Cardiology I: Hypertension, Heart Failure, Endocarditis, Pericarditis & Effusion

Sally M. Hur, PA-C
Instructor,
UMDNJ PANCE/PANRE Review Course
(becoming Rutgers University July 1, 2013)

Progress of JNC 8

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Hypertension

- Essential Hypertension
- Secondary Hypertension
- Malignant Hypertension
- Hypotension
**Essential Hypertension**

- 50 Million Americans (1 in 4)
- Only 70% are aware
- Only 50% are treated
- Only 25% are treated adequately

**Classification**

- **Normal** <120 <80
- **Pre-HTN** 120-139 80-89
- **Stage 1 HTN** 140-159 90-99
- **Stage 2 HTN** ≥160 ≥100

*Treatment Goal is <140/90
*Target is <130/80 in DM & Renal Disease

**Diagnosis**

- Average of:
  - Two or more readings
  - on Two or more different occasions
  - No smoking or caffeine 30 minutes prior
  - Up to 30% have "white-coat HTN" home +/- ambulatory BP monitor
**Work Up**
- Assess co-morbidities
- Look for end-organ damage
  - EKG
  - U/A, BUN/CR, K^+
  - Retinopathy
  - CVA/TIA/Peripheral Artery Disease
- Look for reversible cause: (2^o HTN)
  - CBC, lipids, Ca++

**HTN Co-Morbidities**
- Diabetes
- Dyslipidemia
- Smoking
- Obesity (BMI ≥ 30)
- Inactivity
- Age (>55♂ >65♀)
- Fam Hx of premature cardiovascular disease

**Secondary Hypertension**
- Aldosteronism
- Chronic kidney disease
- Renovascular disease
- Pheochromocytoma
- Cushing’s syndrome
- Coarctation of Aorta
- Medication induced
- Sleep Apnea
Secondary Hypertension

- Less than 5% of all HTN patients.
- Red Flags:
  - HTN starts at early age (< 25yrs) w/o FamHx
  - HTN first develops > 50
  - Previously controlled HTN, now refractory

Aldosteronism

- Presents classically as HTN with hypokalemia (~30% have normal K+)
- May be 5-15% of pts DX with essential HTN
- Best screening test: aldosterone/renin ratio > 25
  - Urine K+ > 40 mEq/L
- Diagnose with CT or MRI of adrenal glands: adrenal adenoma or hyperplasia

Chronic Kidney Disease

- Renal parenchymal disease is most common: glomerular, tubular interstitial, polycystic dz
- Diabetic Nephropathy
- Tests: BUN/Cr, U/A
- Diagnose with Renal biopsy
**Renal artery stenosis**
- Causes 1-2% of HTN
- HTN > 50 y/o with bruits, PAD, refractory HTN  
  (suspect: renal artery atherosclerosis)
- HTN < 30, female with renal artery bruits  
  (suspect: fibromuscular dysplasia (FMD))
- Dx: Arteriography is “Gold Standard”
- Tx: Stent for young, Meds for most older pts.

**Cushing’s Syndrome**
- Less common cause - Glucocorticoid excess
- PE: Truncal obesity with muscle atrophy, striae, acne, hyper-pigmented skin
- Labs: Glucose (hyperglycemia)  
  K (hypokalemia)
- Dx: 24 hr urine free cortisol >3x normal  
  MRI pituitary (>50%), CT lungs/ abdomen

**Pheochromocytoma**
- Rare: causes <0.1% of HTN
- Labile HTN, headaches, tachycardia, diaphoresis
- Labs: plasma/24hr urine metanephrines
- Dx: CT/ MRI of Adrenals (90% sensitivity)  
  Laparoscopic surgical resection  
  Alpha adrenergic blockade (phenoxybenzamine)  
  or CCBs
**Coarctation of Aorta**
- Uncommon cause of HTN
- Usually young, cold feet, decreased or no femoral pulses, high BP arms with low BP legs.
- Narrowing of aortic arch - distal to left subclavian
- Dx: Echocardiography/doppler (MRI/CT useful)
- Tx: stenting or surgery

