue femoral lines after extubation
- Ice chips and advance diet as tolerated
- Up in chair early am and ambulate with physical therapy or nurses 3-4 times/day
- Discontinue PA catheter and arterial line POD 1

✓

## Potential TVAR Complications

- Complete Heart Block due to Aortic Valve edema.
- Hypotension
  - Monitor amount of sedation or vasodilating medications for cause of hypotension
- Check groin sites for bleeding, lower abdomen for signs of retroperitoneal bleed, check peripheral vascular pulses
- Monitor Labs (Hgb/Ht)
- Vasovagal response
- Stroke
  - Assess neuro status with VS's

## Aortic Regurgitation/Insufficiency

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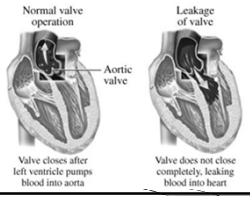
- Backflow of blood into the left ventricle during diastole

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    - Infective endocarditis
  - Aortic Root
    - Hypertension
    - Aortic dissection
    - Marfan's Syndrome
    - Syphilis
  - Severe AI – most frequently caused by bicuspid valve

## Aortic Regurgitation/Insufficiency

Pathophysiology

1. Volume overload leads to compensatory mechanisms
   - Left ventricular hypertrophy
   - ↑ End-diastolic volume which allows normal EF despite ↑ afterload.
2. ↑ LV afterload as the ↑ volume ejected into the high pressured aorta.
3. Eccentric hypertrophy (ventricular wall thickening with dilatation) to accommodate volume overload.
4. Modest concentric hypertrophy (ventricular wall thickens without enlargement but with diminished capacity) to accommodate pressure overload.
5. The balance between afterload excess, preload reserve, and hypertrophy may continue for decades
6. Symptoms occur when this balance can't be maintained and there is a reduction in EF or LV dysfunction

AI – unique with both volume & pressure overload

## Aortic Regurgitation/Insufficiency

Clinical Presentation

- May be asymptomatic for years
- Fatigue
- Dyspnea on exertion
- Angina
- Palpitations
- Widen pulse pressure > 50 mmHG

- Heart Sounds
  - Decrescendo diastolic blowing murmur – best heard in upright position and leaning forward.
- CXR
  - Enlarged heart
  - Dilation of proximal aorta
- EKG
  - Left ventricular hypertrophy

Wide pulse pressure – Diastolic blowing murmur

## Aortic Regurgitation/Insufficiency

Clinical Presentation
Signs

- Austin Flint murmur
  - Decrescendo diastolic blowing murmur
- Hill Sign
  - Systolic BP in lower extremities at least 20mmHg higher than arms
- Duroziez sign
  - Systolic and diastolic bruit heard when femoral artery is compressed by stethoscope
- Corrigan pulse
  - ↑ volume and rate of the rise of the radial pulse when the wrist is elevated perpendicular to the body of a supine patient
- de Musset sign
  - Bobbing of the head with each systolic beat

https://www.youtube.com/watch?v=C6mTmpP9Lvw

## Tricuspid Valve Disease

- Rheumatic
- Endocarditis (esp IV drug abuser)
- Functional (most common form): secondary to left sided pathology often accompanied by pulm HTN
- Congenital: AV canal, VSD, Ebsteins, Myxoma

## Surgery for Tricuspid Valve Disease

- Primary indication for tricuspid valve repair is severe TR in patients requiring surgery for mitral valve disease
- Tricuspid stenosis: don't see often
- TR: with left sided lesion, right sided failure, mod to severe TR

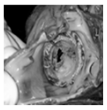
## Valve Replacement Considerations

| | Tissue | Mechanical |
|---|---|---|
| Age | Over 65 yo | Under 65 |
| Longevity | 10-15 years | Potentially Lifetime |
| Anticoagulation | Aspirin lifelong<br>Warfarin – 3 months??? | Warfarin lifelong |
| Reoperation risk | Patient dependent | As low as 1% risk lifetime |

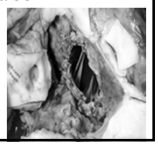
## Determining Replacement Valve Type

- AHA/ACC Guidelines:
  - Mechanical prosthesis:
    - Already have a mechanical valve in the mitral or tricuspid position
  - Bioprosthesis aka "Tissue" valve:
    - Any age who will not take warfarin or who have major medical contraindications to warfarin therapy
    - Aged 65 years or older without risk factors for thromboembolism
    - Woman of childbearing age
  - Homograft:
    - Active prosthetic valve endocarditis
  - TAVI/TAVR:
    - High prohibitive risk for open AVR per FDA guidelines
    - Moderate risk: Partner II trial

