Cardiovascular

Acute Coronary Syndromes

- Concept: atherosclerotic plaque ruptures → exposes endothelium → platelets and fibrin attach → reduced blood flow
- If this results in cell death = MI → (+) troponin
- If no cell death = Angina/Unstable Angina → (-) troponin
- Both may have EKG changes, concerning history, etc.

Myocardial Infarction

Transmural infarction

- Tends to cause ST elevation → STEMI
- Large vessel affected
- May benefit from thrombolysis and/or PCI

Non-transmural infarction

- No ST segment elevation → NSTEMI
- Usually smaller subendocardial artery
- No benefit from thrombolysis
- +/- benefit from PCI

Posterior MI

- ST depressions in V1-V3 = ST elevation in posterior leads
- Still a STEMI, if story concerning → thrombolysis and/or PCI

**No ST elevation = No thrombolysis … EXCEPT in posterior MI

ACS – Causes

- Plaque rupture - majority of ACS
- Inflammation (ie connective tissue disease, lupus)
- Trauma and vessel damage
- Clotting disorders
- Dissection - small vessel or BIG vessel (aortic)
- Drugs (ie cocaine -> spasm and clotting)

Unstable Angina

Stable angina PLUS

- Pain at rest
- New pain starting <2 weeks prior
- Increasing severity of pain
- Hemodynamic changes associated with the pain

Prinzmetal's Angina

- Coronary spasm often seen in women
- Often nocturnal or at rest
- Can see ST segment elevation
- 1/3 have no CAD

EKGs in ACS

- Predicts risk of complications and vessel involved
- Determines thrombolysis eligibility
- Needs to be done serially
- Classic changes

ST Elevation – early (minutes to hours)

- Measured as the elevation of the J point over the T-P baseline
- Many causes, including AMI, pericarditis, BER, etc

ST Depression – could be ischemia or reciprocal change to STEMI or posterior MI

- Can be downsloping, upsloping, or horizontal
- **See Tabas review for more detail

Hyperacute T's – early (minutes)

- Tall, peaked

Q waves – usually late but can be early

- Significant Q = 1 square wide, 1/3 height QRS

**Normal or non-specific EKG seen in 5% of AMI

EKG Territories

LAD

- Anterior wall = V1-V4
- Bad -> CHF, valve ruptures, etc

Circumflex

- Lateral wall = I, aVL, V5, V6

RCA: inferior and posterior walls, R ventricle

- Inferior (PDA) = II, III, aVF
- Proximal RCA (R ventricle infarct) = II, III, aVF + V4 on right-sided EKG
- Posterior wall = V1-V3 (depression)

Pearls

- Branch point for AMI vs angina/UA is positive troponins (cell death)
- ST segment elevation = benefit from thrombolysis/PCI
- There are many causes of ST segment elevation aside from AMI
- ST depression in V1-V3 with the right history may be posterior STEMI
- Do a right-sided EKG in inferior MI to evaluate for RV infarct
- ST elevation is measured as the J point elevation over the T-P segment

Types of MI Complications

**Myocardial Infarction (MI) + new murmur + congestive heart failure (CHF) = blown valve = Surgery!
**MI location and artery can predict complications and prognosis

Early vs Late

- Early: **Arrhythmias** (fast/slow; most common), Shock (due to muscle loss), Shock (due to valve dysfunction)
- Late: Thromboembolism, myocardial rupture/valve rupture (24-48 hours post MI), CHF, **Dressler's syndrome** (autoimmune pericarditis; 2-6 weeks post MI)

Good vs Bad Prognosis

- **"Good"**
    - Wenckebach (Mobitz type 1 atrioventricular (AV) block)
    - Narrow complex bradycardias (AV node not too "sick")
    - Premature ventricular contractions (PVC)
    - Non-sustained ventricular tachycardia
- **"Bad"**
    - Mobitz Type II AV block (often wide complex)

- CHF
- Persistent tachycardia: suggests loss of large amount of muscle
- New left bundle branch block: possible indication for thrombolytics
- Large infarction area
- Failure of reperfusion: clinically patient continues to have chest pain and EKG changes after intervention