**Medication Induced**
- Oral Contraceptive - sodium retention and angiotensinogen
- NSAID - Na++ retention, renal vasoconstriction
- Corticosteroids - like Cushing's
- ETOH >2/day- activates sympathetic system
- Sympathomimetics – cold/diet meds, cocaine...
- Erythropoietin

**Sleep Apnea**
- OSA and HTN linked epidemiologically
- Untreated OSA predisposes to new HTN
- Treating OSA lowers BP
- Possible mechanisms:
  - sympathetic activation
  - elevated angiotensin II and aldosterone
  - oxidative and inflammatory stress
**Treatment**
- Treatment Goal is <140/90
- Target is <130/80 in DM & Renal Disease
- All patients must use lifestyle modification
- Most patients will need two medications
- Consider: DM, Renal Disease, CHF, post MI, High CVD risk, CVA

**Pre HTN: 120-139/80-89**
- Weight Reduction: Target BMI = 18.5-24.9
- DASH Diet: Fruit, veggies, low fat dairy
  - 8-14 point drop in SBP
- Sodium reduction: <2.4G sodium/day
  - 2-8 point drop in SBP
- Aerobic exercise: 30min/daily (ideal)
  - 4-9 point drop in SBP
- Decreased ETOH: <2 drinks/daily
  - 2-4 point drop in SBP

**Stage 1 HTN: 140-159/90-99**
- Life Style Modification
- Life Style Modification alone if low risk (2%)
- Thiazide diuretics for others
- May consider ACEI, ARB, BB, CCB, combo
- Evaluate for “compelling indications”
Treatment: Stage 2 HTN

Stage 2 HTN: ≥160/≥100

- Life Style Modification WITH medications
- Two drugs needed for MOST
- Thiazide diuretic with: ACEI, ARB, BB, CCB
- Evaluate for "compelling indications"

Treatment: Compelling Indications

Diabetes: Thiazide diuretic (Thiaz)
ACE inhibitor (ACEI)
Angiotensin receptor blocker (ARB)
Beta Blocker (BB)
Calcium channel blocker (CCB)

Chronic Renal Disease:
ACEI
ARB

Treatment: Compelling Indications

CHF: Thiazides, β blockers, ACE I, ARB,
Aldosterone Antagonist (spironolactone)

Post MI: β blockers, ACE I, Aldosterone
Antagonist

High CVD risk: Thiazides, β blockers, ACE I,
Calcium Channel blockers

CVA prevent: Thiazides, ACE I
**Malignant Hypertension**

**Hypertensive Urgency:**
- No Sx, BP > 220/125
- Oral meds with partial reduction in BP = OK

**Hypertensive Emergency:**
- Elevated BP with End Organ Damage

**Malignant Hypertension:**
- Encephalopathy or Nephropathy w/ papilledema

**Malignant Hypertension**

**Hypertensive Emergency:**
- Elevated BP with End Organ Damage
  - BP must be lowered within 1 hour to avoid death
    - Hypertensive Encephalopathy/ICH
    - Hypertensive Nephropathy
    - Pulmonary edema/ MI/ unstable angina
    - Aortic dissection
    - Eclampsia – in pregnancy

**Parenteral meds to rapidly lower BP**
- Nitroprusside: controlled (agent of choice)
- Nitroglycerin: less control (useful w/ ischemia)
- Labetalol: potent α and β-blockade

**Too rapidly > cerebral, coronary, & renal ischemia**
- Reduce pressure 25% in 1st 1-2 hrs
- Decrease additional 15% over next 3-12 hrs
- Achieve ~160/110 by 12 hrs
A 32-year old male with a history of Type 2 DM has a blood pressure averaging 135/85 on several measurements. What is the best intervention for this patient?