## Postoperative Valve Considerations



- Physical examination
  - Normal prosthetic heart valve sounds:
    - Mechanical valves:
      - Loud, high-frequency, metallic closing sound
      - Soft opening sound
    - Tissue valves:
      - Closing similar to those of native valves
  - New onset murmurs is a concern
    - murmur – though hard to hear – would raise suspicion



## All Valve Surgeries Postoperative Considerations

- Prosthetic heart valve malfunction:
  - Acute prosthetic valve failure:
    - Sudden onset of dyspnea, syncope, or precordial pain
    - Sudden death
    - Hyperdynamic precordium
    - Pronounced JVD
  - Subacute valve failure:
    - Gradually worsening congestive heart failure
    - Unstable angina
    - Hemolytic anemia
    - Asymptomatic

## Postoperative Valve Considerations

- Embolic complications
  - Stroke
  - TIA
- Anticoagulant-related hemorrhage
  - Hemorrhage site – brain, abdomen, etc.
- Dysrhythmia
  - AV Block
  - Atrial dysrhythmias

## Prosthetic Valve Endocarditis

- Blood borne bacterial traveling to the heart and growing on the valve
- Dental or other procedures may provoke bacteremia

## Case Study

50y/o mitral valve repair and annuloplasty with daVinci Robotic system

- He is discharged on POD # 3
- His post op recovery for the next 8 weeks is unremarkable.
- Went to dentist for dental cleaning six days ago

- A few days ago he began feeling very tired, his pulse was irregular
- Went to ED in rural hospital
- Atrial Fibrillation Rate 140
- Given Lopressor 5 mg IV
- Transferred to referral hospital

## Admission

- BP 137/80
- HR 98
- RR 18
- T 100.9 Orally
- SpO2 97%

| | | 07/13/10 04:25 |
|---|---|---|
| CBC | Order Status | Final Report |
| | WBC | *14.0 H* |
| | RBC | 5.02 |
| | HGB | 14.6 |
| | HCT | 44.7 |
| | PLT CNT | 266 |
| | MCV | 89.0 |
| | MCH | 29.1 |
| | MCHC | 32.7 |
| | NEUT % | 73.1 |
| | LYMPH % | *12.6 L* |
| | MONO % | *12.7 H* |
| | EOS % | 0.5 |
| | BASO % | 0.1 |
| | NEUT # | *10.2 H* |
| | LYMPH # | 1.9 |
| | MONO # | *1.8 H* |
| | EOS # | 0.1 |
| | BASO # | 0.0 |
| | RDW | 13.6 |
| COMPREHENSIVE METABOLIC | Order Status | Final Report |
| | SODIUM | 139 |
| | POTASSIUM | 3.7 |
| | CL | 103 |
| | CO2 | 27 |
| | BUN | 9 |
| | OSMO-CALC | 276 |
| | CREAT | 0.9 |
| | GLUCOSE | 92 |
| | ANION GAP | 9 |
| | T PROT | 6.9 |
| | ALBUMIN | 3.7 |
| | CALCIUM | 8.9 |
| | ALK PHOS | 68 |
| | SGPT | *81 H* |
| | SGOT | 19 |
| | BIL-TOTAL | 0.9 |
| | GFR | 89& |

## More Info

- Clindamycin 600 mg prior to cleaning (Allergic to PCN)
- Started experiencing diarrhea the next two days so took Imodium
- Diarrhea subsided now (three days later)

## Echo

- Echodense mobile structure on the posterior leaflet suggestive of infective endocarditis close to the annular ring on the posterior leaflet without signs of abscess

## Another Patient. MRSA

**Mitral Valve** No mitral leaflet calcification is visualized. There is no evidence of mitral stenosis. There is severe mitral regurgitation observed. A mass is visualized on the mitral valve which appears consistent with a vegetation.Attached to posterior leaflet/17mm by 17mm.ICU RN/team(remi) notified.


## What is SBE?
## Subacute bacterial endocarditis

- Antibiotic prophylaxis is indicated for the following high-risk cardiac conditions:
  - Prosthetic cardiac valve
  - History of infective endocarditis
  - Congenital heart disease (CHD)
  - Cardiac transplantation recipients with cardiac valvular disease
- For these procedures
  - Dental
  - Invasive respiratory (bronch)

## Standard general prophylaxis

- Amoxicillin
  - Adult dose: 2 g PO
  - Pediatric dose: 50 mg/kg PO; not to exceed 2 g/dose
  - Administer once as a single dose 30–60 min before the procedure.
- Ampicillin, Clindamycin, Cephalexin, Cefazolin, or Ceftriazone
  - May be used if allergic or unable to take oral
  - See guidelines for specific doses

## Infective Valve Endocarditis

- Medical therapy is first line treatment
- Surgery indicated for those patients with acute infective valve endocarditis and life-threatening heart failure or cardiogenic shock

