Valvular Heart Disease
Other Cardiac Issues
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CMC–CSC
Test Blueprint

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

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 tricuspid valves
- Prevents regurgitation

Papillary Muscle Rupture
Acute Mitral Regurgitation

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

Clinical Manifestations
- Hypotension
- Tachycardia
- Dyspnea
- Crackles
- S3
- New holosystolic at the apex

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

Symptoms

- Hypotension
- Tachycardia
- Tachypnea
- New holosystolic murmur at lower left sternal border
- Thrill
- Pulmonary congestion
- Syncope

↓ RAP, PAP
↓ Large v on PAOP waveform
↓ SvO₂
↓ 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

Cause

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

Ventricular Septal Defects

Clinical features

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

Normal Heart & Heart with VSD

Holosystolic murmur

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 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
- 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 S1 & S4
- Right ventricular heave at sternal
- CXR
  - Left atrial and RV enlargement
- Pulmonary congestion
- EKG
  - LA enlargement: Notched P waves P1Mitral
  - Right ventricular hypertrophy
  - Fall R waves V1 & 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 l 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

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**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

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**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

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**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

**Endocarditis risk is RARE**

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**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

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**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

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

- 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

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

Pathophysiology

1. Volume overload leads to compensatory mechanisms
   - ↑ LV 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

Aortic Regurgitation/Insufficiency

- 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

Post op AVR for AR

- Due to dilated ventricle use
  - IV vasodilators
  - Inotropic support to promote ventricular emptying
    - 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=zt13cc2EOmM

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 stenosed or nonvalvular aortic valve
- Native aortic annulus size < 18 mm (as a native valve), and no greater than 25 mm – size dependent in cases available currently
- Severe native aortic regurgitation (2+–)

**Relative Exclusion Criteria**
- Evidence of MR within 6 weeks of valve placement
- Hemodynamic or respiratory instability requiring inotropic support, mechanical ventilation, or mechanical heart assistance within 30 days
- Hypotrophic cardiomyopathy with or without obstruction
- Left ventricular ejection fraction < 25 percent
- Severe pulmonary hypertension and right ventricular dysfunction
- A known contraindication or hypersensitivity to all anticoagulant regimens or inability to be anticoagulated for the study procedure
- Dual antiplatelet therapy is required for at least 3 months
- Renal insufficiency (e.g., 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 obstructing atherosclerosis
- Estimated life expectancy <12 months due to noncardiac comorbid conditions
- Severe mitral regurgitation
- Significant aortic disease, including the following abnormalities
  - Could be non-1
  - Needs to be assessed

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
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

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=C6mTmaP9Lw

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

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

<table>
<thead>
<tr>
<th></th>
<th>Tissue</th>
<th>Mechanical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Over 65 yo</td>
<td>Under 65</td>
</tr>
<tr>
<td>Longevity</td>
<td>10-15 years</td>
<td>Potentially Lifetime</td>
</tr>
<tr>
<td>Anticoagulation</td>
<td>Aspirin lifelong Warfarin - 3 months??? Warfarin lifelong</td>
<td></td>
</tr>
<tr>
<td>Reoperation risk</td>
<td>Patient dependent</td>
<td>As low as 1% risk lifetime</td>
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</tbody>
</table>

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%

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
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)

Surgery indicated for those patients with acute infective valve endocarditis and life-threatening heart failure or cardiogenic shock.

Medical therapy is first line treatment

Infective Valve Endocarditis

The left ventricle is normal size in which valvular disease?

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

Another Patient. MRSA

What is SBE?
Subacute bacterial endocarditis

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

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
Valvular Pearls

<table>
<thead>
<tr>
<th>Heart Sounds</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mid diastolic murmur at site of S3, S4</td>
<td>Midsystolic murmur high pitched</td>
<td>Widely split S2 at right sternal border</td>
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<td></td>
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<table>
<thead>
<tr>
<th>Symptoms</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dynpnea</td>
<td>Pulmonary Hypertension</td>
<td>Pulmonary symptoms</td>
<td>Syncope</td>
<td>Dyspnea, Angina</td>
</tr>
</tbody>
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<table>
<thead>
<tr>
<th>Atrial size</th>
<th>MS</th>
<th>MR</th>
<th>AS</th>
<th>AR/AI</th>
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<tbody>
<tr>
<td>LA enlarged</td>
<td>LV normal</td>
<td></td>
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<thead>
<tr>
<th>Ventricular Size</th>
<th>MS</th>
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<tbody>
<tr>
<td>LV enlarged</td>
<td></td>
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Valvular Surgery Pearls

**Aortic Valve**

<table>
<thead>
<tr>
<th>Aortic Stenosis</th>
<th>Aortic Regurgitation</th>
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<tbody>
<tr>
<td>Preop</td>
<td>LV hypertrophy</td>
</tr>
<tr>
<td>Post op</td>
<td>LV may not anticipate in SVR and continue to pump hard</td>
</tr>
</tbody>
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**Mitral Valve**

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<th>Mitral Stenosis</th>
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<tbody>
<tr>
<td>Preop</td>
<td>NS LV function</td>
</tr>
<tr>
<td>Post op</td>
<td>Assess pulmonary hypertension (PVR) Dobutamine or Milrinone - Norepinephrine to ↓ contractility of RV &amp; ↓ PVR Fluids</td>
</tr>
</tbody>
</table>

**Myocarditis**

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

**Acute Inflammatory Disease**

Cardiac “itis”

- Myocarditis
- Endocarditis
- Pericarditis
Myocarditis

Causes

- Infectious
  - Bacterial
  - Spirochetal
  - Viral
  - Protozoal
  - Parasitic
  - Rickettsial
- Viral
  - Human immunodeficiency virus
  - Human herpesvirus 6
  - Human cytomegalovirus
  - Epstein-Barr
- Allergic
  - Environmental
  - Food