1. Diet and exercise
2. Hydrochlorothiazide
3. No treatment necessary

Orthostatic Hypotension

- Positional syncope or near-syncope
- Lying to standing or motionless standing
- Sx: lightheaded, weakness, visual disturbance
- Dx: Orthostatic BP drop > 20 mm Hg systolic
- Causes: often elderly, DM, BP meds, Parkinson’s, volume loss: blood loss, diuretic, N/V
- Cardiogenic Shock
1 in 4 Americans have HTN
95% of all cases are Essential (or primary) HTN
Co-morbidities
Primary Aldosteronism: most common treatable cause of HTN
Chronic kidney disease - most common cause of secondary HTN
Treatment Goal is <140/90, Target is <130/80 in DM & Renal Disease
Compelling indications: CHF, post MI, High CVD risk, DM, Renal disease, CVA

Case 1
73 year old man presents with 2 day history of shortness of breath on exertion and paroxysmal nocturnal dyspnea

What is CHF
Heart failure is defined as the poor contraction and/or relaxation of the heart resulting in insufficient blood volume to adequately meet oxygen demand.
This defective pumping mechanism results in accumulation and redistribution of fluids.
**CHF: Symptoms and Signs**

- **Left Heart Failure**
  - Dyspnea on exertion
  - Orthopnea
  - Paroxysmal Nocturnal Dyspnea
  - Wheezing/Rales/Tachycardia

  *Non-specific* Presentations:
  - Cough, Syncope
  - Fatigue, Weakness, Confusion
  - Insomnia, Nocturia

- **Right Heart Failure**
  - Peripheral edema
  - Jugular-Venous Distention (JVD)
  - Gastro-intestinal
    - N/V/D/Pain complaints
    - Hepatic and bowel edema
    - Hepato-Jugular Reflex (HJR)
    - Ascites

**CHF: Symptoms and Signs**

- Peripheral edema
- Jugular-Venous Distention (JVD)
- Gastro-intestinal
  - N/V/D/Pain complaints
  - Hepatic and bowel edema
  - Hepato-Jugular Reflex (HJR)
  - Ascites

**Case 2:**
69 year old woman with HTN and prior MI's presents with 2 day history of dyspnea on exertion and bilateral edema
In US the most common cause of CHF:
Coronary Artery Disease
- Precipitated by HTN (more in US), DM, hyperlipidemia
- With myocardial injury
- Leading to decreased myocardial function

Common causes of New Acute CHF:
- Massive MI: >40% = cardiogenic shock
- Valve failure: ruptured chordae tendineae of mitral valve
- Myocarditis
- Thyrotoxicosis/ thyroid storm

Factors Determining Performance:
- Preload: How full is the ventricle before it squeezes?
- Afterload: What is ventricle pushing against?
- Myocardial Contractility: The force that the muscle can exert
- Heart Rate: too fast or too slow = increased demand
Measures of Cardiac performance:
- Arterial Blood Pressure
- Cardiac Output (SV x HR)
- Cardiac Index (CO/BSA)
- Ejection Fraction (100 x SV/EDV)

Ejection Fraction
In most cases - Ejection fraction refers to left ventricular ejection fraction.
- 50 - 70% = Normal
- 36 - 49% = Below Normal
- 35 - 40% = may confirm diagnosis of systolic heart failure
- <35% - patient may be at risk of life-threatening arrhythmias

Multi-factorial Syndrome
Heart performance is poor: cannot provide sufficient blood flow to meet demands:
All of these inter-react:
- Preload
- Afterload
- Contractility
- Rate
- Decreased pump function
- Altered Filling
- Increased Demand
Pathophysiology

Mechanical abnormality:
- Valve dysfunction, shunts

Rate/rhythm disorders:
- Bradyarrhythmia/tachyarrhythmia

High Output States:
- Thyrotoxicosis, fever, chronic anemia, blood loss

Pathophysiology

Impaired Systolic Function
Decreased Contractibility (Low EF)
- Ischemic Damage (MI)
- Chronic Pressure Overload (HTN, AS)
- Chronic Volume Overload (AR)
- Dilated Cardiomyopathy

Pathophysiology

Impaired Diastolic Function
- Poor filling (Decreased Preload)
- Myocardial Hypertrophy
  - 1° Hypertrophic Cardiomyopathy
  - 2° Hypertension
- Ischemic Fibrosis (post MI)
- Restrictive Cardiomyopathy
Impaired Systolic Function
- Dilated Cardiomyopathy

Impaired Diastolic Function
- Restrictive Cardiomyopathy
- Hypertrophic Cardiomyopathy

Causes ~25% of all CHF cases
- African American > whites; men > women
- Cause - usually idiopathic
  - Alcoholic Cardiomyopathy
  - Peri-partum Cardiomyopathy
  - Toxins? infections?
- Poor EF, large heart
- Thin, dysfunctional LV Wall
- H/O thromboembolic disease – anticoagulate