## The left ventricle is normal size in which valvular disease?

1. Aortic Stenosis
2. Aortic Insufficiency
3. Mitral Stenosis
4. Mitral Regurgitation

## ANSWER

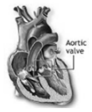
1. .
2. .
3. Mitral Stenosis

| | MS | MR | AS | AR/AI |
|---|---|---|---|---|
| Heart Sounds | | | | |
| Symptoms | | | | |
| Atrial size | | | | |
| Ventricular Size | | | | |

## Valvular Pearls

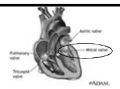
| | MS | MR | AS | AR/AI |
|---|---|---|---|---|
| Heart Sounds | Mid diastolic murmur at the apex<br>S3, S4<br>RV heave | Holosystolic murmur high pitched<br>Widely split S2<br>S3, S4 | Systolic ejection murmur harsh at right sternal border | Decrescendo diastolic blowing murmur – best heard sitting upright |
| Symptoms | Dyspnea<br>Pulmonary Hypertension<br>Pulmonary symptoms | Peripheral edema<br>Cough<br>LV failure<br>New onset AFib | Syncope<br>Dyspnea<br>Angina | Fatigue<br>Dyspnea<br>Angina<br>Palpitations<br>Wide pulse pressure > 50 mmHG<br>▸ Austin Flint murmur<br>▸ Hill Sign<br>▸ Duroziez sign<br>▸ Corrigan pulse<br>▸ de Musset sign |
| Atrial size | LA enlarged | LA enlarged | LA enlarged | LA enlarged |
| Ventricular Size | **LV normal** | LV enlarged | LV enlarged | LV enlarged |

## Valvular Surgery Pearls Aortic Valve




| | Aortic Stenosis | Aortic Regurgitation |
|---|---|---|
| Preop | LV hypertrophy<br>↑ SVR<br>s/s heart failure | LV hypertrophy |
| Post op | LV may not anticipate ↓ in SVR and continue to pump hard<br>Avoid hypertension and stress on suture line | IV vasodilators to ↓ SVR<br>Inotropic support to promote empting LV: Milrinone/Dobutamine<br>IABP |
| | | |

## Valvular Surgery Pearls Mitral Valve



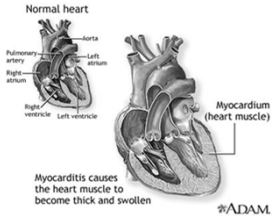
| | Mitral Stenosis | Mitral Regurgitation |
|---|---|---|
| Preop | Nx LV function<br>Pulmonary Hypertension<br>RV failure<br>High atrial & pulmonary pressures<br>Pulmonary congestion | Enlarged left atrium<br>Both common to have atrial fibrillation |
| Post op | Assess pulmonary hypertension (PVR)<br>Dobutamine or Milrinone + Norepinephrine to ↑ contractility of RV & ↓ PVR<br>Fluids<br>↑ CVP may indicate RV decompression<br>Treat atrial fibrillation | Immediate ↑ SVR due to no backflow of blood in LA<br>Pulmonary hypertension & myocardial hibernation take time to reverse<br>Inotropes (Milrinone, Dobutamine) + epinephrine<br>IABP<br>Monitor for RV failure<br>Treat atrial fibrillation |

## Acute Inflammatory Disease Cardiac "itis"

Myocarditis
Endocarditis
Pericarditis

## Myocarditis

- Inflammation of the heart muscle
- Challenging to diagnose and treat



# Myocarditis

Causes

- Infectious
  - Bacterial
  - Spirochetal
  - Fungal
  - Protozoal
  - Parasitic
  - Rickettsial
  - Viral
    - Coxsackievirus
    - Human immunodeficiency virus
- Immune Mediated
  - Allergens
  - Alloantigens
  - Autoantigens
    - Chagas Disease
    - Systemic lupus
    - Sarcoidosis
- Toxic Myocarditis
  - Drugs
    - Amphetamines
    - Anthracyclines
    - Cocaine
    - Ethanol
  - Heavy metals
    - Copper
    - Iron
    - Lead
  - Physical agents
    - Electrical shock
    - Radiation

# Myocarditis

Phases

- Acute: Day 0 – 3
  - Viral infection
  - Myocyte change
  - Myocyte antigens released
  - Cytokines released
- Subacute: Day 4 – 14
  - Infiltrating mononuclear cells
  - Cytokine production
  - T and B lymphocytes activated
  - Neutralizing antibodies
  - Viral clearance
- Chronic myocarditis: Day 15 – 90
  - Fibrosis
  - Cardiac enlargement
  - Apoptosis

