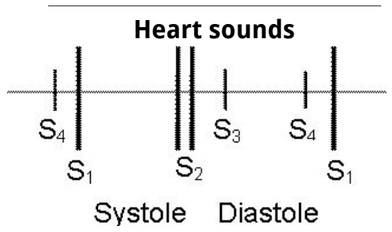
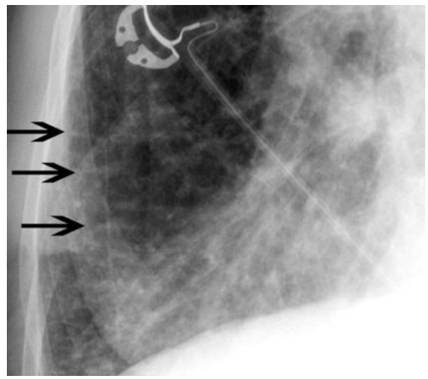
Table of Contents

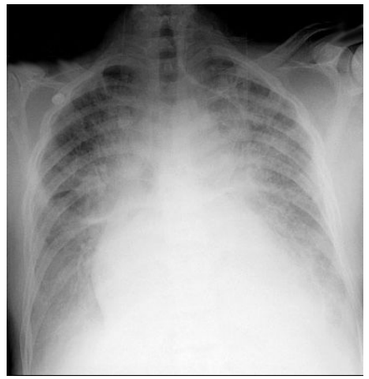
Common Heart Symptoms	Common Cardiomyopathy Signs
• Chest Pain/Discomfort: “pressure”, “tightness”, “squeezing” • Dyspnea • Palpitations, Dizziness, Syncope • Nausea	• Diaphoresis • Cyanosis • Vital Signs: normal or abnormal • Jugular Venous Distention • Rales • Precordial Pulsations • Heart Sounds/Murmurs • Edema

Congestive Heart Failure

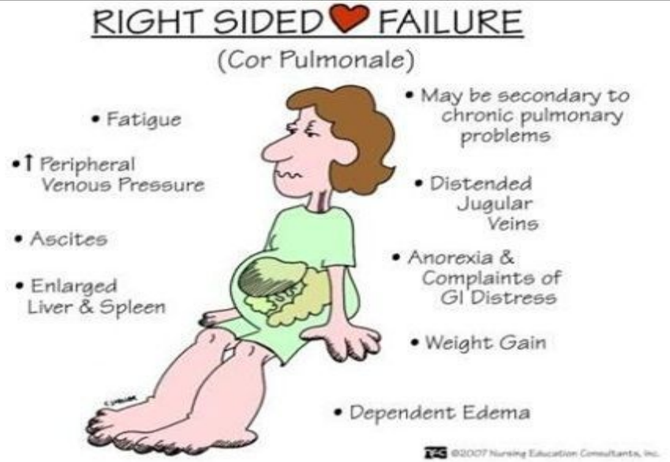
Common symptoms	Common Signs	Labs	Diagnostic Testing	Treatment	Prognosis
• Dyspnea, orthopnea, PND, rest dyspnea • Non-productive cough, nocturia • Fatigue, exercise intolerance • Nausea, decreased appetite (suspected due to hepatomegaly)	• Vital signs may be normal; tachycardia, hypo- or hypertension • Cold extremities because body moves blood to where it is needed (heart, brain, kidneys); diaphoresis; SOB leads to release adrenaline • JVD • Rales at lung bases when sitting or standing • Pleural effusion with dullness at bases • Hepatojugular reflux • Pitting edema, ascites • S4 in diastolic = a stiff wall • S3 in systolic = slush in	• Anemia, increased RDW, abnormal kidney function • Serum BNP sensitive in heart failure ○ less so in older patients, women, & COPD: If their BNP <100 pg/ml with normal ECG then it is unlikely to be CHF 	• **ECG**: dysrhythmias, MI, IVCD, non-specific changes (as in non-specific ST changes) • **CXR**: heart size (cardiomegaly), perivascular edema, **cephalization of vessels** to apex of lungs to compensate, **Kerley B lines** (horizontal white lines due to fluid around vessels), interstitial fluid, alveolar fluid, fluffy bilateral opacities	• Correct reversible causes • Diuretics, ACEI, ARB, spironolactone, beta blockers, digitalis, nitrates & hydralazine, anticoagulation, antiarrhythmic therapy, statins, • Pacemaker/defibrillator, diet, exercise training, coronary revascularization (CABG), cardiac transplantation, ventricular assist devices • Palliative care	• Heart failure with decreased EF has poor prognosis • With treatment, the 5 year mortality rate is approx. 50% • Prognosis has improved because of new treatments (ACE inhibitors & beta blockers)



Kerley B Lines



Cardiomegaly



Left Sided Heart Failure	Right Sided Heart Failure
• Usually systolic: due to ischemic heart disease,mitral/aortic regurgitation, ventricular septal defects, chronic hypertension, or dilated cardiomyopathy ○ **Systolic failure** means that the heart is not able to pump blood hard enough which leads to increased diastolic filling pressure and eccentric hypertrophy (since cardiac myocytes cannot proliferate they get bigger, in eccentric sarcomeres are attached in series to one another) ■ measured by ejection fraction; lowered in HF; @ <40% in HF; normal is 50-70% ■ Use echo to measure the size of heart and measure ejection fraction • Can be diastolic: due to aortic stenosis, hypertrophic cardiomyopathy, or restrictive cardiomyopathies such as pericarditis (wall thickness and chamber size normal) ○ **Diastolic failure** is when you have filling problems; ventricles are too stiff from concentric hypertrophy (sarcomeres added in parallel), which leads to reduced ventricular compliance since it can't distend' also HOCM can cause septal hypertrophy leading to diastolic HF ■ Heart is squeezing but is not filling enough due to a reduced preload ● ***frank-starling mechanism*** = loading the blood with more blood leads to a more forceful contraction and a larger stroke volume ■ Leads to increased diastolic pressure ■ Has normal EF; but low volume capacity	• Often caused by left sided heart failure, but can be isolated due to atrial/ventricular defects or chronic lung disease • If caused by left sided HF, then the increased pulmonary pressure makes it harder for the right ventricle to pump leading to biventricular failure • Isolated causes ○ Septal Defects ■ Increased in fluid volume leads to concentric hypertrophy (frank-starling), increased thickening will lead to ischemia (systolic) or smaller preload (diastolic) ○ Chronic lung disease ■ Hypoxia leads to arteriole constriction (diverting blood away from poorly ventilated areas) which leads to increased pulmonary pressure which leads to right sided hypertrophy and HF = COR PULMONALE ○ Leads to back up of blood into the body's veins, organs, peritoneal space, legs (or sacrum when lying down) ■ Leading to JVD (kussmaul sign), hepatosplenomegaly (tenderness upon palpation due to course III cirrhosis), ascites (after increased portal tension), pitting edema

With heart failure the muscle wall can stretch and thin out or it can thicken and become ischemic (leading to mixed systolic/diastolic failure). In either case, those heart cells get irritated leading to heart arrhythmias. With an arrhythmia, the ventricles don't contract in sync anymore decreasing their capability to pump out blood efficiently exacerbating the patient's condition. Osmosis CHF

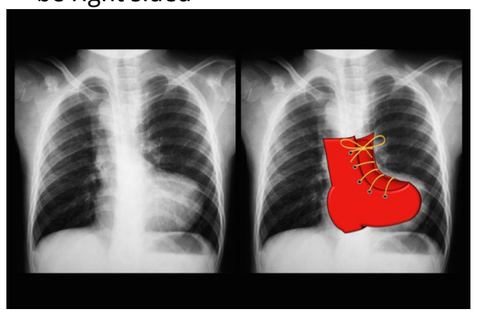
Compensation includes release of ANP (due to atrial stretching) and BNP (due to ventricular stretching) which leads to vasodilation, increased GFR, then natriuresis/diuresis, and also the inhibition of aldosterone. Eventually this leads to increased sympathetic activation which causes increased heart rate, cardiac contraction, as well as vasoconstriction. The RAA system will activate. Both RAAS and sympathetic activation causes increased afterload (vasoconstriction). ADH is released from the anterior pituitary to further increase water retention. Finally the heart can undergo cardiac remodeling with eccentric and concentric hypertrophy. The lungs will exhibit cephalization of vasculature, this is visible in CXR although visualization of cephalization is subjective and cannot be relied on solely for diagnosis.

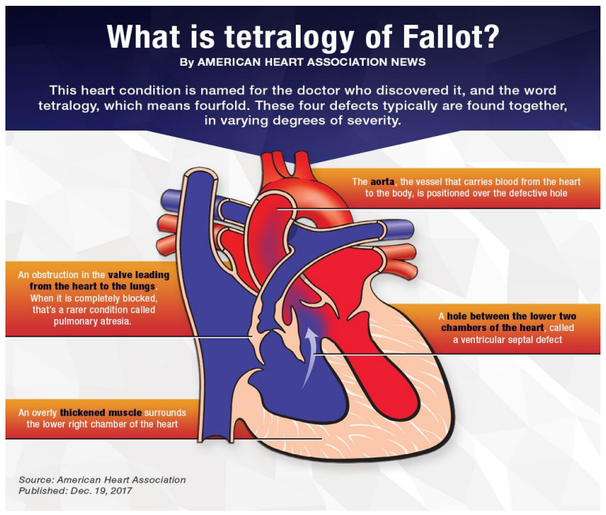
Atrial Septal Defect				
Common symptoms	**Common Signs**	**Diagnostic Testing**	**Treatment**	**Prognosis**
• Asymptomatic unless complications arise	• RV lift; S2 widely split and fixed • Systolic ejection murmur parasternally • Neonates with ***TAPVR***	• echocardiogram: right atrial & right ventricular overload; transesophageal echo (TEE) useful for small ASD • EKG = RAD or RVH; RBB • CXR: large pulmonary arteries; enlarged RA & RV	• correction of the defect percutaneously or by surgery	• large shunts cause symptoms by age 40; if untreated, dysrhythmias (a-fib) & heart failure occur

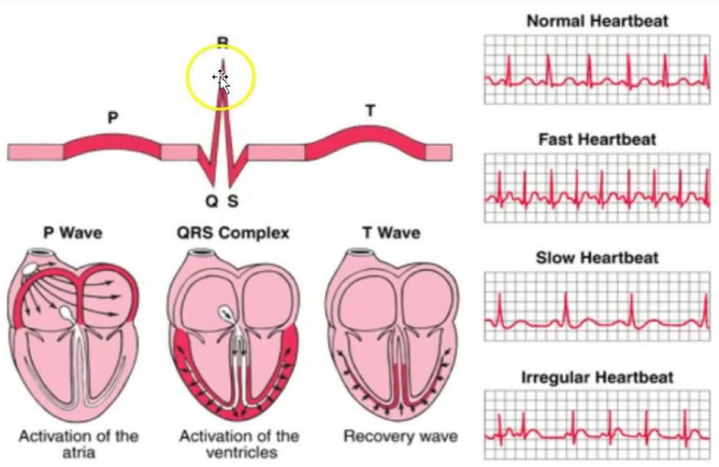
Ventricular Septal Defect				
Common symptoms	**Common Signs**	**Diagnostic Testing**	**Treatment**	**Prognosis**
• depends on the size of defect • Pulmonary hypertension	• Loud, harsh holosystolic murmur in left 3rd, 4th interspace • Systolic Thrill	• ECG normal or may show RVH, LVH or biventricular hypertrophy • echocardiogram/Doppler show size of overloaded chambers & flow across the defect	• medical management of pulmonary hypertension or surgery to correct the defect	• small VSD – normal lifespan (small risk of endocarditis)

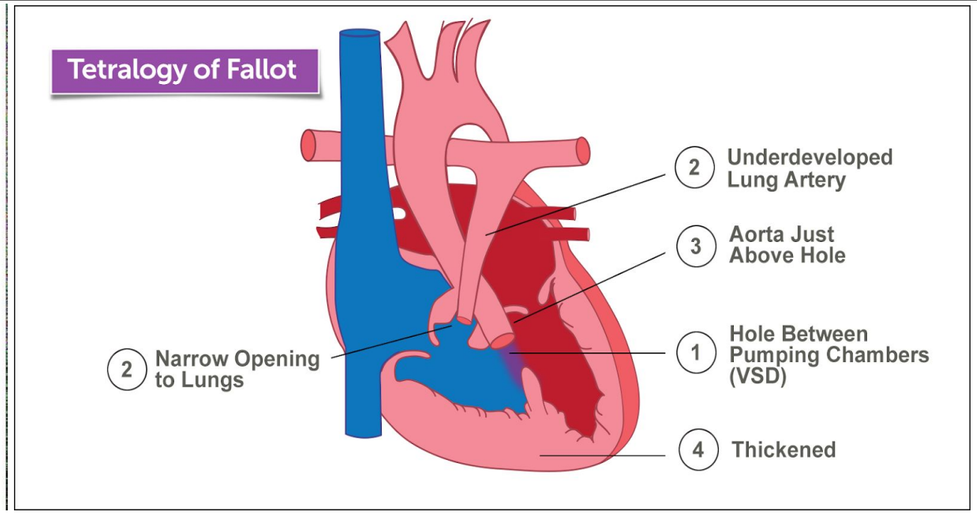
Tetralogy of Fallot

Most common of right to left shunts. It is congenital; caused by failure of neural crest cell migration. Associated with 22q11 deletion syndromes (e.g. DiGeorge)

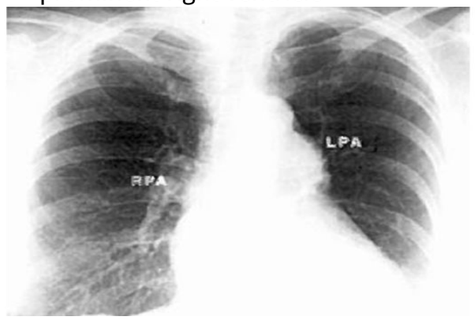
Common symptoms	Common Signs	Diagnostic Testing	Treatment	Prognosis
• Depends on the size of defect and stenosis of pulmonary valve • Pulmonary hypertension • If severe children will be cyanotic • *Some children might squat to dodge "tet spell"* ○ *squatting increases systemic vascular resistance (SVR) forcing more blood upward into the pulmonary circulation*	• **VSD, RVH** • **RV outflow obstruction** due to infundibular (opening of the pulmonary artery) ○ Severe pulmonary valve regurgitation if repaired with transannular patch • **Overriding of aorta** (pushed to the right; over the atrium) • Right sided aortic arch • **QRS width is greater than 180 milliseconds** ○ Picture of typical QRS below • Arrhythmias • **Heart sounds:** ○ pulmonary outflow murmur- Harsh systolic **crescendo-decrescendo murmur** ○ right sided gallop ○ aortic regurgitation murmur might be heard	• EKG=RVH, RAD, RBBB • CXR shows boot shaped heart & prominent right ventricle; aorta may be right sided  • Echo/Doppler makes the diagnosis; also useful in post-op tetralogy repair patients; cardiac CT and MRI may also be useful; cardiac catheterization may be necessary	• Operation with a RV outflow patch and VSD closure • a-fib and ventricular ectopy are common; LV dysfunction may occur; aorta may enlarge & may require surgery; AICD are often utilized to look for rhythmic abnormalities.	• small VSD – normal lifespan (small risk of endocarditis) • Death if QRS greater than 180 msec or if RV becomes too enlarged ○ Holter monitoring periodically to track progression of disease.







Pulmonary Valve Stenosis

Common symptoms	Common Signs	Diagnostic Testing	Treatment	Prognosis
• Asymptomatic unless lesion • Dyspnea on exertion (DOE), syncope, chest pain, RV failure	• Right sided heart failure • High pitched systolic crescendo-decrescendo murmur in the *second left interspace* with radiation to the left shoulder • Delayed or soft/absent P2 • Pulmonary ejection click,	**CXR:** • Right axis deviation • Heart size may be normal on xray or prominent right ventricle  • Prominent RV, sometimes a dilated pulmonary artery	• Echo/Doppler is tool of choice • Balloon valvuloplasty treatment of choice • Surgical commissurotomy or valve replacement may be indicated • Endocarditis prophylaxis unnecessary with native valve	• Mild disease: normal lifespan without treatment • Severe disease: rarely fatal; R heart failure

Pulmonary Valve Regurgitation

Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment
• mostly asymptomatic	• A loud diastolic (***graham-steell***) murmur in high pressure pulmonary valve regurgitation • Soft or no murmur in low pressure pulmonary valve regurgitation (this is well tolerated) • widely split 2nd heart sound	• Pulmonary hypertension	• ECG: little value, but RBBB common • CXR: enlarged RV and PA • Echo/Doppler often helpful • Cardiac CT/MRI may be useful	• rarely needs specific therapy; • rarely, valve replacement (**bioprosthetic valve**) needed

Mitral Regurgitation

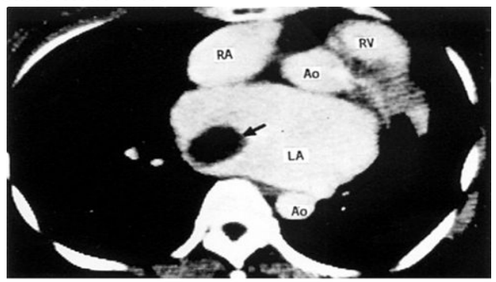
Valvular damage in acute rheumatic fever can cause mitral regurgitation → subsequent group A streptococcal infections cause repeat episodes of ARF and worsening symptoms → chronic rheumatic heart disease

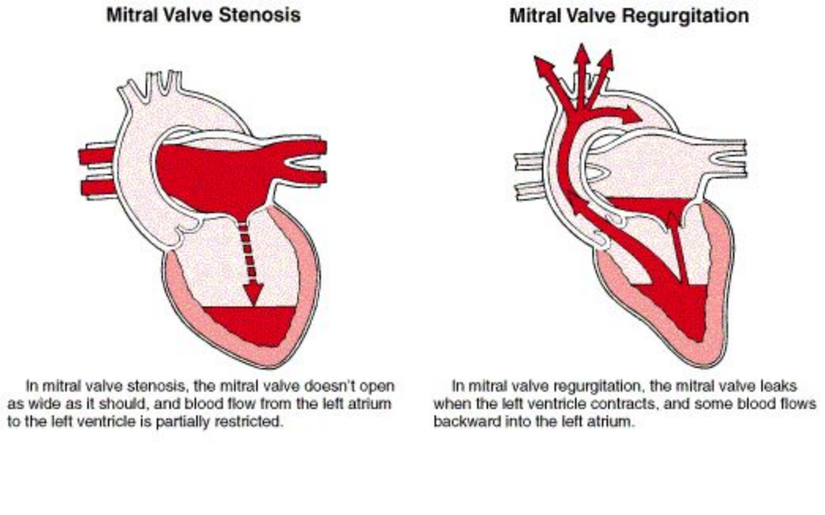
Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment
• **Asymptomatic unless severe** which can lead to LSHF • exertional dyspnea, fatigue, pulmonary edema • Diuresis - functional mitral regurgitation (due to acute left ventricular volume overload) can be corrected with diuresis (decreased murmur)	• **Pansystolic murmur**, S3; **maximally at apex radiating to left axilla** → "sloshing in" • A new holosystolic murmur present in a child with reduced CO and fever should make you suspicious of rheumatic fever • Acute pulmonary venous hypertension (disappears in chronic MR) • **Acute MR will have decreased CO with increased ejection fraction** (to LA and Aorta) ○ an increased ejection fraction maintains cardiac output in chronic compensated mitral regurgitation	• **Mitral Valve Prolapse** that has progressed. • **Myxomatous Degeneration** can cause chordae tendinae rupture and acute mitral regurgitation • papillary muscle rupture following MI • **Infective endocarditis** can damage the chordae tendinae → rupture • **Dilated cardiomyopathy** of left chambers can stretch the mitral annulus → mitral regurgitation ○ **chronic mitral regurgitation** allows for the left atrium dilation and hypertrophy→ less pressure transmitted to pulmonary circuit (no significant pulmonary edema)	• EKG=LA abnormality or A-fib with LVH (eccentric) • Echocardiogram/Doppler and/or TEE; cardiac MRI or cardiac catheterization may be indicated; • CXR shows prominent left atrium ○ chronically increased preload in chronic mitral regurgitation causes eccentric hypertrophy of the left ventricle	• if symptomatic, and if EF is less than 60%, surgery is indicated; • acute mitral regurgitation may develop after MI, endocarditis or hypertrophic cardiomyopathy: and needs emergent surgery • Placement of pacemaker to improve biventricular pacing