MI Complications by Location

Inferior MI: (II, III, aVF)

Keys: blocks “Good prognosis”
Bradycardias: usually **transient** and low grade

- Classically **Mobitz type 1 AV block** (Wenckebach)
- Sinus bradycardia from high vagal tone
- Can cause papillary muscle rupture (causing shock/CHF)
- Even 3rd degree AV blocks has a “good” prognosis
  - Generally narrow (only AV node affected)
- Tx: supportive

Nodal Blood Supply

- Sinus: right coronary artery (RCA) (60%) vs circumflex (40%)
- AV node: RCA (90%) vs circumflex (10%)
- **RCA = Inferior MI = Bradycardia**

Anterior/Septal MI (V2-V5)

Keys: CHF/Shock, ruptures, BAD bradycardias
CHF/Shock: due to large amount of myocardium involved
Bradycardia → “BAD” sign

- Bradycardia due to septum and conduction pathway involvement (extension of left ventricle MI into septum)
- Tends to be WIDE complex
- Not responsive to atropine

Structural Complications

- When: tend to occur 1-7 days post MI
- Who: large MI’s
- Myocardial rupture → initially causes tamponade
  - Tx: fluid load and surgery
- Papillary muscle rupture → present with shock and new murmur
  - Tx: reduce afterload, surgery
- Septal wall rupture → acute ventricular septal defect

    - Tx: reduce afterload, surgery

**MI, New murmur, CHF/Shock = Surgeon!

Right Ventricular MI

- Keys: hypotension, fluid load, associated with inferior MI
- "Inferior MI's evil twin:" same complications of inferior MI plus **hypotension** and much higher mortality
- When to think right ventricular MI?
    - In any **inferior MI** (get right ventricular leads!)
    - When **ST elevation in III > II**
    - **ST elevation in V1 > V2**
    - **ST depression in V2** and not V1
- ** **Leads III and V1 are very "right looking leads"**
- Clinical presentation: Hypotension, jugular vein distension, clear lungs and chest pain
- Tx: **volume load**, beware of nitroglycerin and vasodilators

Posterior MI

- Keys: "missed", ST depression anterior, a/w right and inferior
- Usually **"missed" MI** with ST depression anteriorly
- Associated with right and inferior MI's

Pearls

- Myocardial infarctions associated with a new murmur or congestive heart failure need emergent surgical consults.
- Inferior MI's are associated with narrow complex bradycardias that are usually transient and have a "good" prognosis.
- Bradycardias in anterior/septal MI's are "bad" and usually wide complex (implying a large amount of myocardium is affected).
- Think of right ventricular MI the following situations: Inferior MI's, ST elevation III > II, ST elevation V1> V2, ST depression V2 alone
- The appropriate treatment for right ventricular MI's is fluid, fluid, fluid; beware of nitroglycerin and vasodilators.

Treatment of AMI

General

- ****Defibrillation**: 30% mortality reduction
- ****ASA 165mg to 325mg**: 25% mortality reduction

- Nitrates: unknown mortality effects, but do reduce pain and increase blood flow
- Beta-blockers*:* not part of initial ED management anymore
    - Only give if very hypertensive or tachycardic
    - Only give orally
    - Do not give if contraindicated (CHF)
- ACE Inhibitors (ACE-I): not usually started in ED, but should be started within 24hrs

Thrombolysis

Indications for Thrombolysis

- ST Elevation (STE) (unless posterior MI: ST depression in anterior leads)
- ≥ 1mm elevation in at least 2 contiguous leads
- Chest Pain (CP) >30 min but <12 hours (some say 6hrs)
- No Percutaneous Coronary Intervention (PCI) available within 90 minutes
- No contraindications
- New LBBB is still an indication!
- LBBB with Sgarbossa criteria

Sgarbossa Criteria

- STE ≥ 1mm with concordant (same direction as) predominantly positive, QRS
- STE ≥ 5mm with discordant (in opposite direction from) predominantly negative QRS
- ST Depression ≥ 1mm in leads V1, V2, or V3

Absolute Contraindications to Thrombolysis

Badness in the head:

- Ischemic stroke in last 3-6 months (depends on reference source)
- Cerebral hemorrhage anytime
- Closed head injury last 3 months
- Known intracerebral tumor or AVM
- Active bleeding or bleeding disorder

Other bleeding risks:

- Major surgery last 2 months
- Systolic BP >180 or Diastolic BP >110 after treatment
- Suspected aortic dissection

Relative Contraindications (not as important for testing)

- Current anticoagulant use
- Prolonged CPR
- Known bleeding disorder
- Pregnancy
- Retinal disease esp. Diabetic
- Active Peptic Ulcer Disease (PUD)
- Severe HTN

Complications of Thrombolysis

** Intracranial bleeding (>50% mortality)
- Occurs 1/70 to 1/100
- Risk factors: old age; HTN; female; black; HTN; low BMI
  Major bleeding (5%) ex. GI bleed
  Allergy, especially to streptokinase (not as much with TPA)
  Treat with: blood, FFP, platelets, cryoprecipitate, aminocaproic acid (depending on circumstances)

Reperfusion: How do I know it worked?

- Pain reduction
- ST segment lowering (70% or more)
- Reperfusion arrhythmia
  - **Accelerated idioventricular rhythm** (looks like slow Vtach); lasts < 1min; is a perfusing rhythm; do not suppress.

PCI Indications

- If can be initiated within **90 minutes (door to balloon time)**
- STEMI within 12 hrs of onset
- STEMI ***and*** shock
- Contraindication to thrombolytics
- Failed thrombolytics

Cocaine Chest Pain

- Cocaine causes vasospasm
- Differential: **ACS**, pneumothorax and pneumomediastinum (inhaled cocaine)
  - ACS: generally low risk, if EKG neg, trop rule out → d/c home
- If ST Elevation/ACS: **Tx with benzos**, aspirin, nitrates, **calcium channel blockers**, phentolamine, thrombolysis if ST does not return to baseline after above treatments

Pearls

- Treatment for AMI include defibrillation and ASA; Thrombolytics and PCI in STEMI (includes new LBBB or LBBB with Sgarbossa criteria)
- If STEMI, or NEW LBBB or LBBB with Sgarbossa criteria, then: PCI if door to balloon time is less than 90 minutes; otherwise TPA if no contraindications.
- Most concerning thrombolytic complication is intracranial bleeding, occurs 1/70 to 1/100 patients, and carries 50% mortality.
- Reperfusion is working if pain improves; ST elevations improve (70%); accelerated idioventricular rhythm (perfusing rhythm, lasts <1 min)

Endocarditis

Keys to know:

- Endocarditis presentations
- A LITTLE about murmurs, rheumatic heart disease
- A LOT about specific high-risk diseases: hypertrophic cardiomyopathy (HOCM), aortic stenosis (AS)

Infective Endocarditis

- Abnormal and artificial valves get infected
- Mitral valve is most commonly-infected native valve
- Tooth extractions cause bacteremia and valvular infection (often Strep viridans)
- IV drug abuse (IVDA) introduces *Staph aureus*, which goes to the tricuspid valve
- Two flavors: acute (young pt with high fever, murmur, flu-like symptoms) and chronic (older pt, much more common, fatigue and anemia, usually strep viridans)
- Overall, Staph aureus is the most common organism
- **Remember to think about the mechanism when choosing your antibiotic (IVDA vs. dental procedure vs. recent colonoscopy)**

Typical Lesions in Infective Endocarditis

- Osler nodes: tender and on tips of fingers/toes (remember "**Oh**, that hurts; fingertip looks like an "O")
- Janeway lesions: non-tender, hemorrhagic, flat; on palms and soles (remember **Jane**, who has peripheral neuropathy, falling off her bike and bloodying her palms, but not feeling it because of the neuropathy…also she has no shoes on)
- Roth spots: retinal hemorrhages with central clearing; non-specific (remember **Roth** is so **Rude** that you punch him in the eye)
- Splinter hemorrhages and petechiae are also common

Diagnosis of Infective Endocarditis

- Echo, blood cultures, erythrocyte sedimentation rate, C-reactive protein
- In general, treat with vanco for staph, penicillin for strep, and cover gram-negatives (e.g., with gentamicin)
- Pts with abnormal valves getting procedures should get **appropriate** prophylaxis
    - Dental procedure classically amoxicillin
    - GI/GU need more gram-negative coverage
- Prophylaxis for pts with mitral valve prolapse (MVP) controversial, currently not recommended.