Source: Moser & Riegal. Cardiac Nursing 2008

# Myocarditis

Clinical Presentation

- History of recent flu-like symptoms
  - Fever
  - Arthralgia (joint pain)
  - Fatigue
  - Chest pain
- Labs
  - ↑ Troponin I
  - ↑ CK MB
  - ↑ ERS
  - Leukocytosis
  - Cardiac immunoglobulin antibody titer 1:40 or greater
- Heart Sounds
  - Distant
  - S3 or S4
  - Pericardial friction rub
- EKG
  - Ventricular Arrhythmias
  - Heart Block
  - Diffuse ST segment changes and T wave changes
- CXR
  - Cardiomegaly
  - Pulmonary congestion
  - Pleural or pericardial effusion
- Endomyocardial biopsy for definitive diagnosis

Wide variance in symptoms – minimal symptoms to fulminant Heart Failure

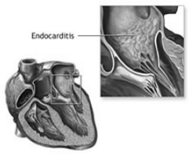
# Myocarditis

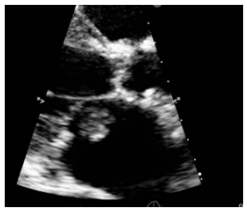
Treatment

- Supportive management of HF symptoms
  - IABP
  - VAD
- Treat cause
  - Antibiotics
  - Antifungals
  - Antivirals
  - Corticosteroids
  - Withdrawal of offending agent
- Anticoagulation if AF
- Early consideration for transplant if:
  - Progressive HF
  - Giant cell myocarditis
- Observe for pericarditis and tamponade
- Bedrest due to increased risk of myocardial inflammation
- Advise to abstain from exercise for several months
- Close and consitent follow-up every 1 – 3 months
  - Persistent inflammation will lead to dilated cardiomyopathy

# Endocarditis

- Inflammation/infection of the endocardium or lining of the heart.
- Usually involves the membrane lining the heart valves
- The invading pathogen typically adheres to the heart valves
- The pathogen creates solid vegetation or masses of bacteria, WBC, platelets, and fibrin that invade and destroy surrounding tissues
- Affects aortic and mitral valves more than tricuspid



# Infective/Bacterial Endocarditis

Causes

- IV drug use
- Body piercing
- Dental procedures
- Poor oral hygiene
- Rheumatic fever
- Invasive procedures

Most common bacterial pathogens are *Staphylococcus and Streptococcus* and fungal pathogens *Aspergillus fumigatus and Candida albicans*

## Infective/Bacterial Endocarditis

High risk groups

- Valve repair or replacement patients
- Patients with invasive lines
  - Central lines
  - Pacemakers
  - Cardiac caths
- Hemodialysis patients
- Congenital or acquired valvular heart disease
  - Septal defects
  - Bicuspid aortic valve
  - Mitral valve prolapse
- Immunosuppressed state

## Endocarditis

Acute infective endocarditis

- Progresses rapidly
- Often caused by *Staphylococcus* infection
- Typically occurs with normal valves and causes severe damage to the valves

Subacute endocarditis

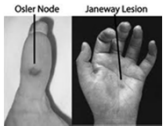
- Manifests initially as flu-like symptoms and progresses slowly and prolonged course
- Often caused by *Streptococcus or* gram negative infection
- Typically occurs with already damaged valves
- Outcome is usually good with adequate treatment

## Endocarditis

Clinical Presentation

- May be nonspecific
  - Fever
  - Diaphoresis
  - Weight loss
  - Myalgia (muscle pain)
  - Night sweats

  or
- Overt complications
  - Embolic stroke
  - Heart failure
- Heart sounds
  - New or changed murmur
  - S3
  - Pericardial friction rub




- Embolic or allergic vasculitis signs
  - Osler's nodes: painful nodules on pads of fingers and toes
  - Janeway lesions: nontender macules on palms and soles
  - Roth's spots: round white lesions on the retina
  - Occur from microembolization of the original vegetation

## Endocarditis

Diagnosis

- Labs
  - ***Positive blood cultures***
  - ↑ ERS
  - Anemia
  - Leukosytosis
  - ↑ C-reactive protein
  - Abnormal urinalysis
  - ↑ WBC
- Echocardiogram
  - Intracardiac vegetation
  - Dysfunctional valves

## Endocarditis

Complications

- Acute Renal Failure
- CVA
- Conduction Abnormalities
- Heart Failure
- Mycotic aneurysm
- Paravalvular abscess, perforation, or fistula
- Pericarditis
- Pulmonary emboli
- Septic arthritis
- Splenic abscess/infarct
- Systemic embolization

Source: Moser & Riegal. Cardiac Nursing 2008

## Endocarditis

Treatment

- Antimicrobial therapy
- Eradiation of endocardial vegetations
- Long term IV bacterial antibiotics
- Supportive therapy to treat and prevent complications from the disease progression
- If not responsive to medical therapy, surgery may be an option
  - Valve repair/replacement
  - Optimal time for surgery is when the patient is hemodynamical stable