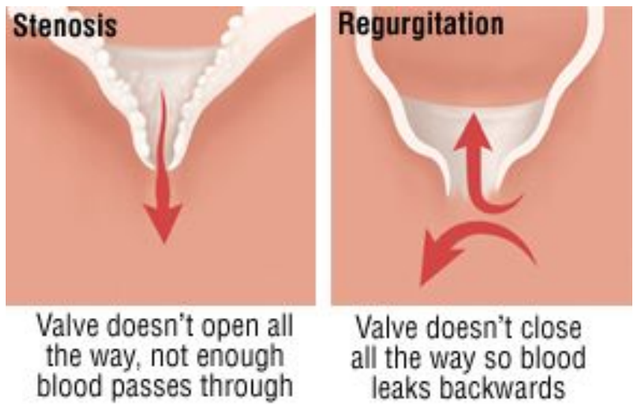
*****Dr. Beckerman: All diastolic murmurs are pathological*****

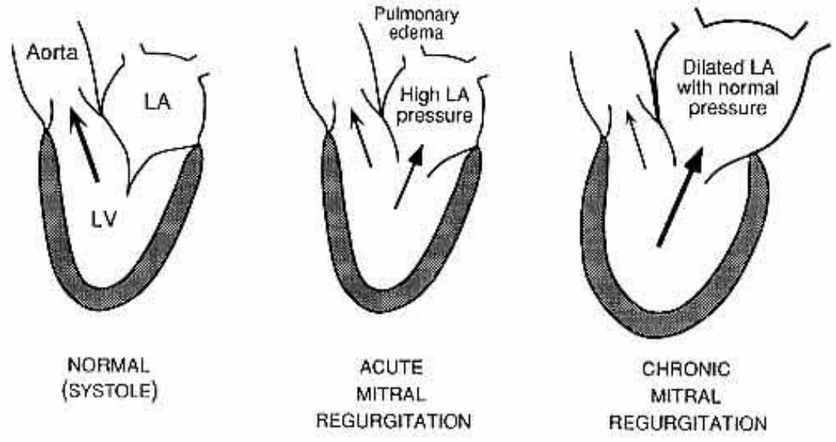
Mitral Stenosis

Regurgitation with years of inflammation and scarring of the mitral leaflets in chronic RHD can lead to mitral stenosis (and sometimes aortic stenosis)
Though uncommon mitral stenosis can also be caused by annular calcification (degenerative calcific deposits in the fibrous ring of the mitral valve in older people)

Common symptoms	Common Signs	Diagnostic Testing	Treatment
• Fatigue, exertional dyspnea, and orthopnea (if severe) • Asymptomatic unless severe • If severe leads to pulmonary hypertension	• Prominent mitral first sound, opening snap, and apical diastolic rumble ○ the **mid-diastolic murmur of mitral stenosis is preceded by and opening snap** (heard over apex of left sternal border) ○ The closer the opening snap is to the S2 heart sound, the greater the severity of mitral stenosis • Auscultatory A2 opening snap • Hypertrophy of the left atrium ○ compression of the left recurrent laryngeal nerve by a dilated LA can cause chronic cough or hoarseness; as well as dysphagia	• ECG= **LA abnormality (dilation) and atrial fibrillation** • Echo is diagnostic • Static blood sitting in RT atrium can form a clot, visible with echo  Source: Maxine A. Papadakis, Stephen J. McPhee, Michael W. Rabow: Current Medical Diagnosis & Treatment 2018 	• **Balloon valvuloplasty or open surgery** (commissurotomy or replacement); • If a-fib develops, early chemical/electrical conversion • Anticoagulation: a-fib may redevelop in 20-30% of patients ○ LA hypertrophy leads to the development of mural thrombi







Mitral Valve Prolapse

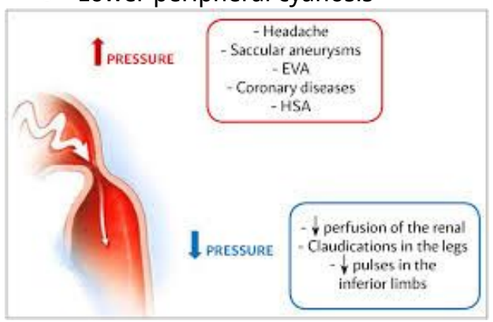
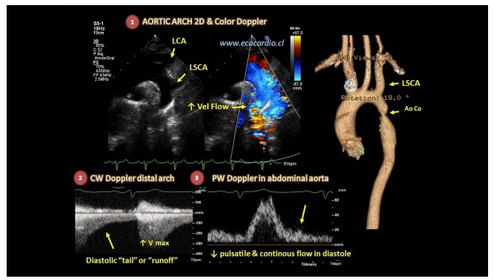
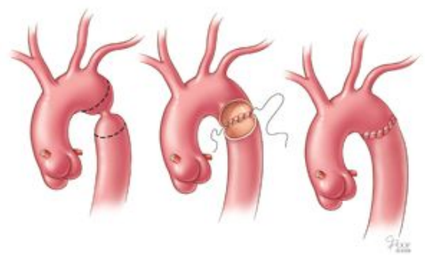
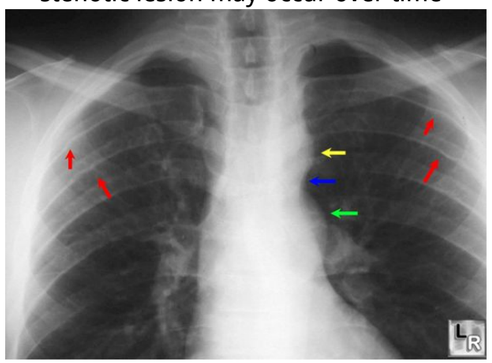
Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment
• May have hyperflexible joints • Usually asymptomatic • Chest pain and palpitations in young adults • dyspnea, fatigue	• **Single or multiple mid-systolic clicks** *caused by the chordae tendineae suddenly stopping the leaflets when they are pulled backward* • Pansystolic or late systolic murmur	• **Skeletal changes** = pectus excavatum, scoliosis, or straight back • myxomatous degeneration (pathologic deterioration of connective tissue) causes mitral valve prolapse • ***Papillary muscle rupture from ischemia***	• clinically made & confirmed by echocardiography	• ***beta blockers*** usually used for symptomatic patients; • **valve repair better than valve replacement**; • endocarditis prophylaxis no longer recommended

Murmur Changes Mitral Valve Prolapse

Mitral valve prolapse presents with a mid-systolic click (sudden tensing of the chordae as valve leaflets prolapse) between the S1 and S2 heart sounds. Maneuvers that decrease preload (e.g. Valsalva) cause the mid-systolic click in MVP to move closer S1. Tachycardia (decreases the diastolic filling time and preload) will cause the mid-systolic click in MVP to move closer to S1. Maneuvers that increase preload (e.g. straight leg raise) cause the mid-systolic click in MVP to move closer to S2. Maneuvers that increase preload (e.g. squatting) cause the mid-systolic click in MVP to move closer to S2. Maneuvers that increase afterload (e.g. sustained hand grip) cause the mid-systolic click in MVP to move closer to S2

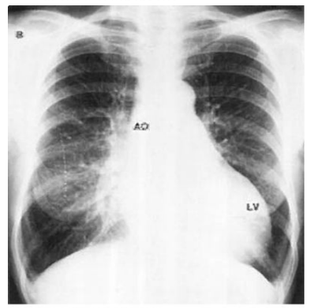
Coarctation of Aorta

Congenital defect that occurs near the insertion of the ductus arteriosus; it can occur in patients with Turner syndrome (monosomy X)

Common symptoms	Common Signs	Diagnostic Testing	Treatment	Prognosis
• No symptoms until LV failure ○ Lower peripheral cyanosis 	• **Systemic hypertension** ○ Systolic pressure higher in upper extremities than in lower extremities / diastolic will be similar • **Delayed pulse** in femoral artery compared to brachial artery • Strong **arterial pulsations** in neck/sternal notch • Systolic ejection murmurs	• Echo/Doppler usually diagnostic; CTA or MRI may demonstrate the coarctation  	• **Resection of coarctation** (mortality 1-4%); endovascular stenting (percutaneous); reoccurrence of the stenotic lesion may occur over time  • **<u>Chest Radiography</u>**: ***LVH, "3" sign, and notching of ribs on CXR*** ○ Coarctation of the aorta can cause inferior "rib notching" on CXR (collateral circulation in the intercostal arteries)	• Heart Failure; especially in neonates • severe coarctation – may die of ○ HTN ○ Aortic rupture (dissection), ○ Infective endarteritis ○ Cerebral hemorrhagic stroke (berry aneurysm)

Aortic Stenosis

Most common in 6th - 8th decades of life

Common symptoms	Common Signs	Causes	Labs	Diagnostic Testing	Treatment	Prognosis
• **angina or syncope** • Heart failure ○ **Orthopnea**	• Delayed diminished carotid pulse in severe stenosis • Soft, absent, or paradoxical split S2 in severe stenosis • ***Harsh crescendo decrescendo systolic ejection murmur at aortic area radiating to the axilla and apex, sometimes with thrill along left sternal border*** • afterload increase on LV; **LVH** (concentric) → S4; myocardial dysfunction; LV failure,	• Congenital unicuspid or bicuspid aortic valve (asymptomatic until middle/old age) • **Degeneration or calcification of the valve may cause the problem** • Risk factors include HTN, hypercholesterolemia, smoking, diabetes • Formerly caused by chronic rheumatic heart disease • Mostly caused by atherosclerotic heart disease	• **BNP** (promotes peeing) is a marker of early LV myocardial failure; high levels suggests poor prognosis	• ECG shows LVH • CXR indicates enlarged cardiac silhouette, calcified aortic valve, dilation or calcification of ascending aorta • Echo & Cardiac Cath are best studies  Aortic Stenosis: LVH, prominent ascending aorta	• valve intervention in symptomatic AS (open surgery or ***TAVR***) • medical therapy to treat CHF • balloon valvuloplasty may be useful • medical treatment does not prevent progression of disease but BP control is beneficial • With mechanical valve replacement: anticoagulation • Consider surgery for asymptomatic patients with severe aortic stenosis	• Aortic stenosis alone is a marker for future cardiovascular events and death ○ aortic stenosis causes chronically elevated pressures in the left ventricle and atrium → LA dilation and hypertrophy → atrial fibrillation ○ jet stream through a stenotic aortic valve can cause hemolytic anemia • 25% of adults >65 years of age have some thickening of their aortic valve

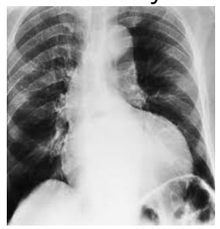
Murmur changes in Aortic Stenosis

Maneuvers that increase preload (e.g. straight leg raise or squatting) increase the murmur of aortic stenosis (due to increased stroke volume across the valve). Maneuvers that decrease preload (e.g. standing, valsalva) or increase afterload (e.g. handgrip) reduce the murmur of aortic stenosis (due to reduced stroke volume across the valve)

****Dr. Beckerman: Syphilis is a risk factor for thoracic aortic aneurysm. Me: Also aortitis in tertiary syphilis can cause aortic regurgitation (due to aortic root dilation)****

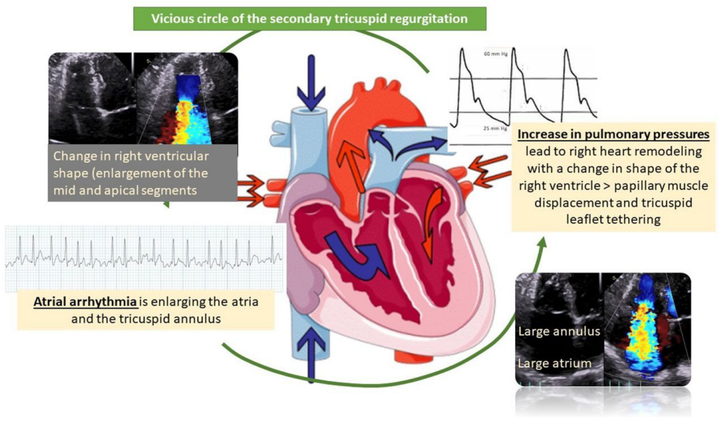
Aortic Regurgitation

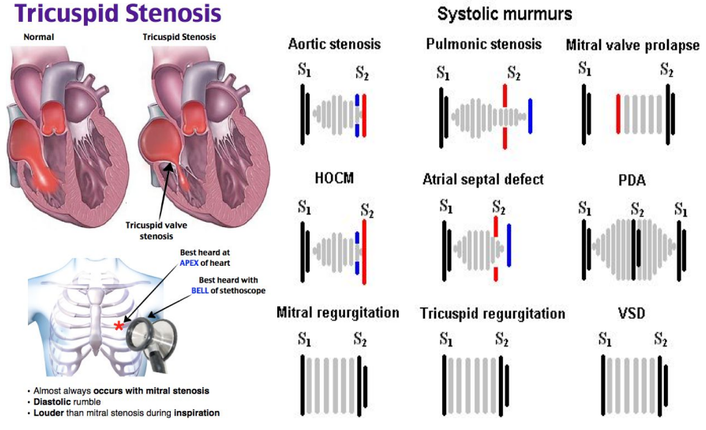
the valvulitis in acute rheumatic fever (most common cause in developing world) and dystrophic calcification can cause aortic regurgitation (most common cause in developed world) → tertiary syphilis; Marfans; and endocarditis can all cause AR

Common symptoms	Common Signs	Labs	Diagnostic Testing	Treatment
• Angina, rarely chest pain; if so usually when older, depends on rapidity with which regurgitation occurs	• Hyperactive, enlarged ventricle (eccentric hypertrophy); S3 • Soft aortic decrescendo **diastolic murmur** along left sternal border • Wide pulse pressure and high stroke volume leading to peripheral findings ○ ***Corrigan's water hammer pulse*** • **Low diastolic pressure (160/60)** ○ increased stroke volume in aortic regurgitation causes an increased SBP and decreased DBP → widened pulse pressure (e.g. 160/60) • LV failure is sudden, and late, event	• serum BNP may be a sign of impending LV failure	• ECG shows LVH; • CXR shows cardiomegaly & LV prominence • Echocardiogram is definitive study; cardiac CT or MRI may be helpful;  *LV Prominence and Cardiomegaly*	• when LV failure occurs, surgical replacement of valve indicated; • medication to reduce preload & afterload on LV may be beneficial; • replacement of valve may also require surgery on the aortic root • The issues discussed with aortic stenosis apply here also

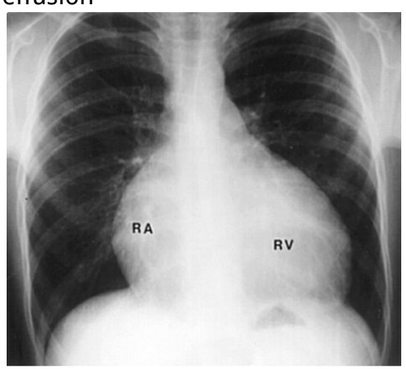
Tricuspid Stenosis

Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment	Prognosis
• hepatomegaly, • ascites, • dependent edema;	• Elevated JVP with prominent a wave • Right heart failure (both dilatation or stiffening) the causes • diastolic rumble along lower left sternal border increasing with inspiration	• **H/o rheumatic heart disease** • Carcinoid disease • Prosthetic valve degeneration	• ECG: right atrial enlargement, enlarged p waves • CXR: marked cardiomegaly with normal Pulmonary artery size • Echocardiography and/or cardiac catheterization	• reduce fluid congestion with diuretics; • valvuloplasty not very effective; cause you will reverse the stenosis and cause regurg • valve replacement is the preferred approach almost always with **bioprosthetic** valves; often done along with mitral valve replacement	• Predominantly in females





Tricuspid Regurgitation

Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment
• identical to those from RV failure of any cause • cyanosis may occur; hepatomegaly, edema, ascites, pleural effusion	• Systolic c-wave in JVP • Holosystolic murmur along left sternal border, which increases with inspiration • Carvallo's Sign: murmur gets louder during inspiration due to increased venous blood return to R side of heart. Used to distinguish between tricuspid and mitral valve regurgitation.	• Pulmonary or cardiac disease • Pacemaker lead placement	• ECG: nonspecific; A. flutter or a-fib are common • CXR: enlarged RA, sometimes pleural effusion  Source: Maxine A. Papadakis, Stephen J. McPhee, Michael W. Rabow: Current Medical Diagnosis & Treatment 2018 Copyright © McGraw-Hill Education. All rights reserved. • Echocardiogram and/or cardiac catheterization	• Oral/IV diuretics (depending on severity) • Treat heart failure • Valve replacement (**bioprosthetic valve**) – often with mitral valve replacement

Coronary Artery Disease

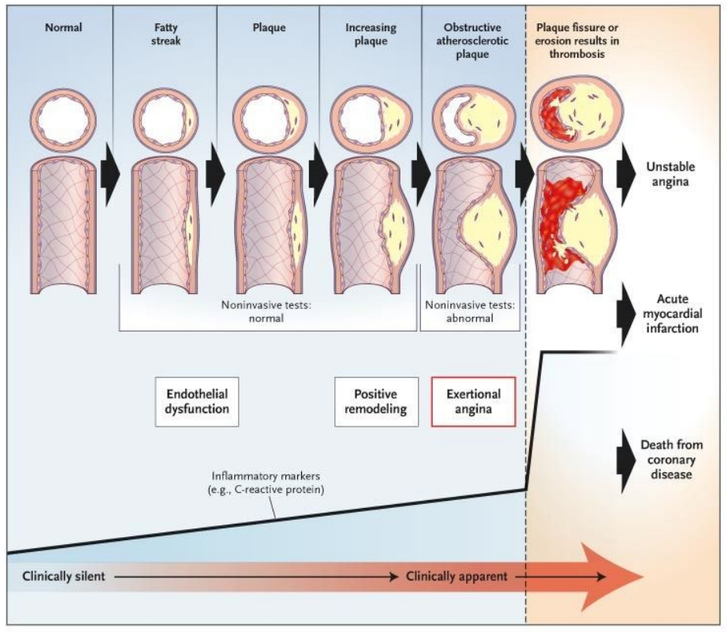
Causes	Labs	Treatment	Prognosis
• **Uncontrollable causes** ○ *Metabolic Syndrome aka syndrome X* ■ Need at least ⅗ controllable ○ Positive Family History ○ Male Sex ○ Blood Lipid Abnormalities ○ Diabetes Mellitus (if genetic) • **Controllable causes** ○ Hypertension ○ Physical Inactivity & Abdominal Obesity ○ Smoking ○ Consuming too few fruits/vegetables ○ Too much alcohol consumption	• Triglycerides >150 mg/dL • HDL Cholesterol <40 mg/dL (men), <50 mg/dL (women) • Fasting Glucose >110 mg/dL • **High sensitivity CRP** ○ acute phase protein that is produced predominantly by hepatocytes under the influence of cytokines such as interleukin (IL)-6 and tumor necrosis factor-alpha ○ If CRP is high, must address hyperlipidemia (low=monitor) • interleukin-6, CD-40 ligand, myeloperoxidase, placental growth factor	• Smoking cessation • Treating dyslipidemia • Lowering BP • Lowering LDL • Widespread use of **statins** - All patients at significant risk for vascular events should receive a statin regardless of their cholesterol levels • Antiplatelet therapy is another very effective preventive measure • vascular disease should be treated with an ACE inhibitor or ARBs	• 37% of people who have a cardiac event (angina or MI) will die in the same year • Treatment to raise HDL levels has failed to show benefit