Toxic Myocarditis

- Drugs
  - Cocaine
  - Amphetamines
  - Antifibrinolytics
  - Anticoagulants
  - Calcium channel blockers
  - Beta blockers
- Heavy metals
  - Lead
  - Mercury
  - Arsenic

Immune Mediated

- Allergens
- Autoantibodies
- Autoantigens
- Drugs
- Systemic lupus erythematosus
- Sarcoidosis

History of recent flu-like symptoms

- Fever
- Arthralgia (joint pain)
- Fatigue
- Chest pain

Labs

- Troponin I
- CK MB
- Leukocytosis
- Cardiac immunoglobulin antibody titer 1:40 or greater

Endomyocardial biopsy for definitive diagnosis

Heart Sounds

- Distant
- S3 or S4
- Pericardial friction rub

ERG

- Ventricular arrhythmias
- Heart block
- Diffuse ST segment changes
- T wave changes

CXR

- Cardiomegaly
- Pulmonary congestion
- Pleural or pericardial effusion

Wide variance in symptoms – minimal symptoms to fulminant Heart Failure

Myocarditis

Phases

- Acute: Day 0 – 3
  - Viral infection
  - Myocyte change
  - Cardiomyocyte 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

Myocarditis

Clinical Presentation

- History of recent flu-like symptoms
- Fever
- Arthralgia (joint pain)
- Fatigue
- Chest pain
- Labs
  - Troponin I
  - CK MB
  - Leukocytosis
  - Cardiac immunoglobulin antibody titer 1:40 or greater

Endomyocardial biopsy for definitive diagnosis

Heart Sounds

- Distant
- S3 or S4
- Pericardial friction rub

ERG

- Ventricular arrhythmias
- Heart block
- Diffuse ST segment changes
- T wave changes

CXR

- Cardiomegaly
- Pulmonary congestion
- Pleural or pericardial effusion

Observe for pericarditis and tamponade

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

**Clinical Presentation**

- May be nonspecific
  - Fever
  - Diaphoresis
  - Weight loss
  - Myalgia (muscle pain)
  - Night sweats
  - 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
  - ↑ ESR
  - Anemia
  - Leukosytosis
  - ↑ C-reactive protein
  - Abnormal urinalysis
  - ↑ WBC

- Echocardiogram
  - Intracardiac vegetation
  - Dysfunctional valves

### Complications

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

**Source**: Moser & Riegal. Cardiac Nursing 2008

### Endocarditis

**Treatment**

- Antimicrobial therapy
- Eradication 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 hemodynamically stable
**Endocarditis**

**Prophylaxis**

- Prevention is essential in high risk groups
- Prophylactic Antibiotics
  - Amoxicillin
  - Ampicillin
  - Clindamycin
  - Azithromycin
  - Cephalexin
  - 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
- SpO2 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 severe hydropherosis 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

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

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

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).

Patient faking seizure in ED

- [https://www.youtube.com/watch?v=Q6sRyr8_UmA](https://www.youtube.com/watch?v=Q6sRyr8_UmA)

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)

**Clinical Presentation**
- Dyspnea
  - Unrelated to exertion
  - Caused by the inability to take deep breaths
- Pericardial friction rub
  - Heard best left lower sternal border, 4th or 5th ICS
  - To hear best, pt should lean forward so the pericardium is closer to the chest wall
- Chills/Fever – may be antecedent
- SOB/Cough
- Weakness

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
- Pleuritic chest pain
  - Persistent, sharp, stabbing
  - Aggravated by inspiration and deep breathing
  - 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 may be relieved by sitting upright and leaning forward
  - Mohammed’s sign

- 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
- Pericardial knock
  - Possible Pericardial effusion
  - Possible Pericardial thickening with constrictive pericarditis

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 cultures 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

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 14-03 Pericarditis

ECG 14-04 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

- 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

Thoracic Type A Aneurysm

- Blunt or penetrating chest trauma
- Retrograde extension of aortic dissection
- Iatrogenic causes
  - Myocardial perforation with invasive lines (pacers, central lines, angiogram)
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.

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

Cardiac Tamponade

Clinical Presentation

- Beck’s Triad
  - Hypotension
  - Low urine output
  - Rising & equalization of CVP & PAD
- Large a and v on PAOP “M”
- Falling SVO₂, CO/CI
- Widening mediastinum on CXR
- Cool extremities
- Tachycardia
- Pulses Paradox >10 mmHG
  - ▼ in systolic BP with inspiration

Tamponade post Cardiac Surgery

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
<table>
<thead>
<tr>
<th></th>
<th>Myocarditis</th>
<th>Endocarditis</th>
<th>Pericarditis</th>
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<tbody>
<tr>
<td><strong>Definition</strong></td>
<td>Inflammation of the heart muscle</td>
<td>Inflammation of the lining and valves</td>
<td>Inflammation of the pericardial sac</td>
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<td>Constrictive – signs of tamponade</td>
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<tr>
<td><strong>Heart Sounds</strong></td>
<td>Distant S3, S4 Pericardial friction rub</td>
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<td></td>
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<td>best heard leaning forward</td>
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<tr>
<td><strong>Symptoms</strong></td>
<td>Wide variance from flu like to HF</td>
<td>Embolic events Ossian’s nodes: Janeway lesions:</td>
<td>Recumbent pain</td>
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<td>Ventricular arrhythmias/ blocks</td>
<td>Roth’s spots: Prophylaxis</td>
<td>Pneumonic chest pain</td>
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<td></td>
<td>Bedrest: no exercise for several months</td>
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<td>Can mimic AMI – diffuse ST +, no reciprocal or Q</td>
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<td>waves</td>
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