## Endocarditis

Prophylaxis

- Prevention is essential in high risk groups
- Prophylactic Antibiotics
  - Dental, oral, upper respiratory tract procedures
    - Amoxicillin
    - Ampicillin
    - Clindamycin
    - Azithromycin
    - Cephalexin
    - Cefazolin
  - Genitourinary or gastrointestinal procedures
    - Amoxicillin
    - Ampicillin
    - Vancomycin
- Avoid unnecessary invasive lines
- "Don't Do Drugs!"
- Avoid body piercing

## Case Study
## 25 y/o white male comes to ED due to SOB and abdominal/back swelling

- Two days ago noticed abdomen and back were swollen. It was difficult to feel his spine as there was so much fluid
- Unable to walk across the room without becoming SOB
- Breathing worse when lying flat
- Difficulty speaking in full sentence
- Dry cough
- Weight gain of 10 pounds in last week

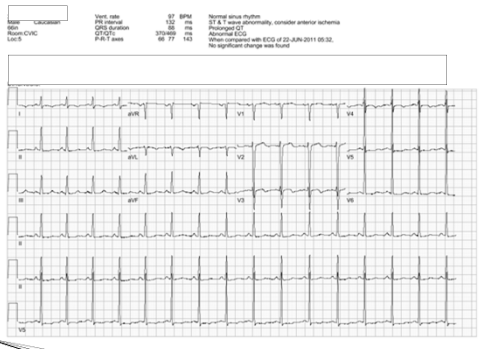
## Admission Vitals

- BP 140/90
- HR 109
- RR 24, moderately labored
- T 98.7
- $SpO_2$ 97% on 3 liters nasal cannula
- Weight 66.2 kg
- Pansystolic murmur 4/6 noted loudest at the left sternal border
- Coarse rales throughout all lung fields

## PMH

- Recent admission five days ago for hematuria, hyperkalemia, and acute renal insufficiency
- Left ureteral stent placed for sever hydrohephrosis of the left kidney
- Received 2 units of blood at that time

- One pack a day smoker
- Rarely drinks
- Occasional use of marijuana
- Has tried cocaine, Ecstasy, Adderall in the past
- Had several tattoos done a few months ago.



## Admission Labs

| | | 06/26/11 11:10 |
|---|---|---|
| BASIC METABOLIC PANEL | SODIUM | 148 H |
| | POTASSIUM | 3.7 |
| | CHLORIDE | 111 H |
| | CARBON DIOXIDE | 23 L |
| | BLOOD UREA NITRO | 34 H |
| | CREATININE | 1.44 H |
| | GLUCOSE | 116 H |
| | ANION GAP | 15 H |
| | CALCIUM | 8.2 L |
| | GFR | 60& |
| | OSMOLALITY (CAL) | 303 |
| CBC WITH DIFFERE | Order Status | Final |
| | WBC COUNT | 12.3 H |
| | RBC COUNT | 2.84 L |
| | HEMOGLOBIN | 8.8 L |
| | HEMATOCRIT | 24.3 L |
| | PLATELET COUNT | 246 |
| | MCV | 85.6 |
| | MCH | 28.2 |
| | MCHC | 32.9 |
| | LYMPHOCYTE % | 14.4 L |
| | NEUTROPHIL % | 80.2 H |
| | ABSOLUTE BASO | 0.0 |
| | ABSOLUTE EOS | 0.0 |
| | BASOPHIL % | 0.2 |
| | EOSINOPHIL % | 0.1 |
| | MONOCYTE % | 5.1 |
| | MONOCYTE # | 0.6 |
| | LYMPHOCYTE # | 1.8 |
| | NEUTROPHIL # | 9.9 H |
| | RDW | 15.5 H |
| CREATININE KINASE | Order Status | Final |
| | CREATININE KINAS | 34 L |
| | Order Status | Final |
| | CREATINE KINASE | 0.8& |

| | 06/26/11 04:45 | 11:10 |
|---|---|---|
| TROPONIN | 0.027& | <0.020& |

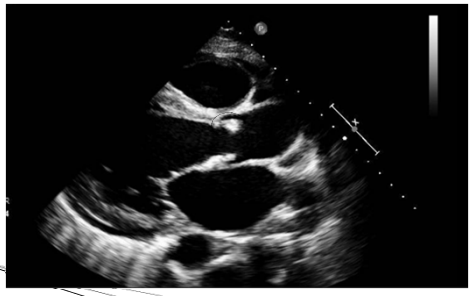
- Procalcitonin 0.14 (normal)
- Urine toxicology (with earlier admission)
  - Positive for methamphetamines and cannabinoids
- DAU-12
  - Positive for benzodiazepines and THC (marijuana)
- BNP 5002