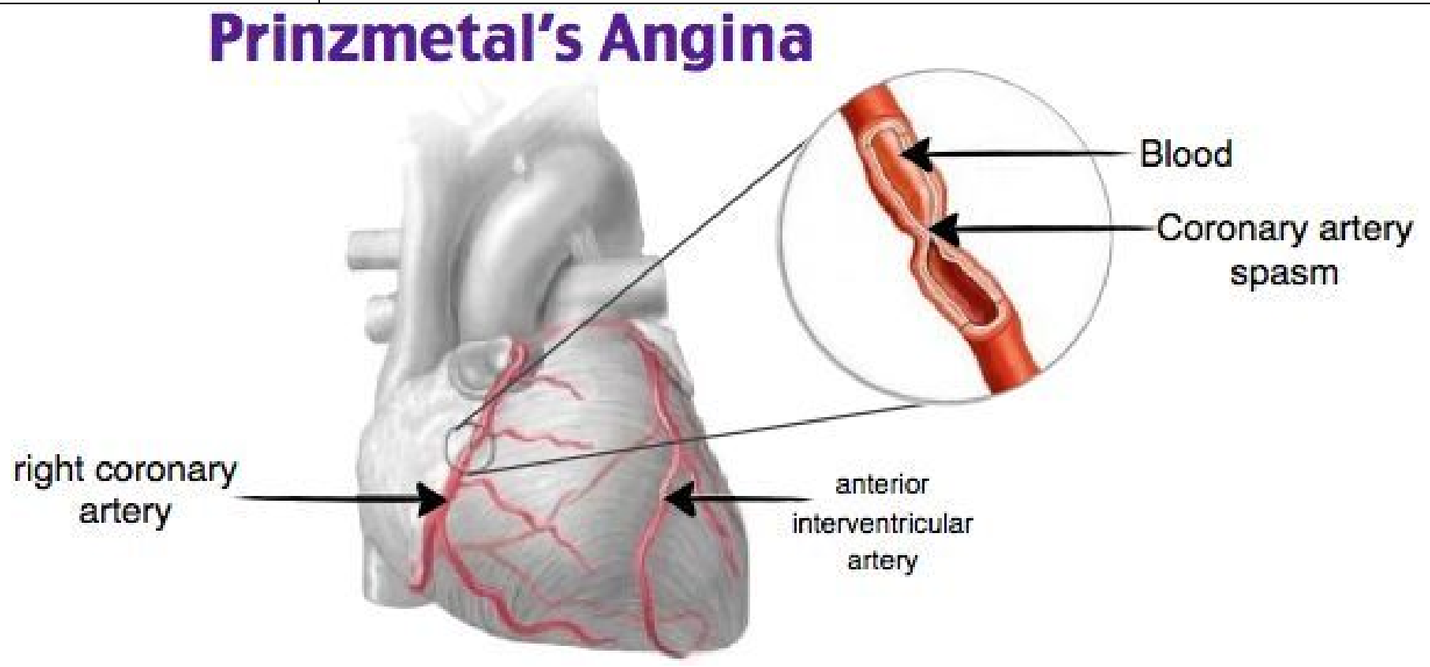
Stable Angina

- fixed coronary plaques causing >70% stenosis will present clinically
- predictable episodes chest pain and pressure worse with exertion
  - stable fixed atherosclerotic plaques cause stable angina
  - involves transient subendocardial ischemia
- typical symptoms of stable angina include chest pressure/pain which can radiate to the left arm and chin, diaphoresis, and dyspnea (elderly, diabetic, female patients may have minimal/atypical sx!)
  - relieved with rest or nitroglycerine

Vasospastic (Prinzmetal) Angina

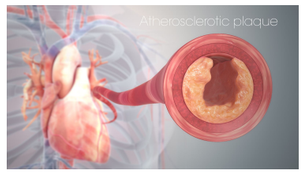
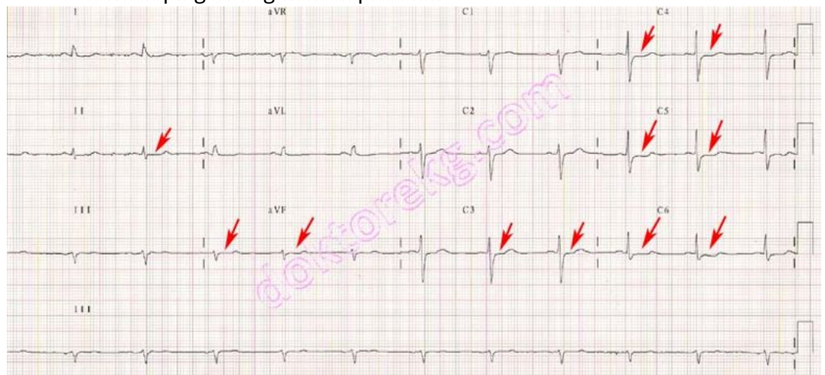
- vasospastic (Prinzmetal) angina symptoms occur at rest, more commonly at night; caused by transient coronary vasospasm
  - likely due to smooth muscle hyper-reactivity in the coronary artery wall
  - commonly occurs over a stable atherscleotic coronary plaque (may occur in disease free vessels!)
- Treat with nitroglycerine; calcium channel blockers are the first-line therapy for long term management of vasospastic (Prinzmetal) angina (these drugs will vasodilation and decrease spasticity)





Angina Pectoris

due to stable plaque → stress induced stable angina
- OR-
due to vasospasm of the coronary artery → random vasospastic angina (prinzmetal)

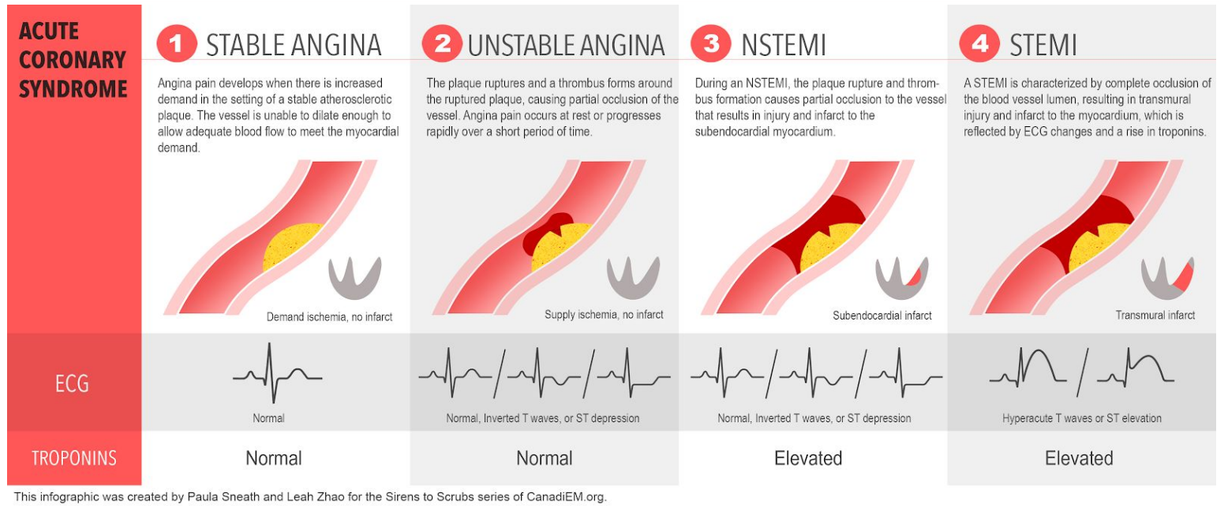
Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment	Prognosis
• **Precordial chest pain**, usually precipitated by stress or exertion • **Described as crushing** tightness, squeezing, pressure, burning, aching, indigestion • Distress is variable and **person knows what precipitates pain,** but usually constant for the individual • **Short duration** of attacks • Coronary vasospasm may occur (leads to **nocturnal angina attacks)**	• **BP changes:** either elevation or hypotension may occur ○ Due to Epi/NR • **Gallop rhythm** may occur • **Apical systolic murmur** may occur	• Usually due to **atherosclerotic** heart disease  • **Syndrome X** • Search for signs of associated diseases (diabetes, HTN, peripheral artery disease) • **Cocaine**	• Abnormalities include old MI, non-specific ST-T changes, LVH • **Angiographic** demonstration of significant obstruction of major coronary vessels • **Resting ECG usually *normal*** • **Exercise ECG Testing**: ○ Most commonly used *noninvasive* procedure for evaluating for inducible ischemia ○ Often **combined with imaging studies** (nuclear or echocardiography) ○ Done on a motorized treadmill or with a bicycle ergometer ○ ***Bruce protocol*** increases the treadmill speed and elevation every 3 minutes until limited by symptoms ○ **At least two ECG leads should be monitored continuously** • During anginal episodes, as well as during asymptomatic ischemia, the characteristic ECG change is horizontal or downsloping ST-segment depression that reverses after the ischemia disappears ○ ECG criterion for a positive test is 1-mm (0.1-mV) horizontal or downsloping ST-segment depression  **<u>Imaging to see perfusion</u>:** • ***Myocardial perfusion scintigraphy*** – uses thallium-201 or technetium-99m; ***radionuclide uptake proportional to blood flow***; will not reach areas that do not have good blood flow • Radionuclide angiography (Multi-Gated Acquisition Scan – MUGA) – images LV and measures EF & wall motion (means you can see the ventricles contracting) • Stress echocardiography - demonstrate exercise-induced segmental wall motion abnormalities; pharmacologic stress with high-dose dobutamine can be used as an alternative to exercise • Positron emission tomography (PET) – demonstrate perfusion of myocardium • CT & MRI Scanning • Electron Beam CT (EBCT) – quantify coronary artery calcification • Cardiac MRI with Gadolinium - high-resolution images of the heart and great vessels without radiation exposure or use of iodinated contrast media • ***<u>Coronary angiography – definitive diagnostic procedure for CAD</u>***	• **Rest or nitrates** • Sublingual nitroglycerin is the medication of choice for acute management: **decrease arteriolar/venous tone, reduce preload/afterload, lowers oxygen demand of heart** • Preventing further attacks: **limit aggravating factors**, nitroglycerin, **long-acting nitrates (isosorbide), beta-blockers, calcium channel blockers**, platelet inhibitors (ASA, clopidogrel), revascularization	• One infarction or death per 1000 tests • Many of the traditional exclusions, such as recent myocardial infarction or heart failure, are no longer used if the patient is stable and ambulatory, but symptomatic aortic stenosis remains a relative contraindication • 60-80% of those with anatomical significant CAD will have positive stress ECG, 10-30% without significant CAD will have also be positive

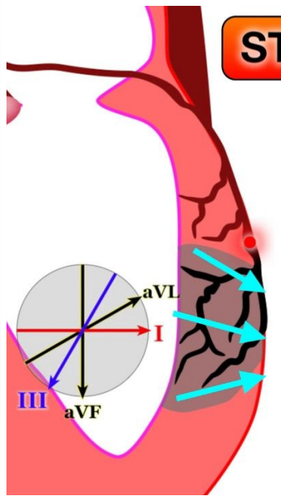
Acute Coronary Syndrome

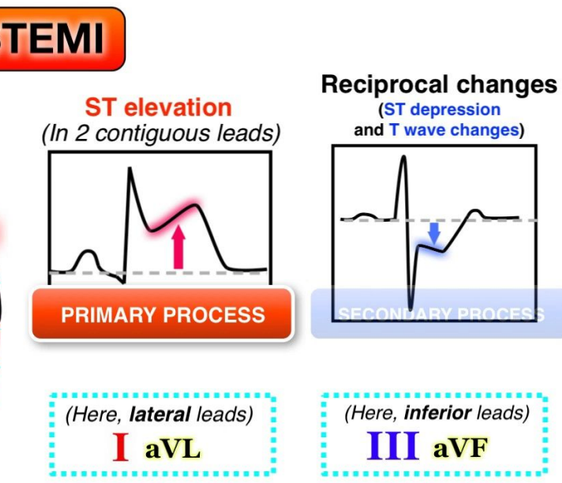
destabilized coronary plaque → partially/fully occlusive thrombus → cardiac ischemia or infarction

Unstable Angina	NSTEMI	STEMI
• Can arise even if thrombus is partially occlusive, occurs even at rest • Thrombus is transient, so symptoms come and go quickly ○ Requires immediate work up cause prolonged ischemia leads to infarction	• Ruptured plaque with overlaying <u>partially occluding</u> thrombus leads to myocardial cell death ○ ST depression localized to particular areas in the heart on the EKG	• Ruptured plaque with fully occlusive thrombus leading to full myocardial infarct, this is called a transmural infarct ○ ST elevations means that entire areas of a heart are affected; will have initial hyperacute T waves that elevates over time, the Q wave deepens (sometime indicative of prior infarcts), and then the T wave inverts

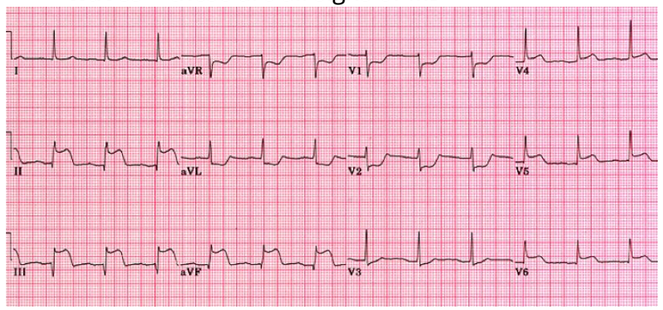
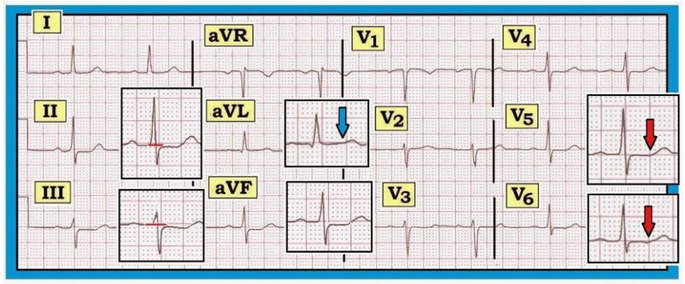
Dr. Beckerman: In acute MI and following reperfusion (thrombolytics), an ECG will commonly show Accelerated Idioventricular Rhythm







NSTEMI

Common symptoms	Common Signs	Labs	Diagnostic Testing	Treatment	Prognosis
• similar to those described in stable angina • 1/3 of MI patients do not have chest discomfort: elderly, women, diabetic	• CHF signs	• Initially cardiac enzymes may be normal ○ Cardiac markers include myoglobin, CK-MB, Troponin I & T	**ECG**: ***may show ST elevation (necrosis), ST depression (ischemia), flattened or inverted T waves*** = these are all signs of MI  Above EKG has ST elevations in 2,3, and F; pt is having an inferior wall MI  	• Antiplatelet and anticoagulation • **General recommendations**: bed rest, monitoring, admission if moderate risk, supplemental oxygen • **Antiplatelet therapy (prevents clotting)**: ASA, clopidogrel, prasugrel • **Glycoprotein IIB/IIIA inhibitors**: tirofiban, eptifibatide • **Anticoagulants (breaks clots)**: heparin, LMWH (enoxaparin), fondaparinux, thrombin inhibitors (bivalirudin) • **Nitrates: Nitroglycerin** • Beta Blockers • Calcium Channel Blockers • Statins	• 10% have signs of CHF: increased risk of death

Why would we not use fibrinolytic therapy in NSTEMI?

Dr. Beckerman: There is a greater risk in giving fibrinolytic therapy because it can release a thrombosis possibly leading to a brain aneurysm. It is more indicated in STEMI because at this point the patient is at risk of dying [non-STEMI is subendocardial, whereas STEMI is transmural] with STEMI; therefore benefits outweigh the risk.

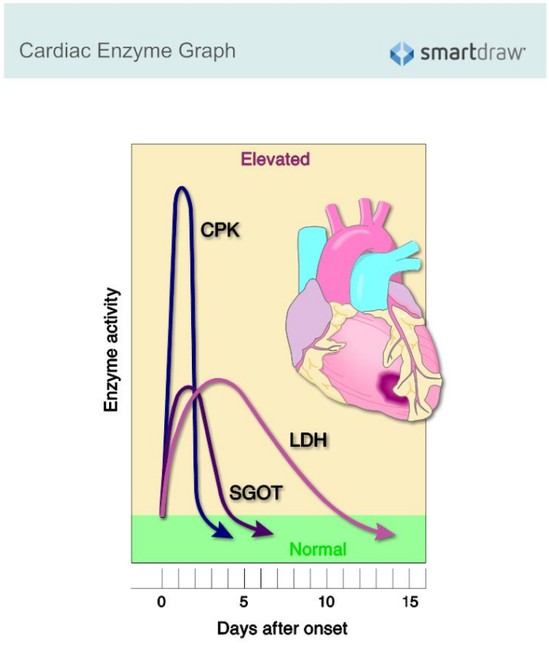
Cardiac Enzymes

When an infarction occurs the myocyte cell membrane will rupture releasing enzymes that can be measured. The most sensitive of these are Troponin and Creatine Kinase (CK-MB). Never send someone home after one set of enzymes. Wait 2-3 hours and then do another set. Usually you will look at troponins first. Serum Troponin T levels will peak in 24 hours and stay elevated for 7 days. CK-MB peaks in 24 hours and disappears in 24 hours after peak (48 hours total). CK-MB is better for measuring re-infarction.