The Endpoint of all Valvular Disease

Heart fails and dilates → valves become regurgitant → ECG shows LVH as ventricles expand/dilate → LBBB develops as heart and conduction system stretches (poor prognostic

sign)

Murmurs: MR. ASS & MS. AID

- Mitral regurgitation (MR) and aortic stenosis (AS) are **systolic** murmurs: MR. ASS
- Mitral stenosis (MS) and aortic insufficiency (AI) are **diastolic** murmurs: MS. AID

Aortic Stenosis

- Truly important
- Initially presents as SOB, progresses to CHF
- **Syncope is a very bad prognostic sign**
- Systolic murmur radiating to neck, slow carotid upstroke
- LVH and LBBB as above
- A cause of exercise-induced syncope, very occasionally exercise-induced V-tach
- Treatment is surgical, either balloon or replacement

Aortic Regurgitation

- **Always think of aortic dissection** as a cause
- Diastolic murmur at left lower sternal border
- Severe, chronic AR can cause weird findings: water-hammer pulse, Austin Flint murmur, Duroziez's murmur, Quincke's pulse, de Musset's sign, many more

Mitral Stenosis

- **Classic presentation**: pregnant woman with cardiovascular collapse in delivery: undiagnosed disease, high-output state, high resistance in the valve causing ventricular dilation leading to A-fib, loss of atrial kick, and sudden cardiac death
- A-fib is common, and can cause decompensated CHF with hemoptysis
- Crashing pt with diastolic murmur and opening snap: **cardiovert** (suspect A-fib)

Mitral Regurgitation

- **Ischemia + shock + new murmur =** ruptured chordae tendineae/papillary muscle
- Decrease afterload with tons of nitrates, call a surgeon
- Murmur radiates widely, especially to axilla
- Atrium stretches over time, causing A-fib

Mitral Valve Prolapse

- Classically anxious young woman with atypical chest pain, SOB, palpitations. **This is wrong.** Often in men & older pts, asymptomatic
- Can worsen to cause mitral regurgitation

Pearls

- Tailor your choice of antibiotics in treatment and prophylaxis of infective endocarditis to the suspected source.
- All valvular disease eventually leads to heart failure, dilation, and regurgitation.
- Syncope in patients with aortic stenosis is a very bad sign; take it seriously as these patients may not live long.

Heart Failure

- Definition: inability of heat to maintain sufficient perfusion for normal organ function
- **Systolic**: failure of forward flow **(poor contraction/pumping)**
- **Diastolic**: failure of heart filling **(stiff ventricles)**
- Left sided: (more common) failure of left ventricle fatigue, renal dysfunction, BP, dyspnea from pulmonary congestion
- Right sided: peripheral edema, JVD
- **Most common cause of right sided failure is left sided failure**

Frank-Starling Curve

- LVEDV = Left Ventricular End Diastolic Volume SV = Stroke Volume
- In a normal heart: LVEDV (filling) causes SV (eventually a plateau is reached)
- In a failing heart: same LVEDV results in SV compared to a normal heart
    - **Increasing LVEDV only serves to worsen SV (no plateau)**
    - Nitro and Bipap are treatments that LVEDV, place back on curve

Systolic Heart Failure Etiology

- **Hypertension and ischemia** (most common in western world)
    - **Over 65 ischemia is most common**
    - Coronary artery disease (MI) “ischemic cardiomyopathy”
- Valve disease
- Non-ischemic cardiomyopathies (myocarditis)
- Many others
- “Classic” systolic HF is LOW output (i.e. you cannot pump)

High Output Failure

- High cardiac output **over time** can cause failure
    - “classic” systolic HF is LOW output
- Etiologies**: thyrotoxicosis**, chronic anemia, arteriovenous malformations (big ones), pagets disease