- Given Lasix with 1300 ml urine output in ED
- Started on Rocephin and Azithromycin
- Plan for echocardiogram

## Echocardiogram

- Severely dilated left ventricular chamber
- EF 50 – 55%
- Normal LV systolic function. Abnormal LV diastolic function
- LV filling diastolic filling pattern is restrictive
- Thickened bicuspid aortic valve which probably is sessile vegetation.
- Freely mobile vegetation in the LV outflow tract
- Moderate to severe aortic insufficiency
- Moderate mitral insufficiency
- Mild tricuspid regurgitation
- Small circumferential pericardial effusion
- Severe pulmonary hypertension
- Left atrium moderately dilated
- Right atrium mild to moderately dilated

Thickened bicuspid aortic valve which probably is sessile vegetation.
Freely mobile vegetation in the LV outflow tract
Moderate to severe aortic insufficiency



Diagnosis

- Acute congestive heart failure
- Bacterial endocarditis of the aortic valve and probable mitral involvement
- Bicuspid aortic valve
- Pericardial effusion
- Mild renal insufficiency

- Diurese
- Start lisinopril
- Cardiothoracic surgery consult
- Infectious disease consult

- Admits to Heroin and LSD

## Infectious Disease Consult

- Streptococcus viridans aortic valve and infectious endocarditis
- Changed antibiotics to ceftriaxone
- PICC line placed. Ceftriaxone 2 grams IV every 24 hours for 6 weeks.

## Streptococcus viridans

- The organisms are most abundant in the mouth, and one member of the group, *S. mutans*, is the etiologic agent of dental caries. Others may be involved in other mouth or gingival infections.
- If they are introduced into the bloodstream, they have the potential of causing endocarditis, particularly in individuals with damaged heart valves. They are the most common causes of subacute bacterial endocarditis.
- Viridans streptococci have the unique ability to synthesize dextrans from glucose, which allows them to adhere to fibrin-platelet aggregates at damaged heart valves. This mechanism underlies their ability to cause subacute valvular heart disease following their introduction into the bloodstream (e.g., following tooth extraction).

Source: Wikipedia. August 29, 2011

## CV consult

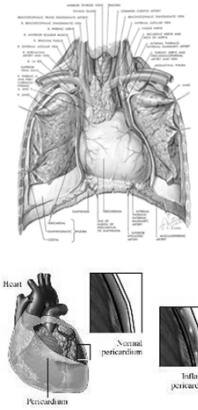
- Aortic Valve Replacement with #23 Trifecta Valve

## Patient faking seizure in ED

- https://www.youtube.com/watch?v=Q6sRyrB_UMA

## Pericarditis

- Inflammation of the pericardial sac
- Inflammation may cause fluid leak into the pericardial space
  - Tamponade is possible
  - Constrictive pericarditis
    - Scarring, thickening and fibrous of the pericardium




## Pericarditis

Causes

- Numerous causes
- Viral – most common
- Idiopathic
- Complication of HIV-AIDS – TB pericarditis
- Bacterial
  - Stab wound
  - Cardiac surgery
  - Pneumonia
  - Endocarditis
- Fungal – more common in immunosuppressed people
- Complication after AMI
- Metastasis
  - Esp in lung cancer (30 %)
- Radiation induced pericarditis
- Uremic pericarditis- chronic renal failure
- Medications
  - Procainamide
  - Hydralazine
  - INH (isoniazid)

## Pericarditis

Clinical Presentation

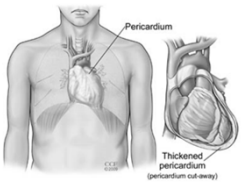
- Can mimic AMI
  - Chest pain
  - ST segment elevation
  - Have patient point to the exact location of the pain
- Pleuritic chest pain
  - Persistent, sharp, stabbing
  - Aggravated by inspiration and coughing
  - Localized to the retrosternal and left pericardium
    - With deep inspiration the diaphragm pulls on the inflamed pericardium causing the sharp pain that radiates
  - Pain is worse when recumbent
  - Pain maybe relieved by sitting upright and leaning forward
    - *Mohammed's Sign*
- Dyspnea*
  - Unrelated to exertion
  - Caused by the inability to take deep breaths
- Pericardial friction rub
  - Heard best left lower sternal border, 4t or 5th ICS
  - To heard best, pt should lean forward so the pericardium is closer to the chest wall
- Chills/Fever – may be antecedent
- SOB/Cough
- Weakness

Pain when recumbent
Dyspnea unrelated to exertion

## Constrictive Pericarditis

Clinical presentation

- Cardiac tamponade as evidenced by
  - True dyspnea and SOB
  - Pulmonary congestion
  - ↓ CO
  - Decreased tissue perfusion
  - Pericardial knock
    - High pitched, sharp, early diastolic sound heard best at lower LSB with patient leaning forward
  - PAP > 50 mmHG