Troponin test	The most sensitive and specific test for myocardial damage. Because it has increased specificity compared with CK-MB, troponin is a superior marker for myocardial injury.	12 hours	Troponin is released during MI from the cytosolic pool of the myocytes. Its subsequent release is prolonged with degradation of actin and myosin filaments. Isoforms of the protein, T and I, are specific to myocardium. Differential diagnosis of troponin elevation includes acute infarction, severe pulmonary embolism causing acute right heart overload, heart failure, myocarditis. Troponins can also calculate infarct size but the peak must be measured in the 3rd day. After myocyte injury, troponin is released in 2–4 hours and persists for up to 7 days.
Creatine Kinase (CK-MB) test	It is relatively specific when skeletal muscle damage is not present.	10–24 hours	The CK-MB isoform of creatine kinase is expressed in heart muscle. It resides in the cytosol and facilitates movement of high energy phosphates into and out of mitochondria. Since it has a short duration, it cannot be used for late diagnosis of acute MI but can be used to suggest infarct extension if levels rise again. This is usually back to normal within 2–3 days.
Lactate dehydrogenase (LDH)	LDH is not as specific as troponin.	72 hours	Lactate dehydrogenase catalyses the conversion of pyruvate to lactate. LDH-1 isozyme is normally found in the heart muscle and LDH-2 is found predominantly in blood serum. A high LDH-1 level to LDH-2 suggest MI. LDH levels are also high in tissue breakdown or hemolysis. It can mean cancer, meningitis, encephalitis, or HIV. This is usually back to normal 10–14 days.
Aspartate transaminase (AST)			This was the first used.[6] It is not specific for heart damage, and it is also one of the liver function tests.
Myoglobin (Mb)	low specificity for myocardial infarction	2 hours	Myoglobin is used less than the other markers. Myoglobin is the primary oxygen-carrying pigment of muscle tissue. It is high when muscle tissue is damaged but it lacks specificity. It has the advantage of responding very rapidly,[7] rising and falling earlier than CK-MB or troponin. It also has been used in assessing reperfusion after thrombolysis.[8]

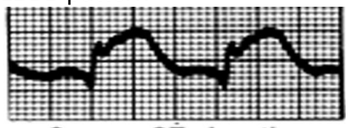
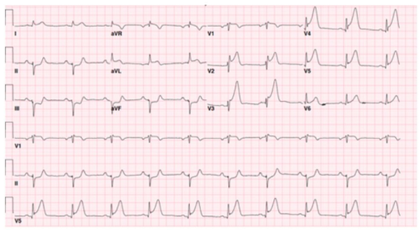
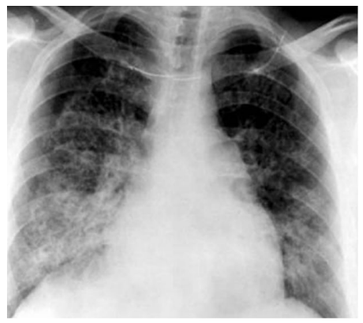
Cardiac enzymes

Enzyme	Initial rise	Peak	Back to Normal
Myoglobin	<2 h	6-9 h	1 day
CK-MB	3-6 h	12-24h	2-3 days
Troponin I	<4hrs	14-24hrs	3-5days
AST	Raises after CPK	48hrs	4-5days
LDH	24-48 h	2-3 days	5-10 day



Graph of elevated enzyme activity versus days after onset of cardiac infarct.

STEMI

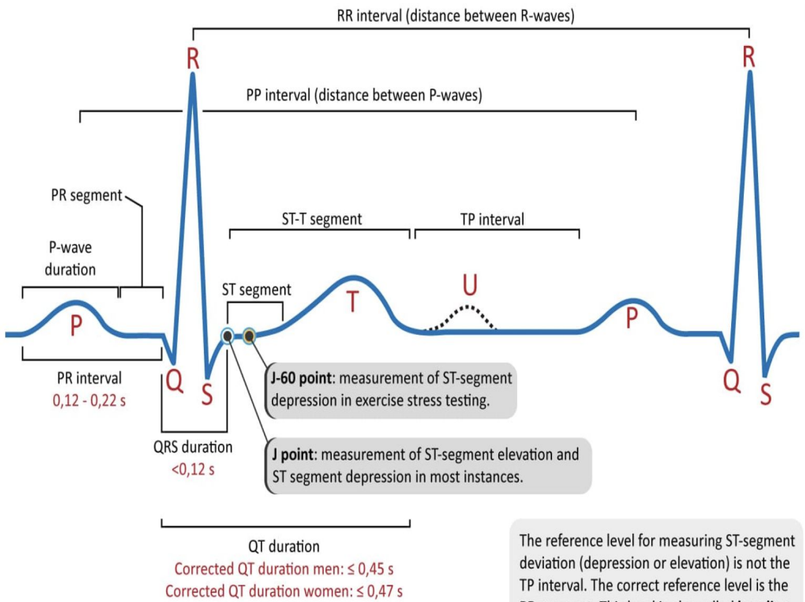
Common symptoms	Common Signs	Labs	Diagnostic Testing	Treatment	Prognosis
• Sudden onset of prolonged anterior chest discomfort**;** sometimes there is a gradual worsening of angina pattern (3 blocks of walking and CP, turns to 1 block of walking with CP) • **Infarction Pain**: Most common at rest & in morning ○ *No change* with nitroglycerin (NTG) • **Associated Symptoms**: (1) Diaphoresis, (2) Weakness, (3) Apprehension, (3) Syncope, (3) Dyspnea, (4) Cough (5) Wheezing (6) Nausea/vomiting • **Painless Infarction:** 1/3 of patients have no chest pain; ○ More serious with worse outcomes, especially in older patients, women, diabetics ○ Can masquerade as acute HF, syncope, stroke, or shock	• **General**: (1) Diaphoresis (2) Abnormal heart rate (3) low or high BP (4) Respiratory distress (heart failure) • **Heart**: (1) Cardiac exam unimpressive or greatly abnormal: JVD (if some HF), soft heart sounds, S4 gallop • **Extremities**: (1) Edema usually not present; cyanosis/cold extremities due to low cardiac output • **Who's at risk?** Watch out for pts with pneumonia, pericarditis, aortic dissection, pneumothorax & panic attacks [hyperventilation syndrome] as they all are risk factors for STEMI	• **Cardiac Specific Biomarkers:** Troponin I, Troponin T, CK-MB	• **ECG**: Extent of changes indicative of seriousness; peaked T waves, ST elevation, Q waves, inverted T waves; new LBBB pattern  Convex ST elevation in myocardial infarction Convex ST elevations are worse than concave ones.  Acute anteroseptal MI • **CXR**: Signs of CHF (later); look for signs of aortic dissection as alternative diagnosis  • **Echocardiography**: Assessment of LV function; looking for a wall that is hypokinetic (not moving as well)	• **Antiplatelet Drugs**: ASA, Clopidogrel, Prasugrel • ***Reperfusion Therapy:** STEMI - primary ***percutaneous coronary intervention*** (PCI) or ***fibrinolytic therapy*** (not useful without ST elevation); fibrinolytics include tenecteplase, reteplase, alteplase, streptokinase ○ Primary PCI (a stent) within 90 minutes of first medical contact is the goal and is superior to fibrinolytic therapy. • **General Measures:** Cardiac monitoring (ASAP) and place on low flow oxygen • **Analgesia**: NTG, IV opioids (vasodilation) • **Beta Blockers:** Modest benefits (except in CHF, hypotensive, or asthmatics) • **Nitrates**: Lowers BP & relieves pulmonary congestion ○ Do not use in patients who have taken Viagra, Cialis etc • **ACE inhibitors:** Short & long term benefit; especially helpful in EF <40%, large infarctions, heart failure • **Angiotensin Receptor Blockers**: May reduce mortality in heart failure • **Calcium Channel Blockers:** Seem to be *contraindicated* in MI → May exacerbate ischemia & cause death	• **Sudden Death/ Arrhythmias:** 50% of deaths occur prior to hospital – usually V. Fib

Common Rate and Rhythm Symptoms

- **Can be symptomatic**
  - **syncope, near-syncope, dizziness, palpitations**
- **or asymptomatic**

Rate and Rhythm Signs and Concerns

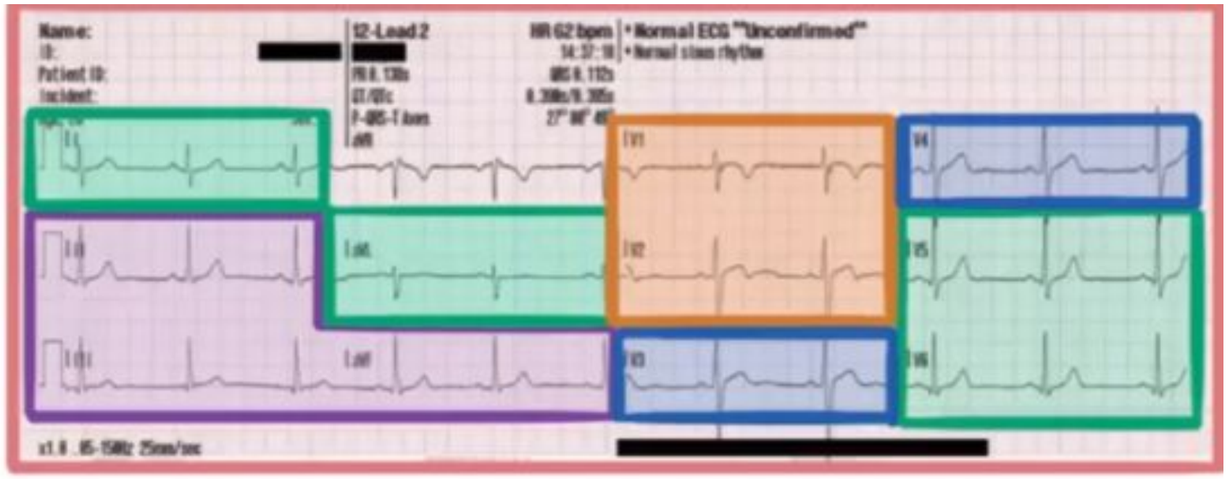
- **Can be lethal (sudden cardiac death) or dangerous (reduced cardiac output)**
- **Detected because of symptoms or found during monitoring**
- **Evaluated using ambulatory ECG monitoring, event recorders, exercise testing, electrophysiologic testing or tests of autonomic nervous system functioning (tilt-table testing)**

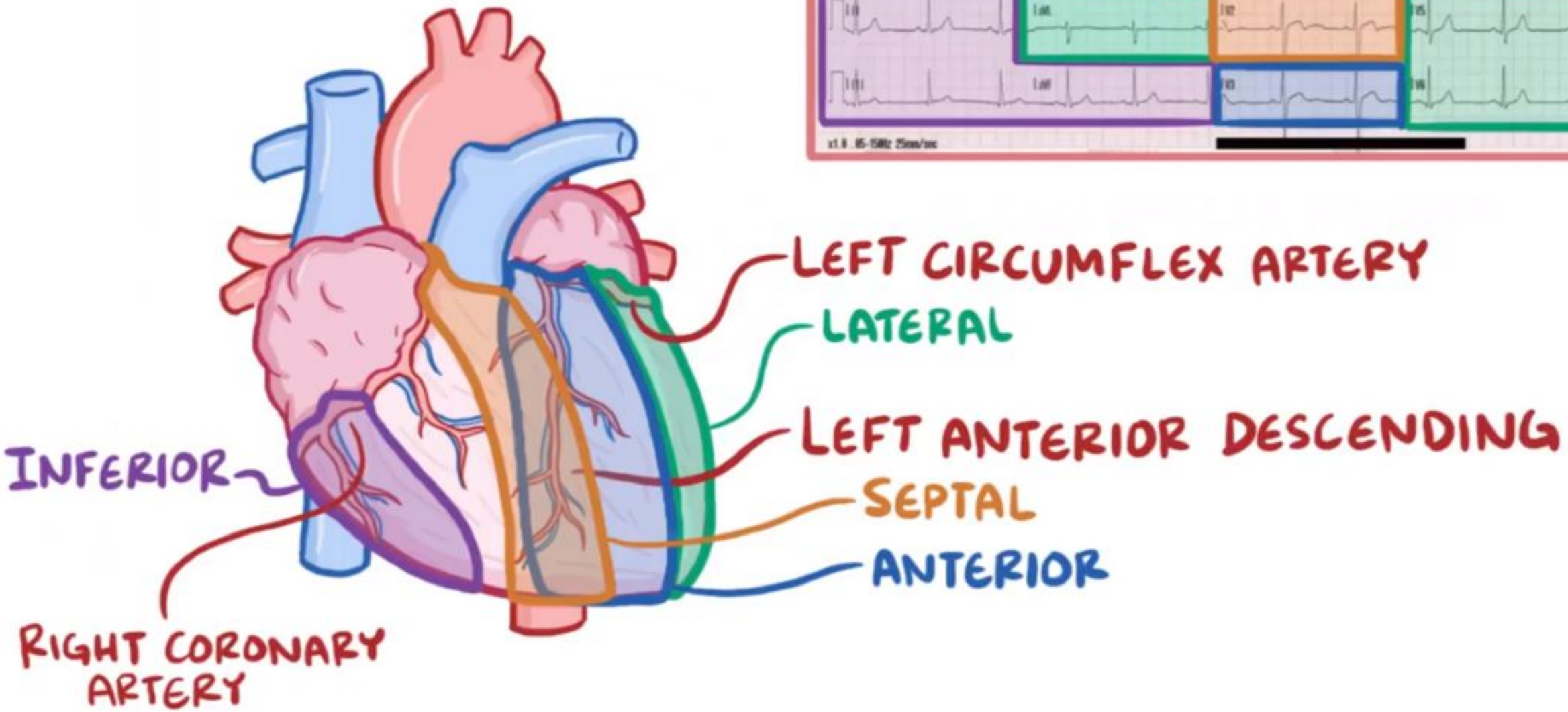


The reference level for measuring ST-segment deviation (depression or elevation) is not the TP interval. The correct reference level is the **PR segment**. This level is also called **baseline** level or **isoelectric level**.

Dr. Beckerman's rules for reading an ECG:

1. **What's the rate? Is it fast or is it slow?**
   300-150-100-75
   If QRS complexes are one box apart – HR is 300, next box over – 150, then 100, then 75
2. **Is it regular or irregular?** ***Regularly irregular*** –every 3rd beat you get a funny QRS complex || ***Irregularly Irregular*** – A-Fib
3. **Are there P-Waves?** May not see P-waves at every lead – look at all the leads (usually Lead 2 is the best one to see P waves)
4. **Is every P wave followed by the QRS? Is every QRS complex preceded by the P wave?**
5. **What's the PR interval? Normal is 0.2 sec – is it prolonged/shorter?**
6. **What is the width of the QRS complex? Normal is 0.12 sec (3 little boxes)**





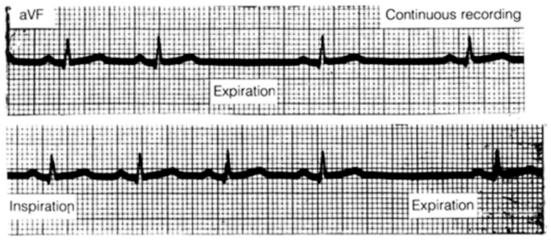
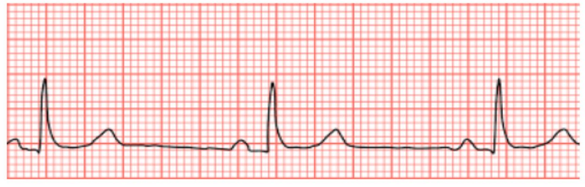
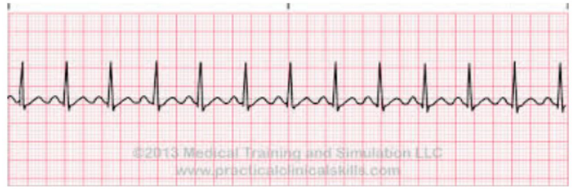
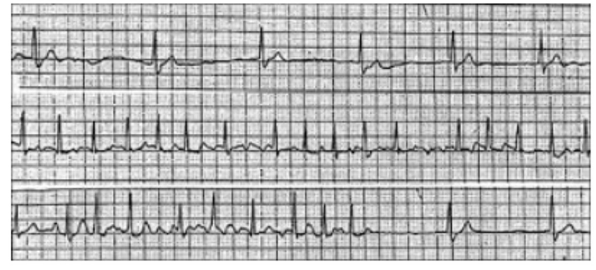
Leads II, III, and aVF are "inferior" leads because they're near the inferior wall of the heart, which receives blood from the right coronary artery

Leads I and aVL, along with two of the chest leads, V5 and V6, are considered "lateral" leads because they're near the lateral wall of the heart, which receives blood from the left circumflex artery

V1 and V2 are considered "septal" leads because they're nearest to the interventricular septum

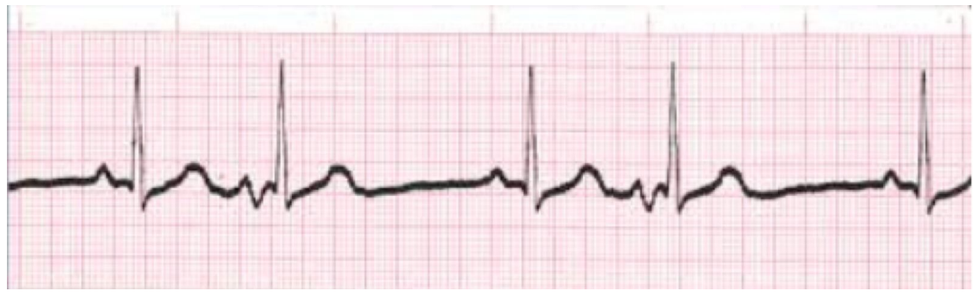
V3 and V4 are "anterior" leads because they're nearest the anterior wall of the heart. Both of the septal and anterior regions are served by the left anterior descending artery

Sinus Abnormalities

Sinus Arrhythmia	Sinus Bradycardia	Sinus Tachycardia	Sick Sinus Syndrome
• Cyclic increase in normal heart rate during inspiration & decrease during expiration • Common & normal in young and old patients ○ **Not a pathologic arrhythmia**  EKG indicates changes in rhythm; clinically it is not presented as dramatically as in this EKG	• **Heart rate** <60/minute caused by increased vagal influence on the normal pacemaker • **Rate** usually increases with **atropine**; well conditioned athletes may have slow heart rates • May cause weakness, confusion or syncope if extreme 	• **Heart rate** >100/minute • **Causes** ○ Fever, exercise, emotion, pain, anemia, heart failure, thyrotoxicosis, response to medications • **Rate** rarely exceeds 160/minute 	• Sinus node dysfunction • May see sinus bradycardia, sinus pauses, sinus arrest • Might sometimes become symptomatic: (1) syncope (2) lightheadedness. ○ Treat with pacemaker 

Atrial Abnormalities

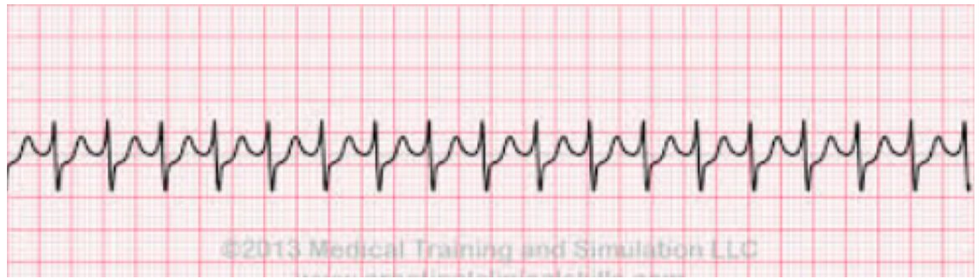
Atrial Premature Beats/ Premature Atrial Contraction (APCs)

Common symptoms	Common Presentation in ECG	ECG	Concerns	Treatment
• Usually asymptomatic • Most common Rhythm DIsturbance	• Ectopic focus in atria fires before the next sinus node impulse • Isolated interruption in regular rhythm; occurs frequently in normal hearts • **How do we know it's a premature beat?** P wave morphology usually differ from Sinus P wave morphology		• Development of **atrial fibrillation** • During prolonged refractory period heart fills up more & contracts stronger = **palpitations** • Decreased Cardiac output when heart does not have enough time to fill	• Rarely indicated However, it's a good idea to repeat lead II EKG

Dr. Beckerman: An irritable spot in a particular spot in the atria causes depolarization independently → sending an electrical wave through the atria → leading to the formation of a p wave & premature QRS that follows; happens randomly and in normal hearts,