Right heart failure predominates
- Pulmonary HTN present
- Usually caused by - Amyloidosis
  - Sarcoidosis
  - Scleroderma
  - Loffler’s syndrome
- Normal EF, normal heart size, large atria
- Normal LV wall, early diastolic filling
**Hypertrophic Cardiomyopathy**
- Often present in early adulthood
- Cause in young: genetic - IHSS (HOCM)
- Cause in elderly: chronic HTN
- Very good EF, large heart
- Thick LV Wall (asymmetric hypertrophy)
- Decreased diastolic filling
- Bisferins Pulse
- Avoid intense physical activity → reduce risk of sudden death

**Taku-Tsubo Cardiomyopathy**
- "Broken Heart Syndrome"
  - LV apical ballooning following a high catecholamine stress.
  - Presents with Acute chest pain or SOB similar to an acute anterior myocardial infarction, but has normal coronaries on catheterization.
  - Most patients recover completely
Common Presentation

Acute Decompensation of “Stable” CHF
- A Disease of the Elderly
  - 75% of case in persons over 65
- Caused by ischemic cardiomyopathy

Precipitating Factors

Acute Decompensation of “Stable” CHF
- Altered cardiovascular condition:
  - Infarction, Arrhythmia, Worsened HTN
- New metabolic stress:
  - Fever, Infection, Blood loss, Anemia
- Medication discontinued
  - non-compliance with medications?

Precipitating Factors

Acute Decompensation of “Stable” CHF
- Dietary indiscretion
  - salty food, alcohol
- Iatrogenic volume overload
  - transfusion, IV NS
- New medication initiated
  - NSAID, B-Blocker, CCB
CHF: Studies & Labs

Chest X-ray
- Cardiac silhouette enlargement

If Acute:
- Enlarged pulmonary veins
- Upper lobe redistribution of fluid
- Interstitial edema with central haziness
- Peri-hilar or patchy peripheral infiltrates
- Pleural effusions are common (R>L)

http://www.radiologyassistant.nl/en/p4c132f36513d4

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CHF: Studies & Labs

**Electrocardiogram (EKG)**

- Evaluate rate and rhythm
- Evaluate for acute or prior MI
- Evaluate for Ventricular Hypertrophy

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**CHF: Studies & Labs**

**Echocardiography:** The most useful and practical test to diagnose and evaluate suspected CHF

- Determine Ejection Fraction
- LV function and wall motion (?prior MI)
- Evaluate valve function
- Dx: dilated, restrictive, or hypertrophic cardiomyopathy
**CHF: Studies & Labs**

**Angiography**

Angiogram: invasive but...
In presence of evidence of ischemia...
Patients benefit from diagnosis and intervention (angioplasty and/or stenting).

**CHF: Studies & Labs**

Labs:
- BNP (B-type Natriuretic Peptide)
  - released by heart muscle in fluid overload
  - sensitive & specific: <100 rules out CHF
- CPK and Troponin (MI)
- CBC (anemia)
- Electrolytes
- Renal Function (BUN/ Creatinine)

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**Prevention**

- **Control HTN:**
  - focus on systolic pressure
  - reduces incidence of heart failure by 50%

- **Prevent first MI**

- **Post MI:**
  - β-Blocker, lower cholesterol,
  - anti-thrombotic, revascularization

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Treatment

Determined by Stage of Heart Failure

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<td>(Pre CHF - at risk)</td>
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<tr>
<td>Stage B</td>
<td>(asymptomatic)</td>
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<tr>
<td>Stage C</td>
<td>(symptomatic)</td>
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<tr>
<td>Stage D</td>
<td>(marked sx at rest)</td>
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*(NYHA depended on patient reported symptoms)*

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Treatment

Stage A: Pre CHF (ACC/AHA):
- No structural heart abnormality
- No signs or symptoms of heart failure
- Presence of risk factors:
  - HTN, CAD, DM, ETOH Abuse,
  - Rheumatic fever, FHx of cardiomyopathy

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Treatment

Stage A (ACC/AHA):
- Control HTN (focus on systolic pressure)
- Lower cholesterol
- ACE inhibitors (for appropriate patients)
- Lifestyle Changes:
  - Smoking cessation
  - Exercise
  - Decrease ETOH