Muffled heart sounds = tamponade
Pericardial knock = constrictive pericarditis

## Pericarditis

Diagnostics

- 12 Lead EKG – most definitive diagnostic test
- CXR – may have enlarged cardiac silhouette (water bottle heart)
- Echo
  - Possibly Pericardial effusion
  - Pericardial thickening with constrictive pericarditis
- LABs
  - Troponin, CK negative
  - ↑ WBC, Sed rate, C-reactive protein
  - Positive blood cultues if due to infection

## Pericarditis post AMI

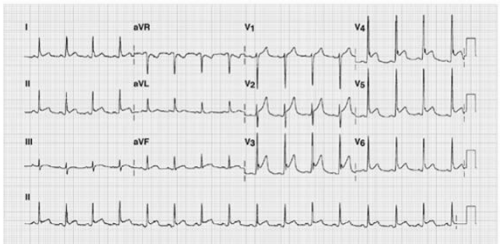
Acute: Pericardial inflammatory response

- Pericarditis occurring 2 – 7 days after AMI
- Related to the inflammation and healing process

Chronic: Dressler syndrome

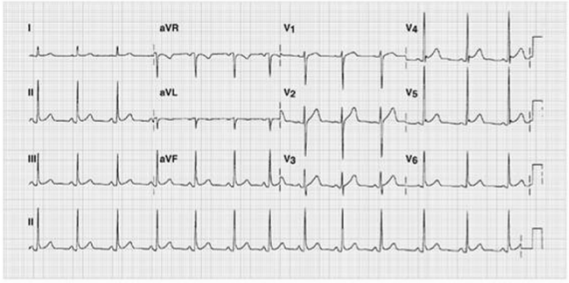
- Autoimmune response to myocardial necrosis
- Involves both the pleura and pericardium
- Occurs 2 – 4 weeks to several months post AMI

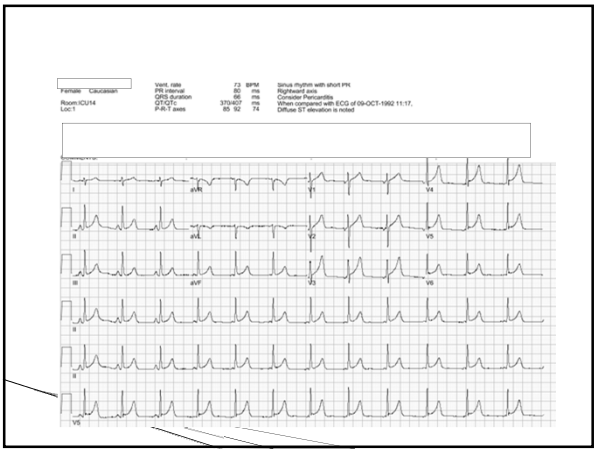
ECG 14-53 Pericarditis




- Diffuse ST elevation
- Scooping upwardly concave ST segment elevation in almost all leads except AVR
- No reciprocal ST depression except in AVR
- PR depression

ECG 14-54 Pericarditis



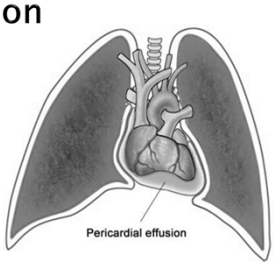
## Pericarditis

Clinical Management

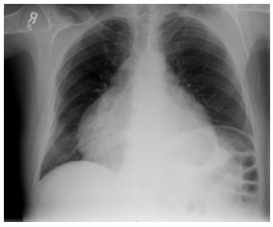
- Relieve pain
  - NSAIDs
  - Indomethacin & colchicines – for intolerance to NSAIDs
  - If unresponsive to NSAIDS
    - Narcotic analgesia
    - Short course of corticosteroids
- Treat cause
  - Antibiotics if bacterial
- If anticoagulants are given, assess for tamponade
- Pericarditis after AMI
  - NO corticosteroids or anti-inflammatory agents
    - Can cause rupture of the infarcted area
- Pericardiocentesis for tamponade
- Pericardial window for recurrent effusion
- Pericardiectomy – constrictive pericarditis

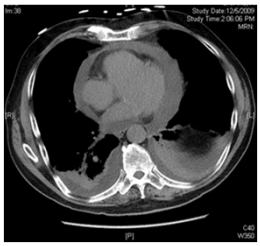
## Pericardial Effusion

- Accumulation of excessive fluid in the pericardial sac
- Sac normally holds 15 – 50 ml of fluid
- When excessive accumulation is slow, the pericardium can stretch.
- Cardiac tamponade occurs when the pericardium cannot stretch to accommodate the excess fluid