Paroxysmal Supraventricular (Atrial) Tachycardia (SVT)

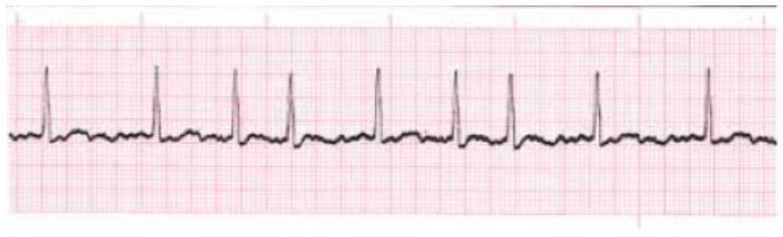
Treat with ADENOSINE 6 mg→ 10 mins →12mg→ 10 mins→12mg

Common symptoms	Common Presentation in ECG	ECG	Treatment
• Asymptomatic or ○ can have palpitations, lightheadedness, dyspnea, chest pain, diaphoresis • Abrupt onset/offset • Most common in young adults ○ Often in people with no heart disease • Syncope (rare)	• Rapid, regular rhythm • Narrow [normal - conduction through the V is normal, problem is in the atria] QRS complex tachycardia (rate 140-280) • **T wave** *is buried in the speed* of QRS complex		• Most resolve spontaneously Responsive to: • **Mechanical Measures:** (1) Valsalva, (2) Carotid sinus massage (beware of dislodging a clot), (3) facial contact with cold water • **Medications: (a)** ***Adenosine****(6mg, wait 10 mins, then 12 mg, wait 10 mins and then 12mg), **(b)** AV Node blockers (Verapamil); Diltiazem, Esmolol, Propranolol, Metoprolol ○ * Drug of choice via IV with NS. • **Cardioversion:** if hemodynamically unstable or medications fail (start at 100 joules)

Dr. Beckerman: (1) Normal Rate& Rhythm (2) Fast (3) Respond to Adenosine

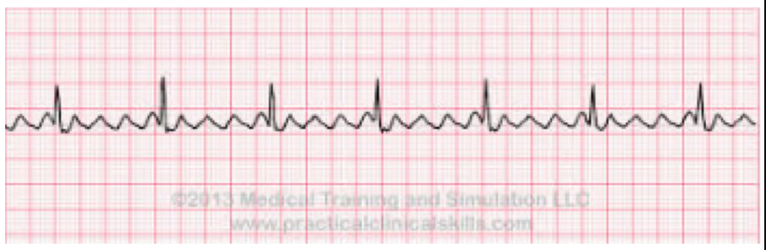
Atrial Fibrillation

Occurs in Mitral/Tricuspid Regurgitation, Mitral Stenosis, Atrial Septal Defect, Tetralogy of Fallot, Constrictive Pericarditis and Hypertrophic Cardiomyopathy

Common symptoms	Common Signs	Common presentation in ECG	ECG	Prognosis
• Usually tachycardic ○ Most common **chronic** arrhythmia; frequency increases with age • Palpitation (Acute Onset), hypotension, dyspnea • Fatigue (Chronic) • High incidence & Prev in elderly	• Irregularly irregular heart rhythm. • Appears in paroxysmal fashion before becoming an established rhythm	• **ECG** : Erratic atrial activity with irregular ventricular response; discreet QRS complexes in an irregular pattern; atrial activity may be fine or coarse • HR may present normal if pt is on B-Blockers [rate control]	 *Note the fibrillatory waves (F waves) instead of the organized p wave*	• AF *alone* rarely life threatening • High risk for thromboembolism • AF may lead to LV dysfunction, CHF or ischemia • CVA

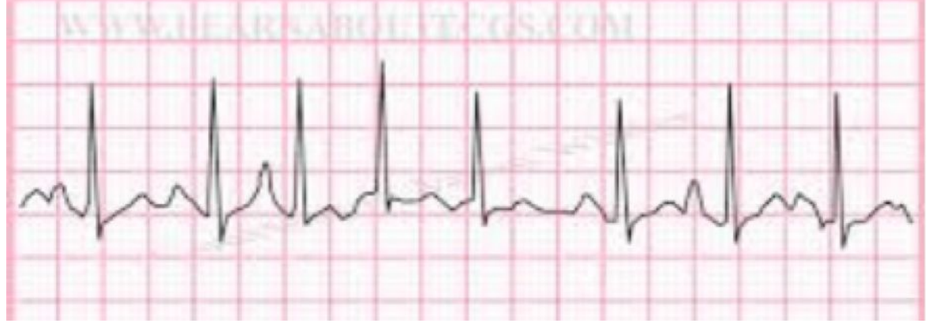
*** Dr. Beckerman: The goal of every cardiologist is to try to put the patient back into sinus rhythm, if that does not work just control the a-fib***

Atrial Flutter
Occurs in Tricuspid Regurgitation; Treat with Catheter Ablation

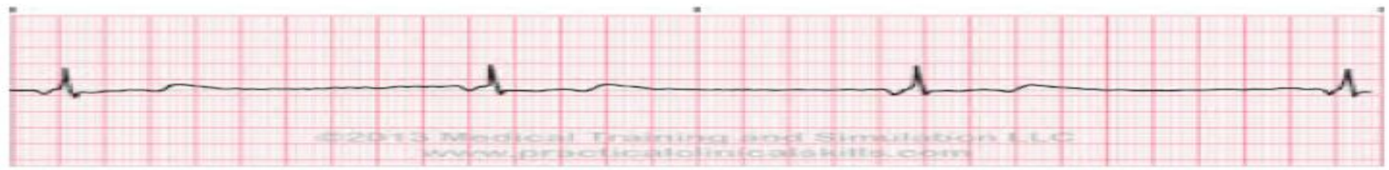
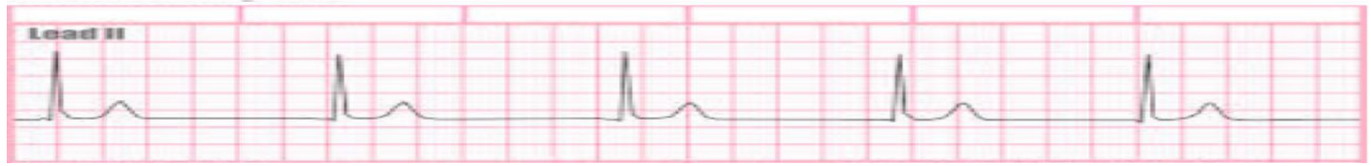
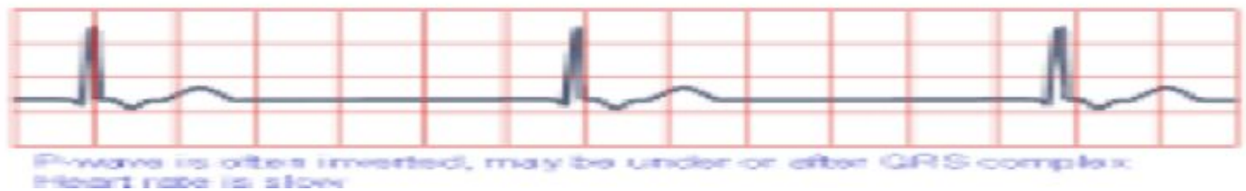
Common symptoms	Common Signs	Common presentation in ECG	ECG	Treatment
• Tachycardic (100-150 BPM) ○ Palpitations (Acute onset) ○ Fatigue (Chronic) • Often seen in conjunction with structural heart disease, rheumatic heart disease, CHF, ASD, COPD.	• Usually regular heart rhythm.	• **EKG**: Sawtooth pattern of atrial activity in lead II, III, aVF. • **Atrial Rates**: 250-350/min. • In a way, they're repetitive P waves [called f waves]	 *Note the Flutter waves masking the P and T waves; P waves and QRS waves are not married, there are numerous flutter waves between each QRS. No obvious baseline.*	• Utilize same medications as AF; rate control more difficult in flutter; chronic flutter is a difficult management problem; • **Catheter ablation has become the standard of care in chronic cases to destroy the abnormal myocyte that is creating the irregular electrical activity.**

* Dr. Beckerman: Flutter is markedly less common than Atrial Fibrillation. Remember when a rhythm gets worse or if the patient presents with a new rhythm, be suspicious of MI and send patient for cardiac enzymes.*

Multifocal Atrial Tachycardia (MAT)
Treat lung disease

Common symptoms	Common presentation in ECG	ECG	Treatment
• Palpitations • Severe COPD causes MAT ○ You can control the MAT by controlling COPD	• **EKG**: Varying P wave morphology (3 or more), which is the defining feature of MAT • Markedly irregular P-P intervals • **Heart rate:** 100-140/minute		• Treat the underlying lung disease

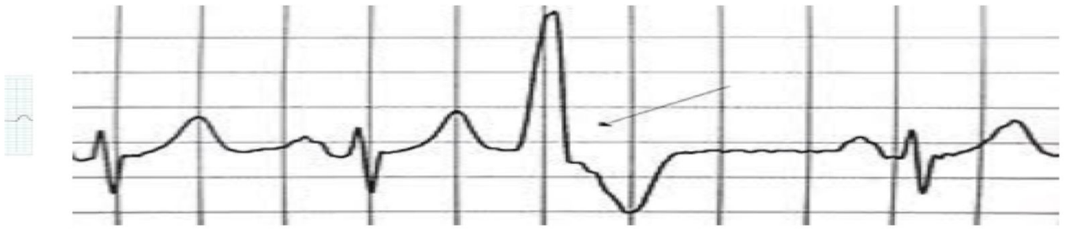
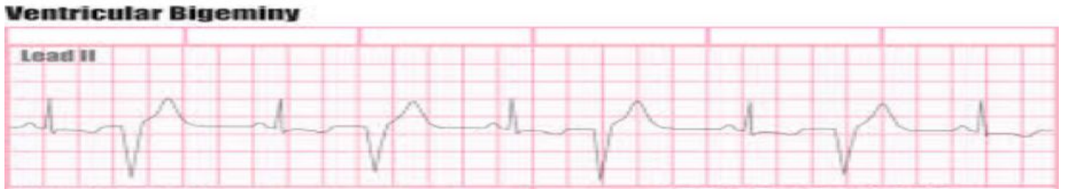
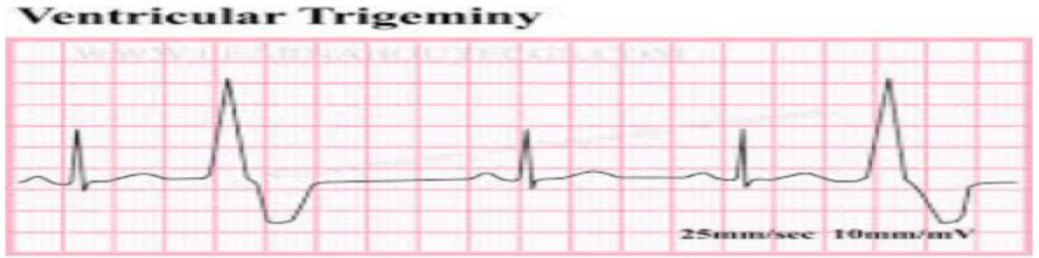
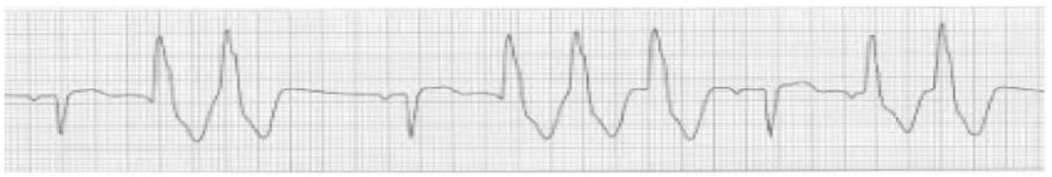
AV Junctional Rhythm

Common Signs	Common presentation in ECG	ECG
• Regular heart rhythm ○ HR usually 35-60/minute • May occur in normal hearts or in CAD, myocarditis, ○ Digitalis (Digoxin) toxicity; not used anymore due to small therapeutic window	• **P wave** ○ May be absent, ○ May precede or follow the QRS complex ○ Usually inverted in lead II; • Can have wide or narrow QRS complex **Causes** **Inverted** Impulses are starting high in AV junction (between AV node & Bundle of His) → Moves backwards into the atria leading to inverted P waves **Absent** Impulse starts in the middle of the AV junction but it takes longer to get back to the atria and is masked by QRS complex **Follow** Impulse happens below AV junction, takes longer to get to the atria and P wave is now following QRS.	 *P wave inversion - sinus, but not right (wrong direction of the current)*  *P wave is missing (signal is not coming from the sinus)*  *P wave after QRS complex: since the signal originated in AV junction, by the time it reached SA node & activated it, ventricular contraction already happened & V is now in refractory*

Ventricular Abnormalities - QRS complexes will no longer be normal

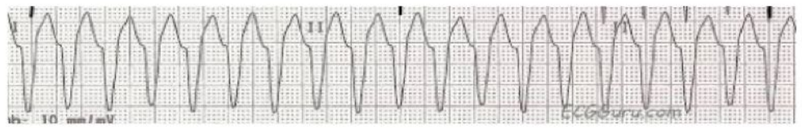
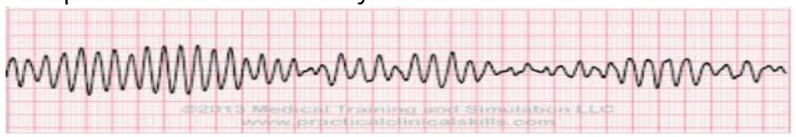
Ventricular Premature Beats (PVCs)

Isolated beats originating from ventricular tissues; *Treat with Treat with beta or Ca+ blockers*

Common symptoms	Common presentation in ECG	ECG	Treatment	Prognosis
• Patients may, or may not, sense the irregular activity Pathophysiology: https://www.ncbi.nlm.nih.gov/books/NBK507715/	• **Wide, bizarre looking QRS** complexes with no P wave ○ Wider than .12 secs • Bigeminy or trigeminy may occur; couplets may also be found • **R on T:** when ventricular extra beat happens during T-wave when the ventricle is supposed to rest but instead it fires (R onset)	 *Wider than .12 seconds and bizzare*  *Every other beat has a PVC*  *Every 3rd beat has a PVC*  *3 PVC in a row → Ventricular tachycardia*	• Exercise generally abolishes premature beats • Removing causative agent • **Palpitations: B-blockers, Ca channel blockers** • **Ectopic focus: radiofrequency ablation**	Sudden death more common when found in diseased hearts Ventricular rhythms are more dangerous

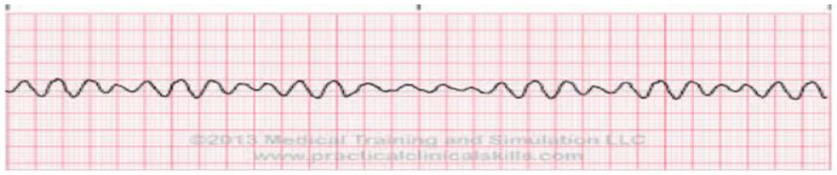
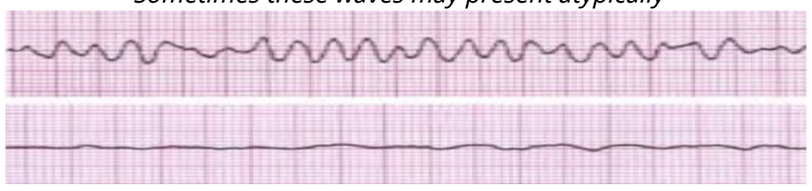
Ventricular Tachycardia (VT)

Due to meds prolonging QT or sever hypo K/Mg; Treat with ICD or IV mag

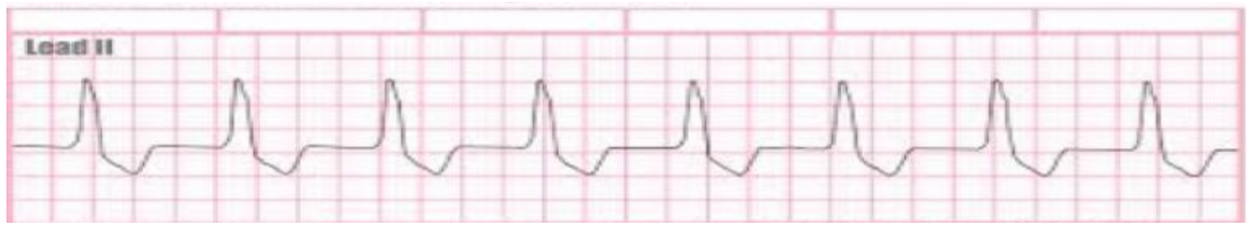
Common symptoms	Common Signs	Common presentation in ECG	ECG	Treatment	Prognosis
• Syncope • Hypotension • Vision Changes	• HR usually 160-240/minute • Vtach leads to a severely decreased cardiac output.	• Fast, wide QRS complex on ECG • Defined as 3 or more PVCs in a row • Often associated with structural heart disease	 monomorphic(reentrant) circuits - one single spot is responsible Polymorphic - multiple areas of pacemaker cells become irritated and develop diff levels of automaticity rate 	• **Implantable cardioverter defibrillator (ICD)** if no reversible cause • **IV Magnesium** to treat for Torsades de Pointes or a shock protocol	• **Torsades de Pointes** may occur in **severe hypokalemia** or after medications prolonging QT interval ○ **Torsades de Pointes:** Twisting of the points on an EKG ○ Hypomagnesemia from surgery. • Frequent complication of MI and cardiomyopathy; may occur in normal hearts

Ventricular Fibrillation (VF)

Treat with defibrillator

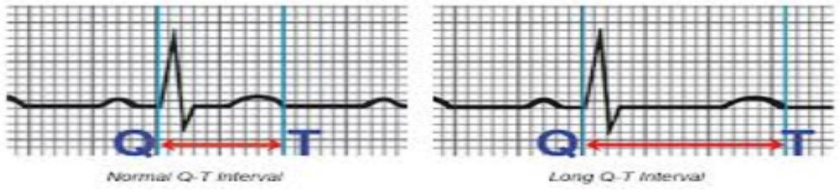
Common symptoms/signs	Causes	Common presentation in ECG	ECG	Treatment	Prognosis
• Disproportionate number occur in early morning • Deadly rhythm	• Usually caused by sudden onset of VF, often preceded by VT • Severe LVH, hypertrophic cardiomyopathy, aortic stenosis, pulmonary hypertension may predispose to VF	• Chaotic, bizarre complexes on ECG Patho development: • VPB → V-tach → V-Fib → death • Aortic Stenosis → V-fib	 *Sometimes these waves may present atypically* 	• Electrotherapy (Defibrillator)	• Sudden cardiac death defined as unexpected non-traumatic death in clinically stable patients • May be the first sign of CAD in up to 20%

Accelerated Idioventricular Rhythm

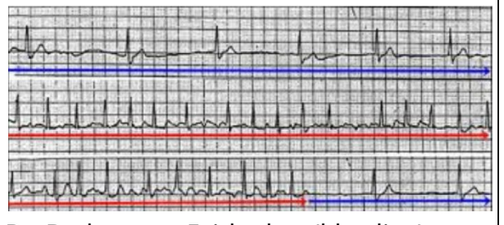
Causes	Common presentation in EKG	EKG
• Three causes: ○ Occurs commonly in acute MI ○ following ***reperfusion (thrombolytics)*** ○ Also common in digitalis toxicity *Technically this is *V-Tach but at a slow rate*	• Regular wide complex rhythm with rate of 60-120/minute	

Interval Changes

Long QT Syndrome

Causes	Common presentation in EKG	EKG
• May be congenital • Can be caused by medications ○ Antidepressants, Antiarrhythmics, Methadone • Electrolyte abnormalities • Myocardial ischemia • Can lead to Ventricular disturbances	• Uncommon disease with recurrent syncope, prolonged QT interval (0.5-0.7 seconds), documented ventricular arrhythmias & sudden death • Normal QT interval 0.36-0.44 seconds: QT interval varies by gender, age and heart rate • QTc corrects for heart rate – usually recorded on the ECG tracing	

Sick Sinus Syndrome
Treat with pacemaker

Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment
• Asymptomatic • Occasionally **syncope, angina, dizziness, lightheadedness, palpitations, confusion,** • symptoms are nonspecific and must be correlated temporally with the presence of SSS with an event recorder • More common in elderly and those with AF • Chronotropically impotent	• Recurrent supraventricular arrhythmia and bradyarrhythmia • heart failure • Concomitant atrial fibrillation	• **Medications** (digitalis, beta blockers, calcium channel blockers, antiarrhythmics) • **Common diseases**: Sarcoidosis, amyloidosis, cardiomyopathy • **Rare**: Coronary artery disease	 Dr. Beckerman: Fairly slow (blue line), then we speed up (red line) and then slow down again (blue line), and we might have a period of Sinus arrest	• Most symptomatic pts require a permanent pacemaker. ○ Permanent pacing does not decrease mortality, but may alleviate symptoms & improve quality of life.