Diastolic Heart Failure

- Failure to fill
- Causes: **hypertension** (thick ventricle), aortic stenosis (thick ventricle), scarring post MI, diabetes (glycosylation of heart), ischemia (acute HF, can reverse once ischemia is gone)

- **End stage of heart failure = big floppy heart = systolic failure**

Chest X-ray in Heart Failure

- Upper lobe diversion: akin to jugular venous distension, fluffy edema, Kerley B lines (short fat horizontal), cardiomegaly, loss of costophrenic angle (pleural effusion)

Treatment of CHF

- IV, O2, Monitor
- Decrease preload: (decrease LVEDV to allow improved contraction) **nitrates**, +/- morphine, +/- diuretics
    - Use nitrates with caution as can make renal failure worse
- **BiPAP**/CPAP: decreases venous return (intrathoracic pressure), work of breathing
- Afterload reduction: nitroprusside, ACE, high dose nitrates
- Inotropes if shock is present
- **Chronic CHF has a VERY high mortality (60-70% 5 Year mortality)**

CHF Review

General

- Cardiac failure: inability of heat to pump enough blood to meet body's needs
- High output (i.e. Thyrotoxicosis) Vs Low output (i.e. systolic failure)
- Systolic (poor contraction, ischemic cardiomyopathy) vs. diastolic (can't fill, HTN)
- Right sided (peripheral edema) vs. left sided (fatigue, dyspnea)
- Acute (i.e. MI) Vs. Chronic (HTN)
- Systolic failure: failure of forward flow (poor contraction/pumping), diastolic failure: failure of heart filling (stiff ventricles)

Congestive Heart Failure Signs and Symptoms

- Left sided: weakness, fatigue, SOB, **S3**, PMI displaced
- Right Sided: **peripheral edema, hepatic congestion**, **JVD** (shortness of breath, cough)

Treatment

- IV, O2, monitor, nitrates, IV ACE, diuretics, **no nesiritide**, pressors (for sick only)

Work Up of Heart Failure

- ECG: non specific changes, left ventricle hypertrophy
- Arrhythmias: atrial fibrillation, ventricular tachycardia
- **Echocardiography: key investigation**
    - Ejection fraction, wall motion, clot present, valves
    - **Transesophageal has higher quality pictures, but transthoracic adequate in most cases**

Cor Pulmonale

- Definition: right sided heart failure from high pulmonary pressures
- **Left sided heart failure is most common cause**
    - Others: COPD, pulmonary fibrosis, interstitial lung disease, pulmonary HTN, vasculitis
- Diagnosis
    - Chronic right ventricular strain on ECG: P Pulmonale (peaked p waves), right ventricle hypertrophy, some acute changes (see below)
    - CXR: left heart failure
    - Echocardiography, cardiac catheterization
- **Pulmonary embolism is the most common ACUTE cause in the ED**
    - ECG changes**: T inversion V1-V3**, Right bundle branch block, **S1Q3T3**, right axis deviation

Pearls

- Cardiac failure: inability of heat to pump enough blood to meet body's needs
- High output (i.e. Thyrotoxicosis) Vs Low output (i.e.. systolic failure)
- Systolic (poor contraction, ischemic cardiomyopathy) vs. diastolic (can't fill)
- Right sided (peripheral edema) vs. left sided (fatigue, dyspnea)
- Acute (i.e. MI) Vs. Chronic
- Systolic failure: failure of forward flow (poor contraction/pumping), diastolic failure: failure of heart filling (stiff ventricles)
- Most common cause of right sided failure is left sided failure
- In heart failure, increasing LVEDV only serves to worsen SV (no plateau)
- hypertension and ischemia are the most common causes of systolic heart failure
- Treatment of heart failure: IV, O2, monitor, nitrates (decrease LVEDV), BiPap, lasix, inotropes if shock is present
- Pulmonary embolism is the most common ACUTE cause of Cor Pulmonale in the ED, look for acute right ventricle strain on ECG.