## Pericardial Effusion





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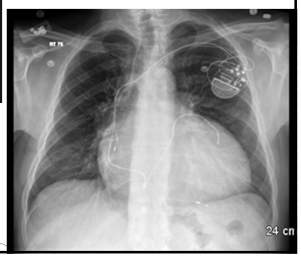
- Cardiomyopathy --- will see the pulmonary hiatus (vasculature)



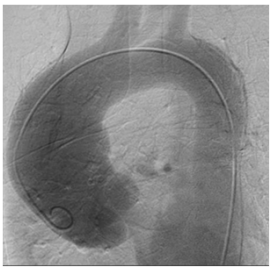
- Pericardial Effusion
- Water bottle
- (Above)

## Pericardial Effusion

Causes

- Initial causes of pericarditis
- Malignancy or radiation
- Autoimmune disease
- AMI
- Complications of cardiac surgery
- Drugs
- Chronic renal failure on dialysis
- Hypothyroidism
- Heart failure
- Blunt or penetrating chest trauma
- Retrograde extension of aortic dissection
- Iatrogenic causes
  - Myocardial perforation with invasive lines (pacers, central lines, angiogram)

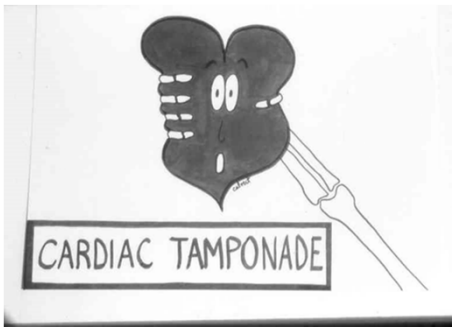
## Thoracic Type A Aneurysm



## Pericardial Effusion

Clinical Presentation

- Slow occurring effusions
  - Asymptomatic
  - Often undetected as the gradual stretching of pericardium doesn't compromise ventricular filling
- Rapid occurring effusions
  - Accumulation of pericardial fluid or buildup of a volume of pericardial fluid exceeding the pericardium's ability to expand
  - Leads to tamponade
  - Compromises ventricular filling
  - ↓ Cardiac output
  - 100 – 200 ml of extra fluid can elevate pericardial pressure from 1 – 5 mmHg to 30 mmHg or greater.



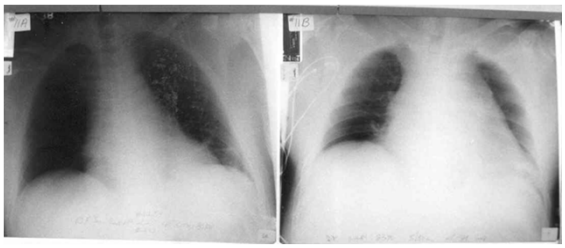

- Compression of the heart due to collection of fluid or blood in the pericardial space

## Cardiac Tamponade

Clinical Presentation

- Beck's Triad
  - Hypotension
  - Neck vein distention
  - Muffled heart sounds
- Hypotension
- Low urine output
- Rising & equalization of CVP & PAD
- Large *a* and *v* on PAOP "M"
- Falling $SVO_2$, CO/CI
- Widening mediastinum on CXR
- Cool extremities
- Tachycardia
- Pulses Paradox >10 mmHG
  - ↓ in systolic BP with inspiration

## Tamponade post Cardiac Surgery



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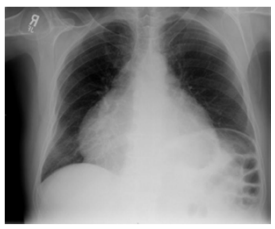
## Cardiac Tamponade

Clinical Management

- Closed Pericardiocentesis (preferred)
- Open Surgical Pericardiocentesis
  - Creation of a pericardial window to drain pericardium



Pericardial Effusion

Post pericardial window for pericardial effusion

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| | Myocarditis | Endocarditis | Pericarditis |
|---|---|---|---|
| Definition | | | |
| Heart Sounds | | | |
| Symptoms | | | |

| | Myocarditis | Endocarditis | Pericarditis |
|---|---|---|---|
| Definition | Inflammation of the heart muscle | Inflammation of the lining and valves | Inflammation of the pericardial sac<br><br>Constrictive – signs of tamponade |
| Heart Sounds | Distant<br>S3, S4<br>Pericardial friction rub | New or changed murmur<br>S3<br>Pericardial rub | Pericardial friction rub – Pericardial knock<br>best heard leaning forward |
| Symptoms | Wide variance from flu like to HF<br>Ventricular arrhythmias/ blocks<br><br>Bedrest– no exercise for several months | Embolic events<br>Osler's nodes:<br>Janeway lesions:<br>Roth's spots:<br><br>Prophylaxis | Recumbent pain<br>Pleuritic chest pain<br>Can mimic AMI – diffuse ST ↑, no reciprocal or Q waves |