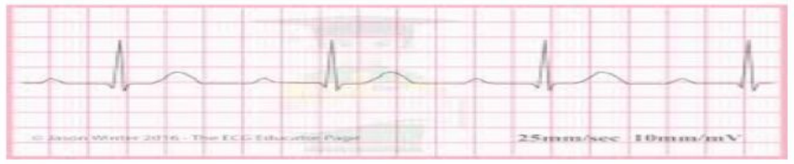
AV blocks *will definitely* be a written test question

AV Blocks (1º, 2º, & 3º) - All about PR!

Treat with pacemaker

1º

PR interval >0.20 seconds; all impulses are conducted to the ventricles



Dr Beckerman: In general every P wave is followed by a QRS wave. 1º AV block is a benign finding.

- Benign but worth asking yourself a question if it's a new development: Did the patient have an MI recently?

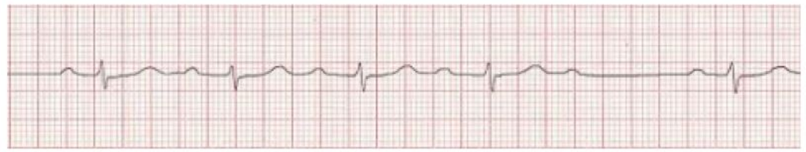
- Heart block is a type of arrhythmia; can be caused by damage/fibrosis of conduction system, ischemic heart disease, cardiomyopathies or myocarditis

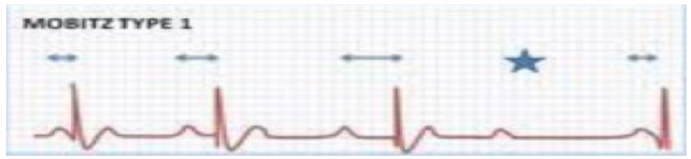
2º- Mobitz Type 1 (Wenckebach)

PR interval gradually lengthens and R-R interval slowly shortens

- Then there is a dropped beat (dropped QRS), there is no longer a 1 to 1 relationship between the P and QRS

Almost always due to abnormal conduction through the AV node

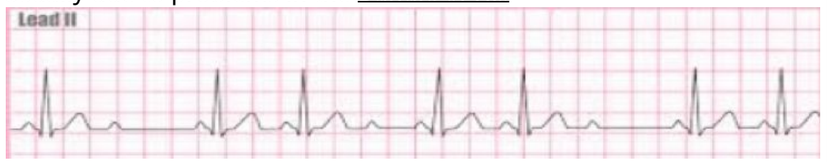


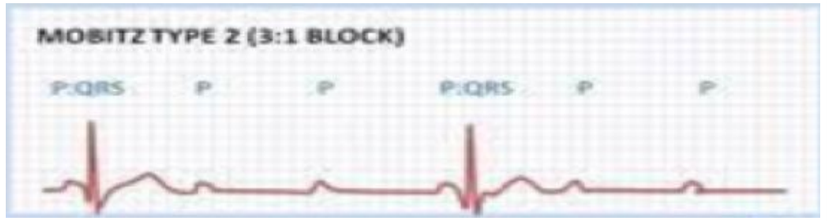


Mobitz I may occur in normal people
Causes: Abnormal function of AV node; May occur as a side effect of medications (digoxin, calcium channel blockers, beta blockers
Almost always due to organic disease
Prognosis Usually good; May progress to complete heart block
Treatment: May not necessarily need a **pacemaker**, depends on pt's case.

2º- Mobitz Type 2

PR interval stays the same but QRS randomly drops [no one to one conduction btw P & QRS - lonesome P wave]
Usually due to problems in the Bundle of His

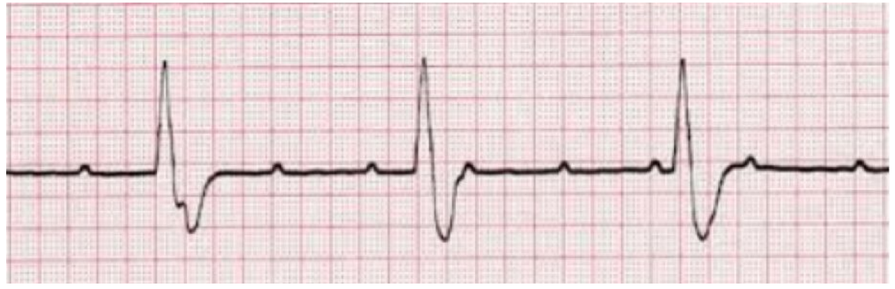




Treatment: Usually require **permanent** pacemaker

3º (Complete Heart Block) aka AV dissociation

- **Cause**: Advanced form of block due to lesion distal to the Bundle of His
- **ECG**: QRS wide and slow (usually below 50/minute) but regular. *QRS is widened because they are conducting their own electrical activity*.
  - ECG shows both an atrial and ventricular pacemaker
  - P waves and QRS complexes are each regular but unrelated to each other because they are going at independent rates. Therefore, there is no true PR interval due to this independent rate.
- **Symptoms:** Patients may be asymptomatic or have dyspnea, syncope, weakness; if pt is asymptomatic, they still need a pacemaker because they are at risk for cardiac arrest.
- **Treatment**: The patients almost always require **permanent pacemaker**: if this will be delayed, then temporary external pacing is indicated

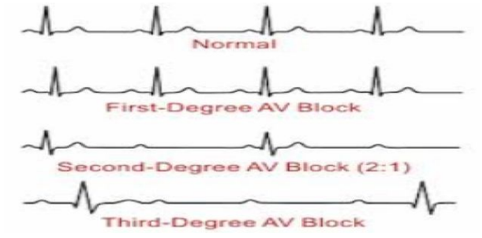


Only AV block where a P wave can be buried within the QRS complex

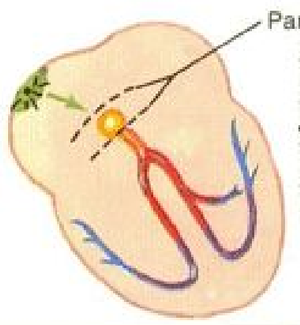
Complete heart Block. P waves "march through" the QRS complexes

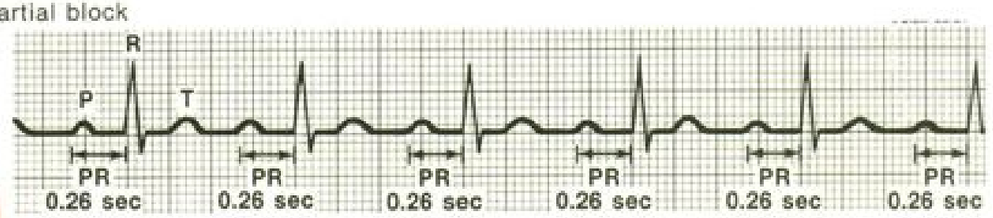
Supplemental Information:

- Lev's disease and MI cause problems with the conduction system at the AV node. The transmission from the AV node to the ventricles is affected.



1° AV Block





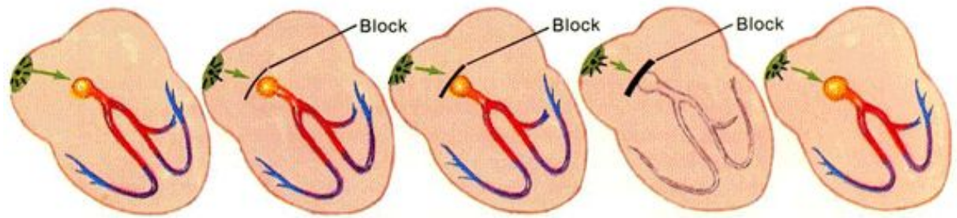
Rhythm: regular

Rate: (that of underlying rhythm)

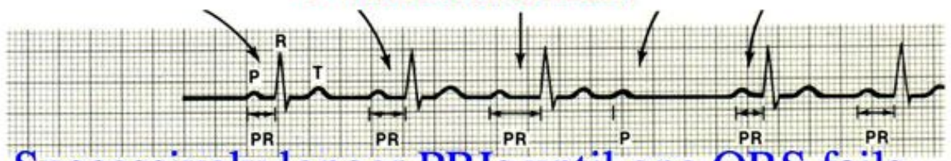
PRI is > than .2 seconds

QRS: usually normal

2° AV Block Mobitz I



P Waves look Similar!



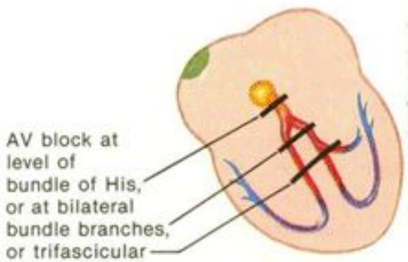
Successively longer PRIs until one QRS fails

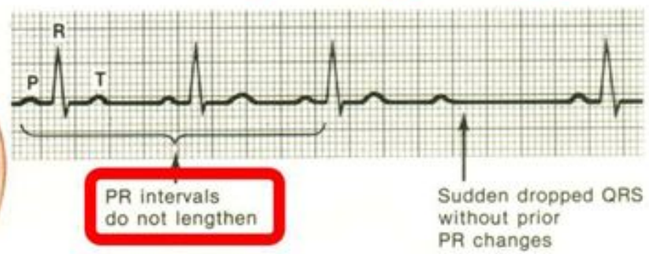
Rhythm (ventricular) is often irregular

Atrial rhythm is ~ **regular,** QRS is normal

2° AV Block Mobitz II

Suddenly dropped QRS





P waves are punctual and similar, **unlike a non-conducted PAC which is EARLY!**

Ventricular rhythm = irregular, atrial rhythm is regular

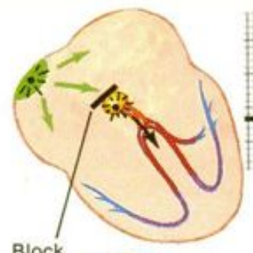
PRI normal or prolonged

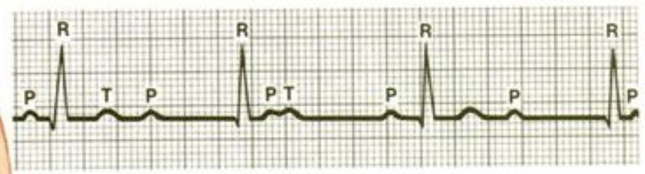
QRS: often abnormal

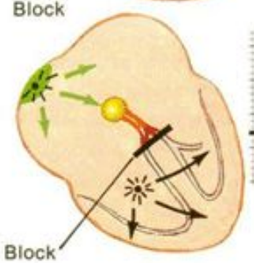
3° AV Block

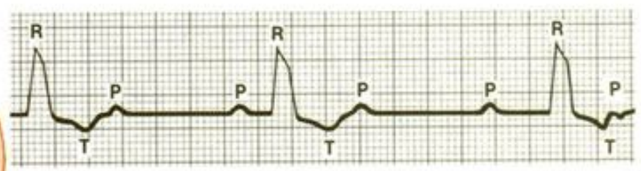
Atria and Ventricles are depolarizing independently

No association between atria & ventricles



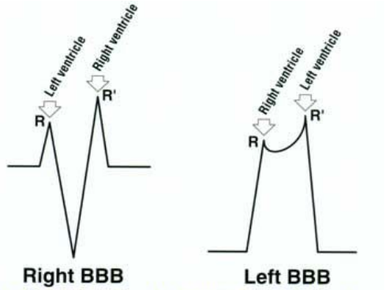
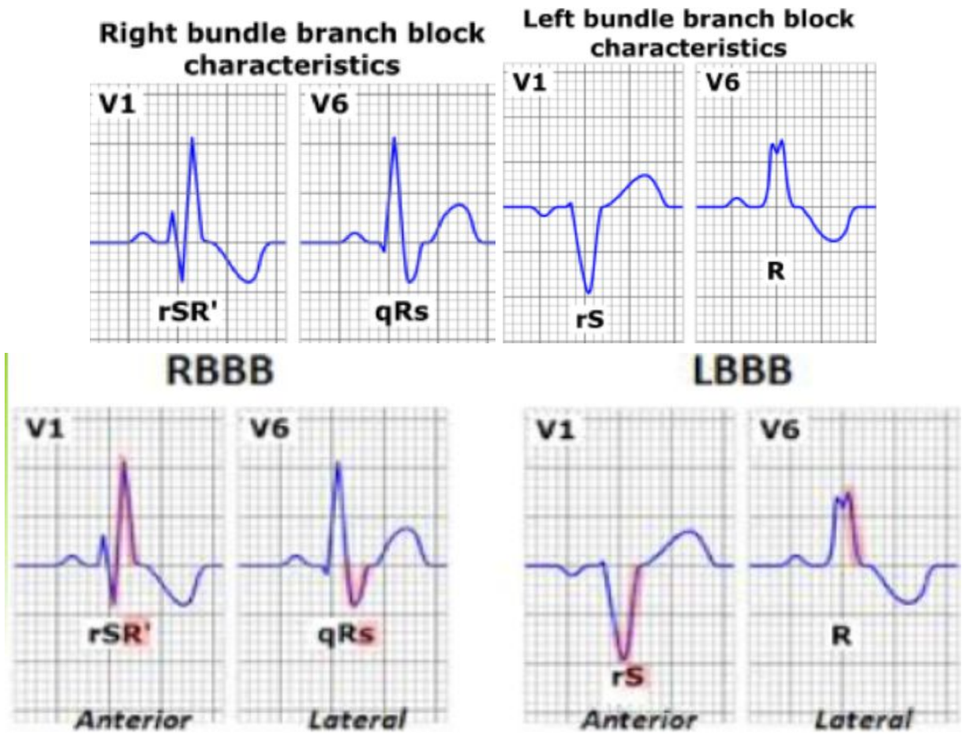


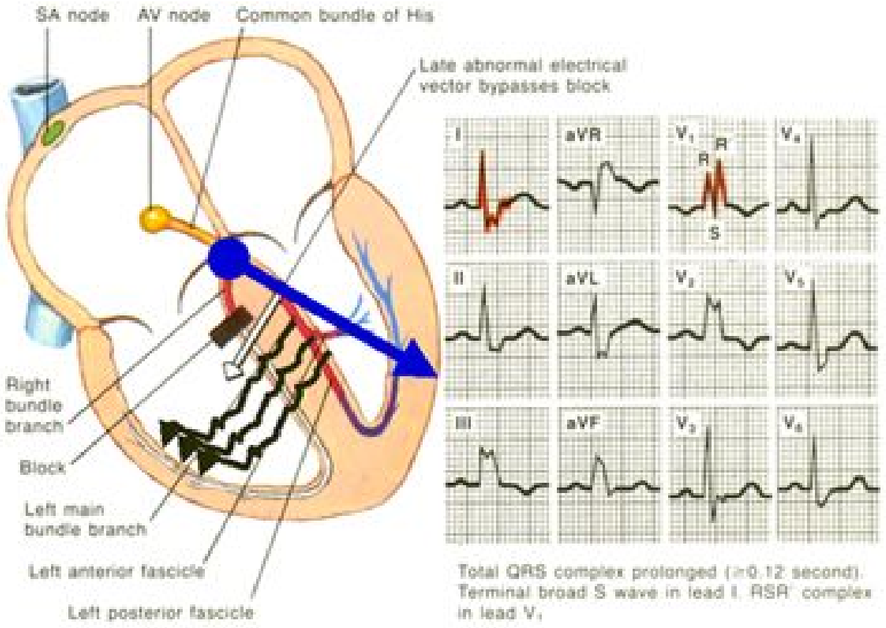




Intraventricular Conduction Defects

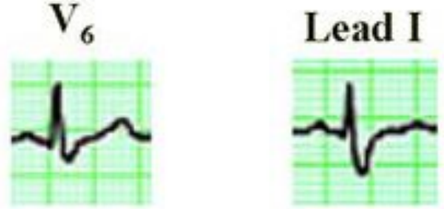
LBBB: Occurs in Syncope, Dilated Cardiomyopathy, and STEMI
RBBB: Occurs in Atrial Septal defect, Tetralogy, Pulmonary Valve Regurgitation

Common symptoms	Diagnostic Testing	Prognosis	EKG
• Common in pts with normal heart beats and many disease processes.	• **EKG**: Below the Bundle of His the conduction system divides into 3 fascicies: (1) right bundle, (2) left bundle, which further divides anterior & posterior fascicles ○ Conduction blocks in any of these fascicles can be observed on the EKG. • With a RBBB or LBBB, ventricular contraction is not synchronized. ○ Leads to **widened QRS complex** (> .12 seconds)	• Prognosis depends on the underlying disease process. • LBBB is associated with a higher risk of developing cardiac disease. **EKG Findings** • **RBBB** is better visualized on V1. You will have two uneven R waves one after another & widened QRS complex (**RSr'**). • **LBBB** is better visualized in V6. QRS is widened and difference in the "**rabbit ears**" are less distinct.  R and R' in Right and Left BBB often look like above	

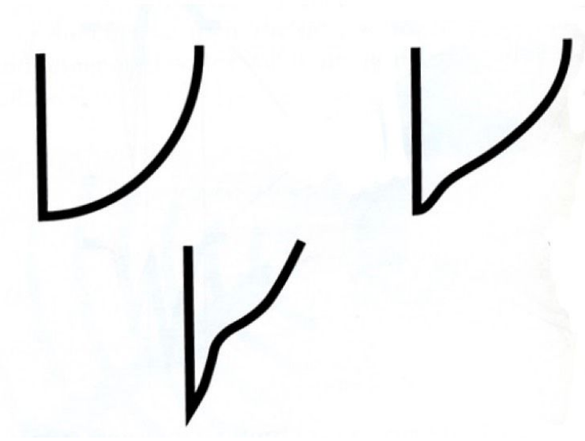


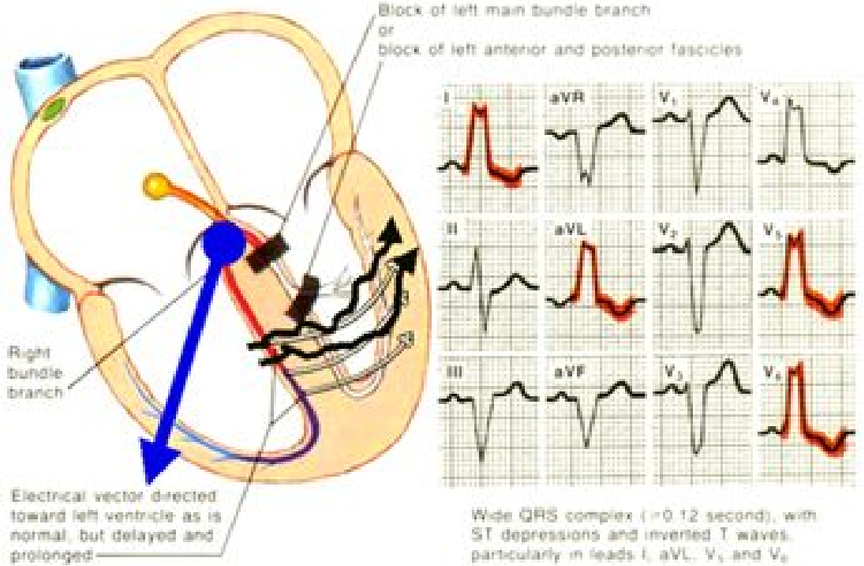
RBBB Criteria (Check QRS 1st)

- **Look in V_1 & V_2**
  - R, R¹ wave!
- **Look in V_5, V_6, & Lead I**
  - "slurred S wave"



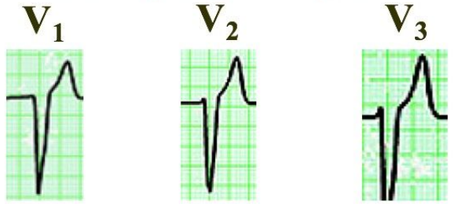
Example "Slurred S waves" in V_5, V_6 and Lead I





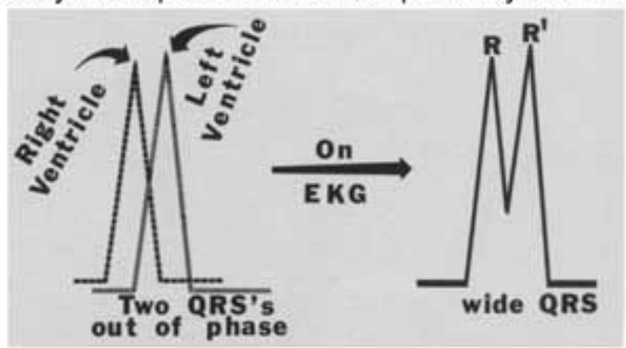
LBBB Criteria (Check QRS 1st if abnormal)

- **Look in V_5, V_6, (and/or Lead I)**
- 'blunted or stubby' QRS
- **Look in V_1-V_3**
  - predominately negative QRS



What happens to Right and Left ventricular depolarization if one bundle branch is blocked?