Cardiomyopathy

General

- Endpoint of cardiomyopathy is heart failure, arrhythmia, death
- Most common is dilated cardiomyopathy
- Classic textbook classification is dilated vs. restrictive vs. hypertrophic
- New classification is intrinsic (no identifiable external cause) vs. extrinsic (primary pathology outside heart)

Dilated Cardiomyopathy

- Poor contraction, low ejection fraction, heart dilates to compensate
- Most common cardiomyopathy
- Etiologies
    - Most common causes are hypertension and ischemia (i.e. CHF, not technically considered a cardiomyopathy)
    - Other etiologies: genetic, viral, alcohol abuse, postpartum
    - Sometimes reversible, usually not
- Treatment: remove cause (e.g., alcohol); treat CHF/dysrhythmias; anticoagulate; transplant if severe and non-reversible; meds for different subgroups

Restrictive Cardiomyopathy

- Fibrosis or infiltration of heart muscle causes stiffness
- Can also be caused by stiff pericardium, e.g., due to TB
- Causes diastolic dysfunction (difficulty filling)
- Heart failure with normal-sized heart

Hypertrophic Cardiomyopathy

- Heart muscle is too big or asymmetric; often no obvious cause
- Usually left ventricle (LV)
    - Most classically: septum and inferior to aorta are hypertrophied
- Severe symptoms with **exercise**
- Besides exercise-induced syncope, symptoms can include shortness of breath, chest pain, fatigue
- **Classic** is **hypertrophic obstructive cardiomyopathy (HOCM)**, a.k.a. idiopathic hypertrophic subaortic stenosis (IHSS), a.k.a. asymmetric septal hypertrophy (ASH)
- Work up
    - Echo
    - CXR usually normal
    - EKG: LV hypertrophy (tall QRS), non-specific T-wave changes, needlelike Q waves
- Treatment: avoid exertion; avoid positive inotropes; beta blockers (slow the rate and speed of contraction); AICD +/- pacemaker for ventricular arrhythmias; surgical/non-surgical ablation of septal hypertrophy

HOCM

- Caused by septal hypertrophy → turbulent flow (worse in systole)
- At worst → complete obstruction of outflow causing syncope or even sudden death
- Classic murmur at left lower sternal border

    - Murmur enhances with decreased LV volume, e.g. with Valsalva (increases intrathoracic pressure). Think of half-filledl milk jug making more noise when shaken than full jug
    - Murmur decreases with maneuvers that increase peripheral vascular resistance, dilating aorta (e.g., squatting, clenching fists)
- Treatment: beta blocker to increase filling time

Pearls

- Dilated cardiomyopathy results from the heart attempting to compensate for poor contraction and low ejection fraction; it is more common than restrictive or hypertrophic.
- Hypertrophic obstructive cardiomyopathy is can present with shortness of breath, chest pain, syncope with strenuous exercise, or sudden cardiac arrest during exercise.
- Beta-blockers are the mainstay of HOCM treatment.

Pericarditis

- Definition: inflammation of the outer covering the heart- pericardium.
- Symptoms
    - Sharp chest pain (radiates to neck, better on sitting up), dysphagia, low-grade temp, diffuse ST elevation
    - Classic to have friction rub: comes and goes, best heard sitting up and leaning forward
- **IMPORTANT: NO trop leak → if trop + or persistent tachy think myocarditis**
- EKG Stages
    - PR depression, diffuse ST elevations
    - Return to Baseline
    - T wave inversion
    - Normalization
- Causes: Viral most common; post MI (Dressler Syndrome: usually weeks post MI), radiation, uremia, connective tissue and many others
- Rx: NSAID, tx underlying cause; Echo to rule out pericardial effusion

Myocarditis

- Definition: inflammation of the myocardium **(Pericarditis' evil brother)**
- Clinical
    - Unresolving tachycardia common
    - Presents as viral syndrome, CHF, arrhythmias/atrial, sinus tach
    - **Positive troponin**
- Workup
    - Echo: normal or decreased EF
    - EKG is nonspecific/pericarditis changes
    - CXR: normal or large
    - Positive troponin