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Stage B: Early CHF (ACC/AHA)
- Structural heart abnormality present
- No signs or symptoms of heart failure
- Example:
  - LV Hypertrophy or Fibrosis, Valvular disease
  - LV Dilation or Hypocontractility, prior MI

Treatment
Stage B (ACC/AHA):
- All - Stage A measures
  - *B*-Blockers (for appropriate patients)
  - ACE inhibitors (for appropriate patients)

Stage C – CHF (ACC/AHA)
- Underlying structural heart disease
- Current/prior signs or symptoms of heart failure
- Examples: LV Dysfunction managed w/meds
Treatment

**Stage C** (ACC/AHA)
- All - Stage A measures
  - Medications for routine use:
    - Diuretics
    - ACE inhibitors
    - β- Blockers
    - Digitalis
    - Dietary salt restriction

**Stage D** (ACC/AHA)
- Advanced structural heart disease
- Marked symptoms of heart failure at rest
- Example:
  - Patients awaiting transplant
  - Pts require continuous IV meds
  - Pts require mechanical assist device

**Stage D** (ACC/AHA)
- All - Stage A thru C measures
- Mechanical Assist devices
- Transplant
- Continuous IV infusion of inotropic medication
**Pulmonary Edema/CHF**
- Loop Diuretic: Lasix (double current po dose)
- SL/IV NTG: ensure normotensive
- O2, CPAP
- ACE inhibitor
- Treat Cause: MI? HTN?

**Cardiogenic Shock**

Shock caused by the Heart's diminished CO
- “Pump failure” 2° to MI, CHF, Cardiomyopathy
- Dysrhythmia: Tachy or Brady
- Acute valve dysfunction - (regurgitant)
- Rupture of ventricular septum or wall

**Cardiogenic Shock**

General Treatment Measures
- Continuous IV infusion of inotropic medication
  - **Dopamine** is pressor agent of choice
- Mechanical assist devices
  - Intra-Aortic balloon pump
  - maintains adequate perfusion and circulation
- Transplant?
Cycle of CHF

- Cardiac output decreases.
- Neurohormonal mechanisms attempt to increase perfusion of the kidneys. Renin is released: BP is raised & fluid retained
  - HTN increases afterload (decreases EF) and work (increases demand)
  - Volume overload decreases contractility (decreases EF)
- Low CO causes catecholamine release:
  - Heart rate increases= less filling/preload (decreased EF) and increases work (increased demand)
  - BP increases= causing higher afterload (decreased EF) and increases work (increased demand)

Medications

Diuretics – (furosemide):
- Reduce peripheral vascular resistance
- Reduce plasma volume
- take off excess salt and fluid
- control fluid retention
- decrease volume overload

Negative: electrolyte depletion, hypotension, increase neurohormonal activation…

Medications

ACE inhibitors: Interfere in the renin-angiotensin system (RAS)
- Block angiotensin - a potent vasoconstrictor
- Decrease aldosterone mediated sodium retention
- Slow myocardial remodeling and fibrosis (also ARBs and spironolactone)
Medications

β-Blockers: Interfere w/ changes caused by catecholamine release
- Slow heart rate
- Decrease afterload/blood pressure
- Decrease cardiac output
- Cause rise in EF and reduction in LV size

Medications

Digitalis: Controls symptoms of decreased contractility
- Inotropic effects
- Controls rate in afib
- Useful with CHF Symptoms despite ACE and β-Blockade
Negative: dig toxicity, arrhythmia

Prognosis

5 year survival: <50% after acute onset of CHF. Only 35% of men and 50% of women are alive after 5 years.
Worse prognosis: older patients, men, pts with CAD, pts with reduced EF

Class D (IV): > 30% annual mortality
Class B-C (I-II): >5% annual mortality
A 73-year-old presents with a 2-day history of shortness of breath on exertion and paroxysmal nocturnal dyspnea. What is the most likely underlying diagnosis?