What do you suspect the QRS complex may look like?



Syncope

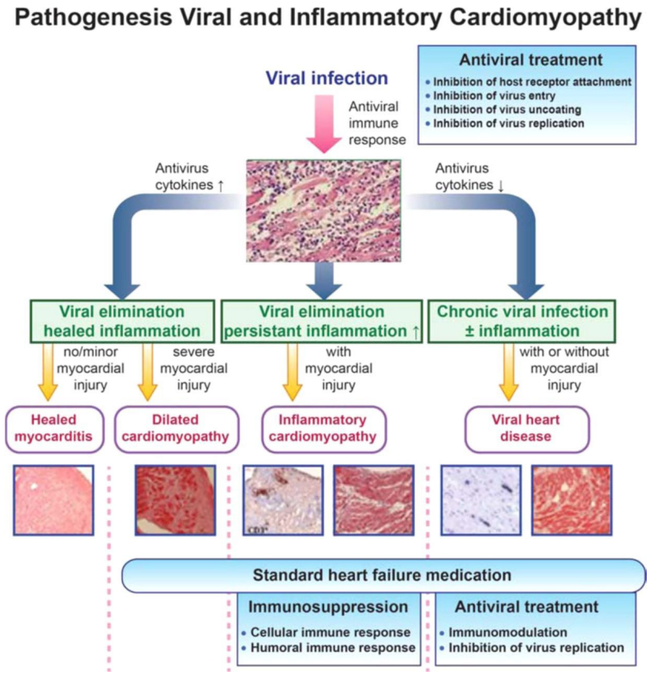
Common symptoms	Common Signs	Causes	Diagnostic Testing	Treatment	Prognosis
• Transient self limited loss of consciousness and postural tone from	• Nausea, diaphoresis, pallor, tachycardia are common • Episodes can be aborted by lying down	• Either vasopressor or cardiogenic causes • For many patients with recurrent syncope or near-syncope the cause is *not* dysrhythmia	• **ECG**: ○ Resting ECG recommended ○ **High risk findings**: non-sinus rhythm, LBBB, LVH ○ If rhythm disturbance suspected: continuous monitoring or event recorder • **Autonomic Testing**: ○ Useful when diagnosis unclear • **Electrophysiologic Testing** ○ Limited role especially when no structural abnormalities present ○ Diagnostic yield in patients with structural cardiac disease: 50% • **Exercise Testing**: when symptoms are associated with exercise or stress, exercise testing may be helpful	• Prompt recovery without resuscitative measures	• Prognosis usually favorable unless underlying heart disease present • History is most important component in evaluation of syncope

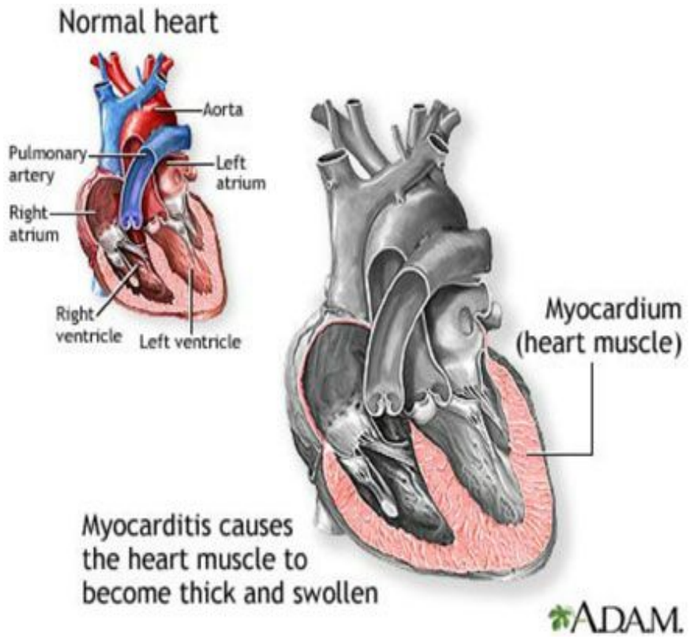
Myocarditis and Cardiomyopathies

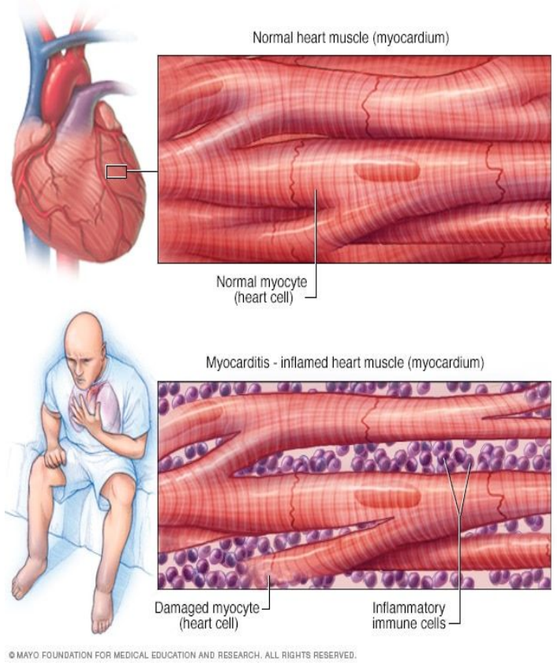
Infectious Myocarditis

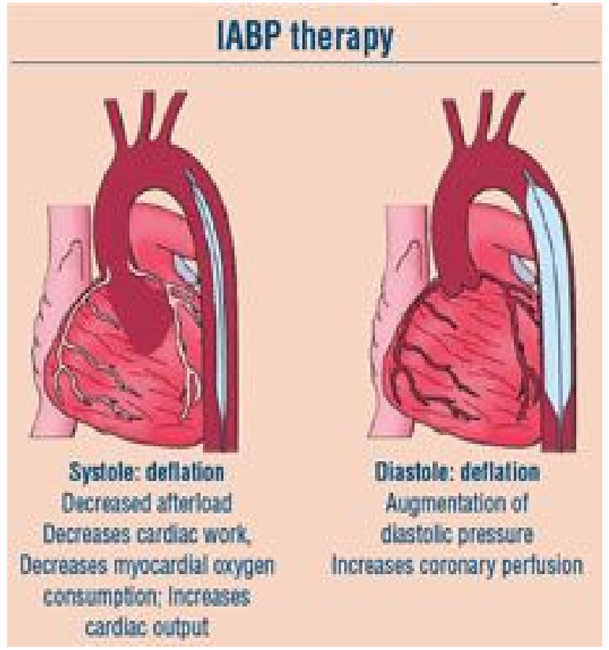
May mimic MI on ECG; check cardiac markers to r/o MI; and correct arrthymias
Causative Agent: Coxsackievirus, enterovirus, adenovirus, HIV, parvovirus, HHV 6, Chagas *(*parasite*: Trypanosoma cruzi)*, bacteria (strep), and fungi

Causes	Symptoms/Signs	Labs	Diagnostic Testing	Treatment
• Presumably caused by (1) acute viral infection or (2) post viral immune reaction • Genetic predisposition is a likely factor in some cases • Autoimmune myocarditis may occur without identifiable viral infection **Supplemental Info:** • anthracyclines (e.g. doxorubicin, daunorubicin) can cause free radical damage and myocarditis • Lyme myocarditis may develop after Lyme infection/ineffective Tx	**Symptoms** • Often follow URI or febrile illness • May present with chest pain (pleuritic or nonspecific) OR signs of signs of heart failure, which is the usual presentation (watch out for young ppl w CHF symptoms!). • Dyspnea, chest pain, arrhythmias may occur (may be position-dependent)ca **Signs** • Pericardial friction rub may be present • Tachycardia, gallop rhythm or other heart failure signs • Presentation ***may mimic MI*** (ST elevation, elevated cardiac markers, ventricular wall movement abnormalities)	• No specific lab findings consistently present, but WBC, ESR, CRP usually elevated ○ WBC can be elevated because the myocardium is damaged. **Endomyocardial Biopsy** • Confirmation still requires histologic evidence • Usually performed between 2 weeks and 2 months after onset of symptoms • Since abnormalities are patchy, diagnosis may be missed in 50% of cases	• **EKG**: Shows Cardiomeagly and abnormalities in contractility of ventricles. ○ Sinus tachycardia, other rhythm disturbances, non-specific repolarization changes, conduction abnormalities ○ Ventricular ectopy may be the only clinical finding. • **MRI** with ***gadolinium***, which can show spoty areas of injury because the ***gadolinium*** only spreads to certain areas. • **CXR**: Cardiomegaly frequent with signs of CHF	• Treatment directed towards the clinical scenario with ACE Inhibitors, beta blockers and, often non-steroidal anti-inflammatories ○ **Arrhythmias** should be suppressed with antiarrhythmic drugs • **Appropriate antimicrobial therapy** if a specific infecting agent is identified • ***Digoxin has little value & should be avoided*** • **Fulminant myocarditis requires** aggressive short-term support (IABP or LVAD) ○ **Intra Aortic Balloon Pump**









Noninfectious (Secondary) Myocarditis

Causes	Labs	Diagnostic Testing
• Secondary myocarditis caused by non-viral pathogens, medications, chemicals, physical agents or inflammatory diseases (such as SLE) • Medications, illicit drugs, toxic substances can cause myocardial injury • Systemic disorders also may cause myocarditis • Chemotherapy is a large cause of cardiomyopathies and is increasing	• Measure BNP levels	• Chemo patients should regularly be screened with **echocardiography, cardiac MRI**

Dilated Cardiomyopathy

Causes	Common Symptoms/Signs	Labs	Diagnostic Testing	Treatment	Prognosis
• Associated with HIV, rheumatologic disorders, sleep apnea, amyloidosis (causing end stage heart failure), sarcoidosis & end-stage kidney disease	• **S/S:** Similar to signs of heart failure • **PE**: Rales, elevated JVP, cardiomegaly, S3 gallop rhythm, edema or ascites	• BNP & other biomarkers • Myocardial biopsy rarely useful	• **Echo:** LV dilatation, thinning, and global dysfunction ○ Reduced ejection fraction (EF) below 40% with LV enlargement • **EKG:** Sinus tach, LBBB, atrial or ventricular arrhythmias • **CXR**: Cardiomegaly, pleural effusions, evidence of heart failure • **Exercise Stress Test** to r/o CAD.	• Manage HF with ACEIs, ARBs, beta blockers, aldosterone antagonist & diuretics. • CPAP may improve LV function • Control BP and rhythm disturbances • AICD/Pacemaker is reasonable • Digoxin a second-line treatment	• Severity of RV dysfunction is critical in long term • Accounts for 10,000 deaths yearly • Blacks affected 3 times more than whites • Poor prognosis: 50% mortality at 5 years • Multiple causes: 20-35% have familial component • Many cases are idiopathic

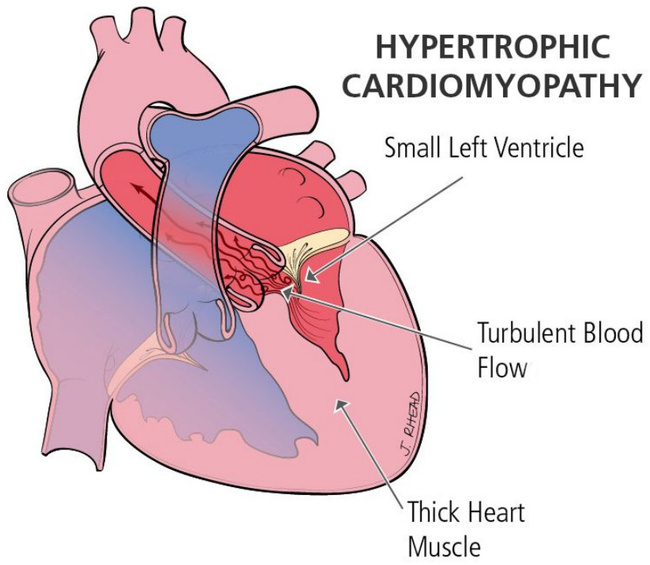
Stress Cardiomyopathy

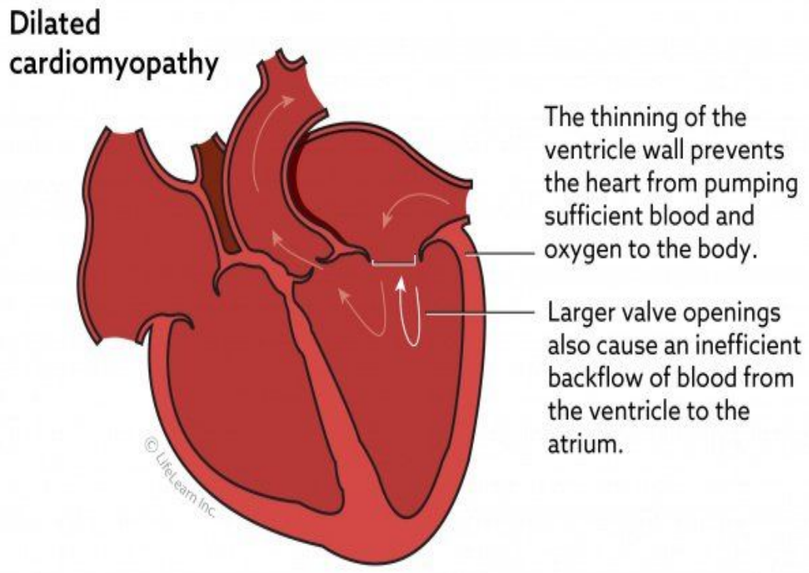
Epi surge; mimics MI on ECG but won't have any coronary abnormalities

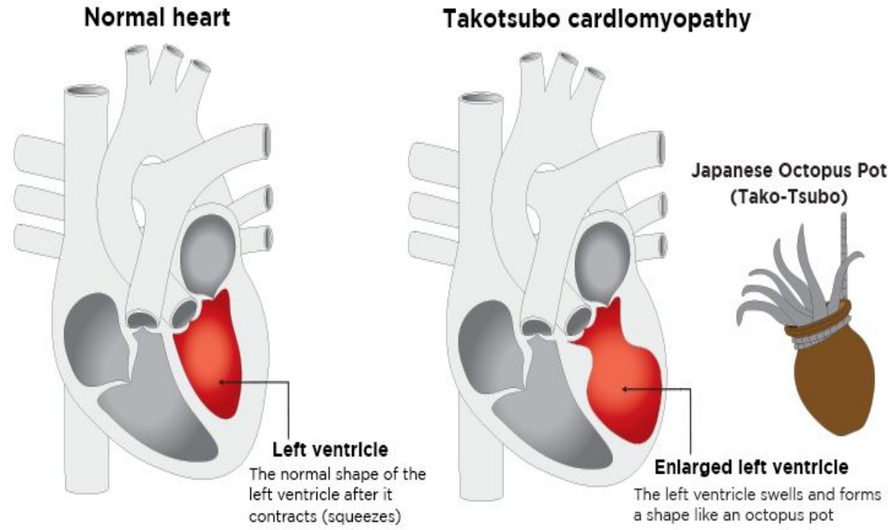
Causes	Common Symptoms/Signs	Diagnostic Testing	Treatment	Prognosis
• Major catecholamine discharge (also called Tako-Tsubo Syndrome) • Predominant in menopausal women • Pheochromocytoma, hyperthyroidism?	**Symptoms**: • Similar to any acute coronary syndrome • Acute chest pain (angina) and shortness of breath • Arrhythmias not uncommon; syncope rare • >60% of patients report a prior ***emotional or physically stressful event*** • These patients have more ***psychiatric & neuro issues*** **Signs** • Pericarditis & tamponade have also been described	• **ECG**: ***ST segment elevation*** & ***T wave inversion*** • Presents as acute anterior myocardial infarction, but *coronaries are normal at cardiac catheterization* • **CXR**: normal or pulmonary congestion • Imaging reveals apical left ventricular ballooning due to anteroapical stunning of the myocardium. • **Echo**: LV dyskinesia not consistent with any coronary distribution	• Similar to any acute MI • Most patients receive ASA, beta blockers, ACEI	• Most patients recover completely, although there are complications similar to those of MI and ACS