- Tx: some reversible but some not; supportive care

Hypertensive Emergencies

- Classic Associations: Aortic Dissection, Encephalopathy, Malignant HTN, ACS, Pulmonary Edema, Stroke, Renal Failure, Preeclampsia
    - We TREAT them DIFFERENTLY!
- HTN + Neuro badness: get BP down by about 30%
    - Nitroprusside (old school): arterial dilator; decreases afterload; short onset and short half-life (1-2 mins); produces cyanide metabolites; photosensitive; can produce tachycardia
    - Nicardipine (new school; like diltiazem for the brain): affects the coronary and cerebral circulation
- HTN + Dissection
    - Beta blocker **(reduce rate first!)** + Nitroprusside
    - Another option is **Labetalol:** drops BP and beta blocks at the same time
        - Alpha + beta blocker but 8x **more beta than alpha**; onset 5-10 mins; half life <6 hours; no tachycardia; **contraindicated in CHF, asthma, cocaine and MAOI**
- HTN + Cocaine badness
    - Benzo and Phentolamine (pure alpha agonist = vasodilate)
    - NO labetalol
- HTN + Pregnancy: Magnesium, delivery, hydralazine, labetalol
- HTN + ACS/Pulm Edema: IV nitroglycerin
- Disease and Drug
    - Hemorrhagic stroke: Nicardipine
    - Pregnancy: Hydralazine or Labetalol
    - Cocaine OD: Benzo/Phentolamine
    - Aortic Dissection: Esmolol then Nitroprusside
    - Pulmonary Edema: Nitroglycerin

Pearls

- Pericarditis trop negative. Myocarditis trop positive.
- Hypertensive emergencies included: Aortic Dissection, Encephalopathy, Malignant HTN, ACS, Pulmonary Edema, Stroke, Renal Failure, Preeclampsia.
- Aortic Dissection with HTN: reduce rate first with esmolol then reduce BP with nitroprusside or use LABETALOL, which does both!
- Tx of Cocaine and HTN is Benzo first then phentolamine (pure alpha agonist), NO Labetalol.

Syncope

Definition: Temporary loss of consciousness due to reduction of blood flow to the brain

Near syncope is the same as syncope for causes and work-up

Causes of Syncope

- **Cardiac:** most important cause of syncope, and **worst prognosis**
- **Non-Cardiac:** neurologic (10%), medication-induced, bleeding (hypotension), idiopathic (30%)
- **Vasovagal or "idiopathic"** is the **most common cause of syncope**

Admission Criteria for Syncope

- **San Francisco Syncope Rule:** features are remembered with the CHESS Mnemonic
    - C: CHF
    - H: hematocrit < 30
    - E: EKG (abnormal)
    - S: shortness of breath
    - S: systolic pressure < 90 mmHg
    - Admit these patients, they are at high risk for a serious outcome (death, myocardial infarction, PE, stroke, arrhythmia, etc)
- **Admission based on History**: in addition to a thorough history, **high risk features** include **exercise-induced** syncope/family history of sudden death
- **Admission based on concerning EKG findings**: ischemia, WPW, Brugada, hypertrophic cardiomyopathy, QT prolongation, (bradycardia/tachycardia)
    - **WPW**: look for the **short PR-interval**, and the **delta wave**
    - **Brugada**: look for **RSR** and **ST segment elevation in the anterior leads**
    - **QT prolongation**: QT is more than **500 msec**, or if the T wave is halfway or more than halfway between the R to R interval
    - **Hypertrophic cardiomyopathy**: **needle-like Q waves**, and **large voltages** (LVH) → get an echo
    - Other findings: right ventricular outflow tract, ventricular tachycardias, etc.

**Fun Fact: Syncope and near-syncope have the same causes and require the same work-up in the Emergency Department!*

Pearls

- Causes of syncope are classified as cardiac and non-cardiac causes; **cardiac causes** are the **most lethal** and important to identify.
- The San Francisco Syncope Rule includes **admission criteria** for patients with **CHESS** (CHF, hematocrit <30, concerning EKG findings, shortness of breath, systolic BP <90 mmHg).

- The most important EKG findings to identify include **ischemia, WPW, Brugada, HCM,** and **QT prolongation**, admit patients based on these abnormalities.
- Pre-syncope requires the same workup as syncope.