1. Coronary artery disease
2. Pheochromocytoma
3. Sarcoidosis
4. Systemic infection

Endocarditis, Pericarditis & Effusion

- Infective Endocarditis
- Acute Pericarditis
- Pericardial Effusion
- Cardiac Tamponade
Infective Endocarditis
- Preexisting Heart Lesion (Valvular)
- Fever (>38°C)
- New Heart Murmur
- Positive Blood Cultures
- Evidence of Septic Emboli
- Echocardiography w/ Vegetation

Infective Endocarditis
Classic Signs:
- Petechia - palate, conjunctiva, subungual
- Subungual splinter hemorrhages
- Osler node: (painful lesion- finger/toes/feet)
- Janeway lesion: painless red lesion palm/sole
- Roth spot: exudative retinal lesion (25%)

Which Lesion is painful?
- Osler
- Janeway

Osler = Ouch
Acute Infective Endocarditis
- Acute bacterial infection typically *Staph. aureus*.
- Rapid onset of high fevers, rigors.
- New regurgitant murmur.
- Labs: leukocytosis & positive blood cultures.
- Sx: 2^o to emboli to lungs, kidneys, joints, bones: cough, CP, back/flank pain, arthritis.
- Rapid deterioration with CHF (70%).

Subacute Infective Endocarditis
- Slow insidious bacterial infection of heart valve.
- Typically *Strep. viridans*.
- Patients have weeks of constitutional Sx:
  - nausea, vomiting, fatigue, and malaise.
  - (-) fever: w/ elderly, CHF or renal patients.
- Regurgitant murmur.
- Labs: anemia of chronic dz, normal WBC.

Infective Endocarditis
Prosthetic Valve Disease:
- Fever or Prolonged Constitutional Sx.
- Agent = (early) soon after valve implant:
  - Staph, Gram neg., Fungi.
- Agent = (late) after 2 months:
  - Strep and Staph.
- Treatment = 6 weeks of treatment:
  - Staph = 6wk Vanco + Rifampin + 2wkGent.
**Infective Endocarditis**

**Injection Drug Users**

- All febrile IDU’s should be evaluated for IE
- Agent = 60% Staph aureus
  - Also Enterocci/Streptococci
- Treatment = 2-4 wk Depends on the Agent

**Infective Endocarditis**

**Bug Specific Treatment**

- Empiric = Vancomycin + Ceftriaxone
- Strep. viridans
  - 4 wk PenG or 2 wk PenG + Gent
  - 4 wk Ceftriaxone or Vancomycin
- Staph. aureus
  - 6 wk Nafcillin or Oxacillin
  - 6 wk Vancomycin
- HACEK: Haemophilus, Actinobacillus, Cardobacterium, Eikenella, Kingella
  - 4-6 wk Ceftriaxone

**Infective Endocarditis**

**Lesions Requiring Prophylaxis**

**High Risk**

- Prosthetic Valves
- Prior Infective Endocarditis
- Cyanotic Congenital Heart Disease

**Moderate Risk**

- Rheumatic (or other acquired) valve disease
- Hypertrophic Cardiomyopathy
- Mitral valve prolapse WITH regurgitation
Lesions NOT Requiring Prophylaxis:
- ASD/ VSD/ PDA - post repair
- CABG
- MVP without regurgitation
- Rheumatic fever without valve dysfunction
- Previous Kawasaki’s/ Pacemakers/ AICD-Defibrillators
- Hypertrophic Cardiomyopathy*
- Mitral valve prolapse WITH regurgitation

Procedures Requiring Prophylaxis:
- Dental: Extraction/ Root Canal/ Tonsillectomy
- GI: Surgery/ ERCP/ Colonoscopy w/ Biopsy
- GU: Prostate surgery/ cystoscopy

Procedures NOT Requiring Prophylaxis:
- Respiratory: Flexible Bronchoscopy*
- GI: Endoscopy w/ Biopsy*
- GU: Hysterectomy, C-section/ Vaginal delivery*
- Dental fillings/ local injection/ fluoride/ orthodontic adjustment
- Respiratory: flex bronchoscopy/ tympanostomy/ ET Tube
- CV: TEE, angioplasty
Infective Endocarditis

Major Criteria:
- Two (+) Blood Cultures with typical agent
- TEE Demonstrates Endocarditis
- New Murmur

Minor Criteria:
- Fever >38C
- Vascular phenomena
- Immunologic phenomena
- (+) Blood Culture