Aortic Dissection

- Medical dissection is a disease of long-term hypertension or in patients with connective tissue disorders (Marfans). Rarely: Pregnancy, coarctation, bicuspid valve.
- Classic: tearing/ripping, severe pain, pulse deficit, aortic regurg, tamponade, inferior MI (rare), CXR wide mediastinum
- ECG may be normal or MI if dissection back into coronary
- Chest pain AND something else (pulse deficit, neuro finding, leg weakness, abdominal pain) = dissection!
- Stanford Criteria
    - Type A: ascending aorta, surgical tx
    - Type B: descending aorta, medical tx
- Diagnosis
    - CT with contrast (new gold standard) vs. Angoigram
    - MRI: sensitive but takes long time
    - TEE: sensitive but user dependent and needs cardiologist
    - CXR: poor sensitivity and specificity; wide mediastinum
    - **DON'T SEND UNSTABLE PATIENTS TO CT**
- Treatment
    - **REDUCE the RATE** then lower the BP
        - Esmolol then Nitroprusside or Labetalol as a single agent is often used because it reduces rate and decreases BP
        - Rate reduction considered important to reduce shear stress on aortic wall

Abdominal Aortic Aneurysm

- Definition: pathological dilation of the aorta; if it ruptures = death
    - >3 cm is bad, >5.5cm is surgery
- Disease of arteriosclerosis therefore all the same risk factors
- Classic presentation: sudden death, syncope, abdominal pain, FLANK pain, blue toes (peripheral emboli), abdominal mass
- Rarely presents with: erode into gut (GI bleed); erode into IVC
- Diagnosis
    - Ultrasound: fast/accurate
    - CT: fast/very accurate but not for unstable patients
    - Plain Films: fast, not accurate shows Ca2+
- Rx: blood and surgery

Acute Arterial Occlusion

- Causes: emboli block the pipe → the pipe bursts then dissects and the crap in the pipe

bursts (ruptured plaque)
- Etiology
    - FAST sx onset: Embolism or Dissection (aortic or local)
    - SLOW sx onset: Thrombosis of a plaque, low cardiac output
    - **Proximal embolic source: CARDIAC** (MI, Afib, Endocarditis)
- Acute Limb Ischemia: **5 Ps - Pain, Pallor, Paresthesias, Paralyzed, Pulseless**
- Diagnosis: Angiogram gold standard; CT angiogram used all the time; US - no contrast, fast, pics not as pretty as CT
- Rx: Heparin (stop more clotting), thrombolysis, embolectomy, bypass
    - Slow ischemia is black dead feet, may not need acute surgery if not infected, may need excision

Venous Thrombosis

- General
    - **Classic triad (stasis, hypercoagulable, endothelial damage)**
    - Swelling, pain increased with palpation, erythema, fever (low grade)
    - Homan's sign of DVT: pain in the calf on dorsiflexion of foot at ankle while knee is fully extended (50% sensitive)
    - Well's criteria for DVT: if >3 points then 75% likely (1 point for each of the following: active cancer, paralysis/immobility, bedridden >3 days with sug in last 4 weeks, tender vein, unilateral calf swelling >3cm, unilateral pitting edema, entire leg swollen, collateral superficial veins)
- Diagnosis
    - Ultrasound with duplex new gold standard (old gold standard venography, which can produce a DVT)
    - CT venography emerging standard
    - D-dimer and Well's criteria to rule out low risk.
- Treatment
    - Heparin, then oral anticoagulant
    - Thrombolysis or interventional if HUGE (50% have malignancy)
        - Phlegmasia cerulean dolens: literally mean painful blue leg (HUGE DVT)
        - Phlegmasia cerulean albicans: complete arterial occlusion as well

Pearls

- There are 4 types of vascular emergencies (aortic dissection, abdominal aortic aneurysm, acute arterial occlusion and venous thromboembolism)
- First reduce the heart rate then the blood pressure in aortic dissection by using esmolol then nitroprusside.
- Size matters for AAA and >3 cm is bad, >5.5cm is surgery
- Acute limb ischemia has the 5 classic findings Pain, Pallor, Paresthesias, Paralyzed, Pulseless.