Definitive Dx: 2 major OR 5 minor OR 1 major + 3 minor
Possible Dx: 1 major + 1 minor OR 3 minor

Complications: depend on organism, valve and time to diagnosis
- Staph aureus: more valve damage, more abscesses and emboli
- Aortic valve: embolization to brain/myocardium embolization to spleen/kidneys
- Tricuspid: septic pulmonary emboli
- Damage leads to rapid deterioration with CHF
**Infective Endocarditis**

**Prognosis:** Medical treatment is usually effective

Valve replacement surgery indicated if:
- Valve regurgitation with CHF
- Infections not responding to ABX
- Recurrent infection with same agent
- Fungal endocarditis
- +/- continued embolization

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**A patient with a diagnosis of infective endocarditis tells you that they have a history of Janeway lesions. What will most likely be found on physical exam?**

1. Exudative retinal lesions
2. Painful lesion on finger
3. Painless lesion on sole
4. Subungual splinter hemorrhages
Myocarditis
Myocardial Inflammation (Focal or Diffuse)
- Sudden onset Heart Failure
- Sx: SOB/ pleuritic chest pain
- PE: Edema/ S3 gallop
- EKG: Nonspecific ST changes w/ conduction delay
- Echocardiogram > Dilated Cardiomyopathy
- Dx: Myocardial biopsy = gold standard

Myocarditis
Myocardial Inflammation (Focal or Diffuse)
1ª = viral Infection or immune response
- Coxsackie B (measles/ influenza/ varicella)
- Kawasaki’s
2ª = Bacteria/ Toxins/ Systemic illness
- Lyme/ RMS fever/ Syphilis/ Chagas
- Radiation/ doxorubicin/ cocaine
- Lupus

Pericarditis
Localized inflammation of anterior, lateral and inferior walls may cause ST elevations in multiple areas
Acute Pericarditis

Common: Viral/often post URI
- Coxsackie B (echo/influenza/varicella)
- Men under 50 most common (post URI)
- Pleuritic/positional chest pain
- Pericardial friction rub
- Treatment: ASA/indomethacin/NSAID
- <5% develop tamponade

Acute Pericarditis

Pericardial Inflammation
- Uremia/Renal Failure → need dialysis
- Post cardiac surgery
- 2-5 days Post MI/Dressler’s Syndrome
  - CP relieved by leaning forward

Often with associated Myocarditis
- Lyme/Lupus/radiation/cancer/RA/drugs
- Hypothyroid (myxedema)

Disease: Pericarditis

[Heart waveform image]

http://www.thelhospitalist.org/details/article/2785031/How_is_Acute_Pericarditis_Diagnosed_and_Treated.html
Pericardial Effusion

Accumulation of fluid in pericardium
- Rapid accumulation = tamponade
- Fluid 2° to inflammatory process = painful
- Slow accumulation (neoplasia/uremia) = no pain
- Large effusion >1000cc w/ neoplasia

Pericardial Effusion

Diagnostic Studies
- PE: JVD/ muffled sounds/ paradoxical pulse
- CXR: enlarged/ globular cardiac silhouette
- EKG: low voltage/ T-wave Δ's,
  - maybe electrical alternans
- Echo: Primary method for detecting effusion

Pericardial Effusion

Paradoxical pulse - exaggeration of the normal variation in the pulse during respiration, in which the pulse becomes weaker as one inhales and stronger as one exhales; it is characteristic of constrictive pericarditis or pericardial effusion.

Electrical alternans - describes alternate-beat variation in the direction, amplitude, and duration of any component of the ECG waveform
**Cardiac Tamponade**

Intrapericardial pressure
- venous return and ventricle filling

- Stroke volume falls > BP falls > Shock > Death
- Dyspnea and cough
- Tachycardia
- Narrow pulse pressure
- Paradoxical pulse / Electrical Alternans

**Pericardial Effusion**

**Treatment**

**Tamponade:**
- Urgent pericardiocentesis
- Sub-xiphoid with echocardiography

**Small effusions:**
- Follow clinically (JVD) and serial echo

**Large effusions:**
- Pericardial window

Thank you!! Enjoy the rest of the review and good luck on your boards!