Hypertrophic Cardiomyopathy

Causes	Common Symptoms/Signs	Diagnostic Testing	Treatment	Prognosis
• Occurs when there is LVH without inciting volume or pressure overload. • The interventricular septum may be disproportionately involved • The amount of obstruction is preload and afterload dependent & varies day to day	**Symptoms** • Dyspnea, chest pain, syncope **Signs** • Arrhythmias are a common problem, especially **a-fibrillation** • Ventricular arrhythmias may occur leading to sudden death • Triple apical pulse, loud S4 & loud systolic murmur along left sternal border • Consequence of hypertrophy is **elevated LV diastolic pressures rather than systolic**	• **Echo**: Diagnostic when LV wall thickness greater than 1.5 cm; Septal wall motion tends to be reduced • **ECG**: A-fib, ventricular arrhythmias • **CXR**: LVH nearly universal in symptomatic patients but may be entirely normal in 25%; CXR often unimpressive	• Initially treat with Beta blockers to slow HR and improve diastolic filling • Calcium Channel Blockers also effective • Diuretics are frequently needed • Excision of part of the septum may help in severe cases • **AICDs (defibrillator**) useful when ventricular arrhythmias occur or syncope common	• Increased risk of sudden death • Natural history of hypertrophic cardiomyopathy extremely variable • Some people are asymptomatic for many years • Genetic testing may be beneficial • Sudden death may be the presenting symptom • This is the disease most often *found in athletes who die suddenly* • Endocarditis prophylaxis not necessary



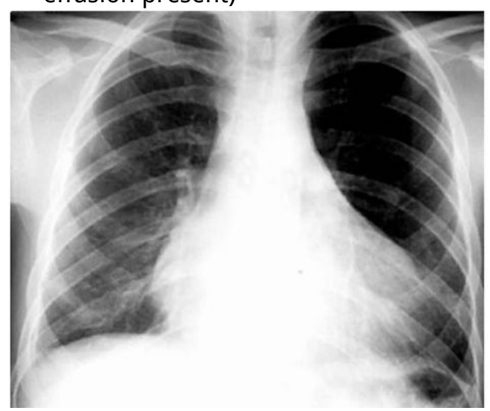


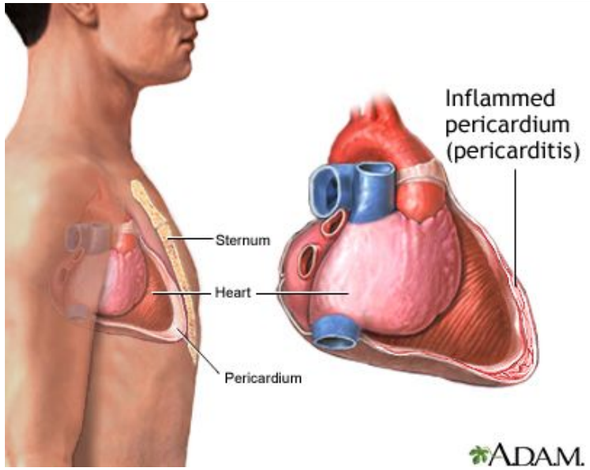


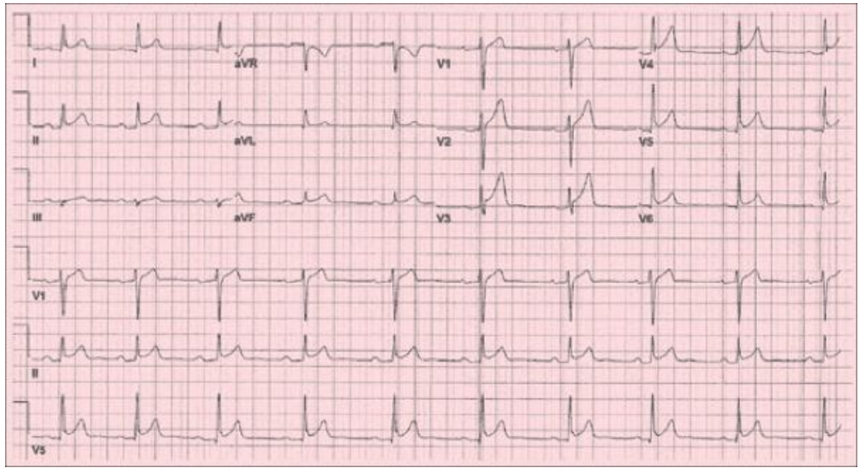
Restrictive Cardiomyopathy					
Causes	**Common Symptoms/Signs**	**Labs**	**Diagnostic Testing**	**Treatment**	**Prognosis**
• Rt heart failure > Lt heart failure • **Amyloidosis** is the most common cause but this condition is relatively uncommon. *NOTE*: Amyloidosis & Restrictive Cardiomyopathy are so strongly correlated that you should think about these in conjunction with one another.	• Pulmonary HTN • Angina, syncope, stroke, peripheral neuropathy • Periorbital purpura, thickened tongue & hepatomegaly suggestive of amyloidosis.	• BNP is usually elevated	• Low voltage EKG along with ventricular hypertrophy on echo are suggestive. • Myocardial biopsy/ cardiac MRI confirms extent amyloidosis	• Little specific treatment is available at present • Treatment of amyloidosis with chemo and stem cell transplantation may be of benefit • Beta Blockers slow heart rate & improve filling • Diuretics may be helpful • AVOID Digoxin as it may cause arrhythmias	• Men > Women; rare below age 40.
Sketchy: restrictive cardiomyopathy and constrictive pericarditis cause diastolic heart failure with normal chamber size and wall thickness					

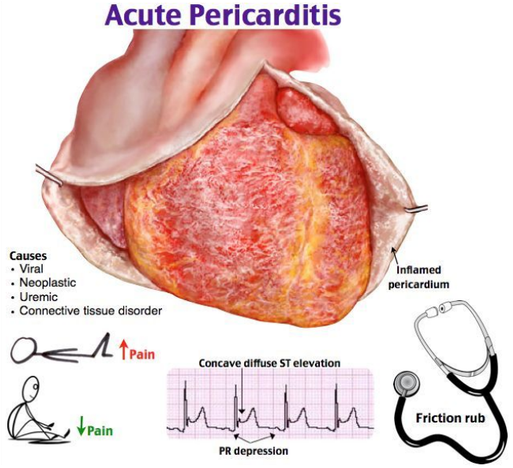
Acute Inflammatory Pericarditis

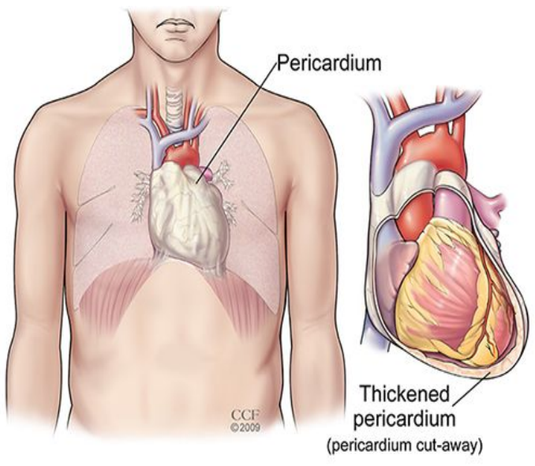
Diffuse ST-segment elevation with PR depression

Causes	Common Symptoms/Signs	Labs	Diagnostic Testing	Treatment
• Acute inflammation may be infectious or due to systemic diseases (autoimmune syndromes, uremia), neoplasm, radiation, drug toxicity, post cardiac surgery • In many cases the disease process involves the pericardium & myocardium • Postcardiotomy pericarditis (Dressler's Syndrome) may follow heart surgery ○ Dressler's syndrome is a type of pericarditis. Dressler's syndrome is believed to be an immune system response after damage to heart tissue or to the pericardium, from events such as a heart attack, surgery or traumatic injury.	**Symptoms** • Fever, dyspnea • Anterior pleuritic chest pain that is worse supine than upright **Signs** • Pericardial rub • Effusion may be present & may be large ○ Fluid build up is secondary to inflammation	• CRP & ESR (inflammatory markers) usually elevated How to distinguish Pericarditis from MI? Pericarditis has diffuse ST elevations, MI - only in leads showing ischemic areas of the heart	• **EKG**: ***Diffuse ST-segment elevation*** with PR depression. ○ Repolarization changes on ECG may mimic ischemic changes • **CXR**: Normal or enlarged heart (if effusion present)  *Pericardial Effusion*	• Decrease Activity • Provide Anti-Inflammatories (ASA, NSAIDs) with gastric protection ○ Systemic steroid for severe cases • Colchicine should be added in the acute attack to prevent recurrences

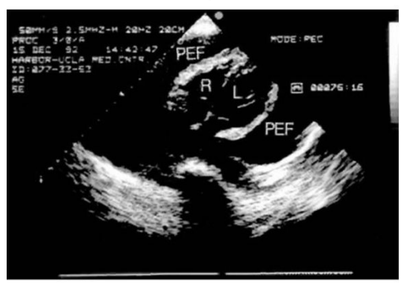
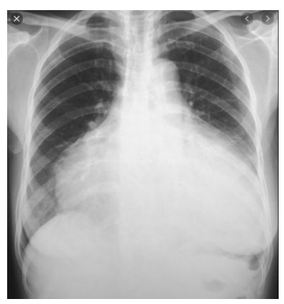








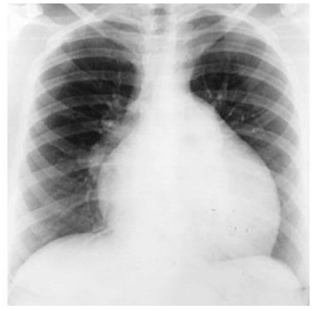
Pericardial Effusion & Tamponade
Beck's triad

Causes	Common Symptoms/Signs	Diagnostic Testing	Treatment	Prognosis
• Can occur during any acute pericarditis process and the speed of the fluid accumulation determines the physiologic importance. • Tamponade characterized by elevated intrapericardial pressure which restricts venous return and ventricular filling	**Pericardial Effusion**: • Depends on speed of accumulation **Tamponade**: • Tachycardia with an elevated JVP and either hypotension or a paradoxical pulse. Muffled heart sounds. Decreased ECG voltage ○ ***Pulsus paradoxus*** refers to an exaggerated fall in a patient's blood pressure during inspiration by greater than 10 mm Hg **Both**: • Pleuritic chest pain or no pain • Dyspnea & cough common; pericardial friction rub initially • Stroke volume and arterial pulse pressure fall; heart rate & venous pressure increase	• **CXR**: Enlarged cardiac silhouette with globular appearance • **ECG**: Non-specific ST-T changes; decreased voltage because the leads are further away from the heart due to the fluid. • **Cardiac CT & MRI** may show effusion • **Diagnostic pericardiocentesis** may be indicated • **Echo** often helpful  	• Small effusions can be closely followed • If tamponade present, urgent **pericardiocentesis** or cardiac surgery required • Repeat drainage of fluid may be needed	• Tamponade can be fatal

Constrictive Pericarditis
Kussmaul and A-fib

Causes	Common Symptoms/Signs	Diagnostic Testing	Treatment	Prognosis
• Inflammation can lead to a thickened, fibrotic, adherent pericardium • Restricts diastolic filling & causes elevated venous pressures • TB was once the most common cause, but now rare in US • Most commonly occurs after bacterial pericarditis • Other causes: post-cardiac surgery, RT, connective tissue diseases	• **Kussmaul Sign-** No fall or an elevation of the JVP with inspiration • Gradual progressive dyspnea, fatigue & weakness • **Right HF**: Edema, hepatic congestion, ascites usually present, elevated jugular venous pressure • **a-fib** is common	• **CXR**: Heart size either normal or enlarged; pericardial calcifications on lateral view (uncommon) • **Cardiac CT/MRI**: Only helpful occasionally; pericardial thickening • **Echo**: Reduced mitral inflow velocities with inspiration; thickened pericardium • **Cardiac Catheterization**: Usually confirmatory	• Aimed at specific etiology • NSAIDs may be beneficial • Diuresis is mainstay of treatment • ***Pericardiectomy*** may be needed if doesn't control symptoms	• Poor prognostic indicators for surgery: prior RT, renal disease, abnormal LV function, liver dysfunction, older age

Pulmonary Hypertension

Causes	Common Symptoms/Signs	Labs	Diagnostic Testing	Treatment
• Complex disorder with multiple causes • Clinically classified into 4 groups • The disease restricts blood flow through the lungs • Involves the smaller pulmonary arteries; the endothelium is dysfunctional & produces vasoconstrictor substances	• Mean pulmonary ***pressure of 25 mm Hg or more*** • Dyspnea and often cyanosis • Exertional dyspnea, chest pain, fatigue, lightheadedness • Later symptoms: Syncope, ascites, peripheral edema • ***Elevated JVP and RV heave***	• Laboratory evaluation includes search for a secondary cause (hypercoagulability, sleep apnea, chronic pulmonary emboli)	• **CXR**: Enlarged pulmonary arteries • All patients at high risk for Pulmonary Hypertension should undergo **right heart catheterization** • **ECG**: RVH, RA enlargement • **Echo** & **PFTs**  *Cardiomegaly, prominent right pulmonary artery & right ventricle*	• Continues to evolve & depends on etiology • ***Sildenafil*** (Vasodilator) may be helpful • Various medication combinations have been approved • Anticoagulation usually recommended • Lung transplantation may be indicated

Hypertension

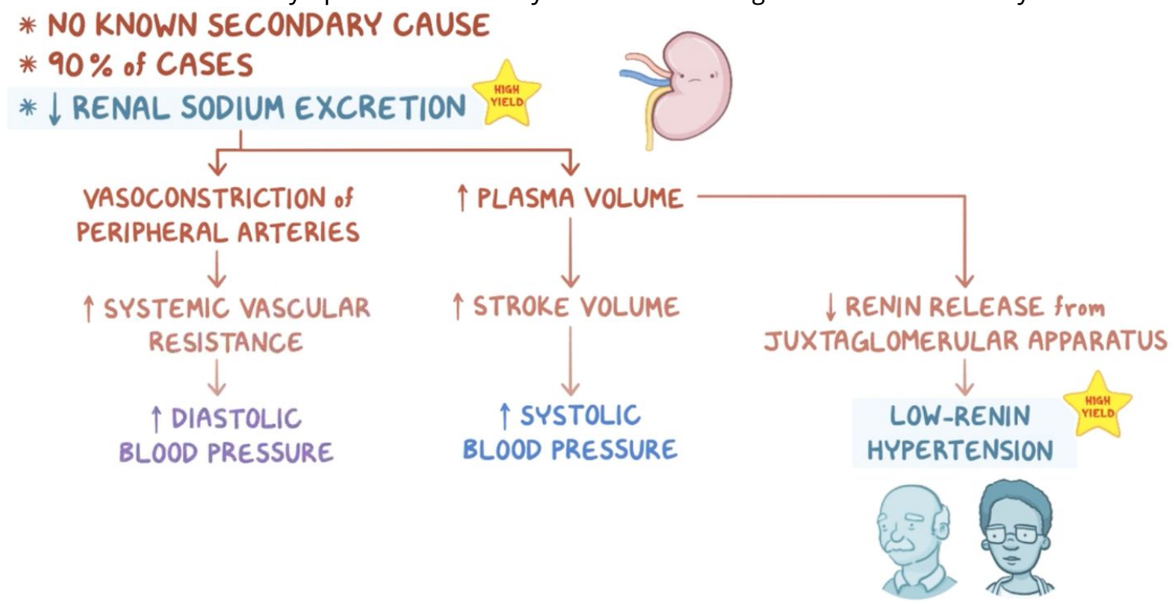
Primary Hypertension
HTN || Clinical Reasoning || Pregnancy

Fun Facts

- 95% of patients with HTN have "Essential Hypertension"
- Occurs in 10-15% of white adults and 20-30% of black adults
- Onset usually between ages of 25 and 50
- High levels of insulin promotes renal sodium retention

Causative Pathways

Overactivation of sympathetic nervous system and renin-angiotensin-aldosterone systems



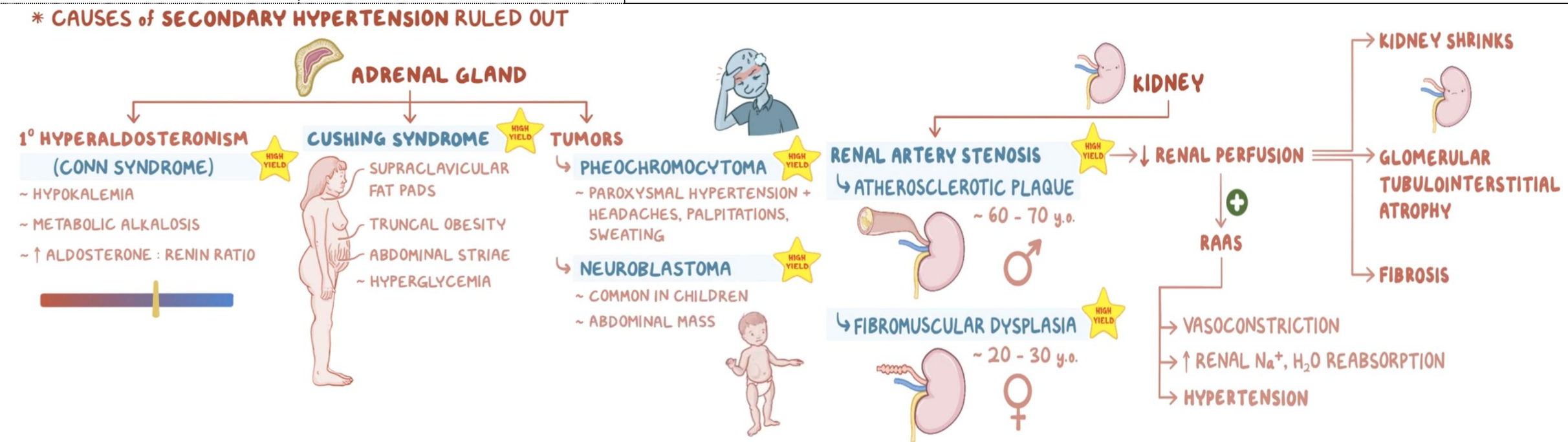
Sympathetic Nervous System Hyperactivity	Renin-Angiotensin System Activity
• Most apparent in younger people with HTN; they often have tachycardia & increased cardiac output • Correlations between BP and circulating catecholamines poor	• Renin secreted by cells surrounding glomerular arterioles in response to decreased renal perfusion pressure, decreased intravascular volume, circulating catecholamines, increased sympathetic activity • Renin acts on Angiotensinogen -> Angiotensin I -> Angiotensin II • Renin probably does not play a significant role in most essential HTN

Exacerbating Factors

- Obesity
- Sleep apnea
- Increased salt intake
- Excessive alcohol use
- Cigarette smoking
- Relationship of exercise to HTN variable
- Relationship between stress & HTN not established
- "Metabolic Syndrome" (upper body obesity, insulin resistance, hypertriglyceridemia) associated with HTN risk

Secondary Hypertension

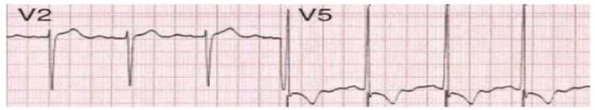
Fun Facts	Causes	Example: Hypertensive Cerebrovascular Disease & Dementia
• 5% of patients have HTN 2/2 identifiable cause • Suspect secondary HTN when it develops at an early age or after age 50 • Suspect secondary also when previously controlled HTN becomes refractory to treatment	• Genetic syndromes, • Kidney disease, (chronic caused by bilateral renal artery stenosis) • Renal vascular disease, • Cushing Syndrome, • Coarctation of aorta, • Pheochromocytoma, • Hyperthyroidism • HTN related to pregnancy • estrogen use and medications	• HTN major predisposing cause of ischemic & hemorrhagic stroke • Cerebrovascular events more closely associated with systolic BP • Incidence markedly reduced by antihypertensive therapy • HTN associated with higher incidence of vascular dementia and Alzheimer's Disease • Home BP measurements may be a better predictor of cognitive decline in older people

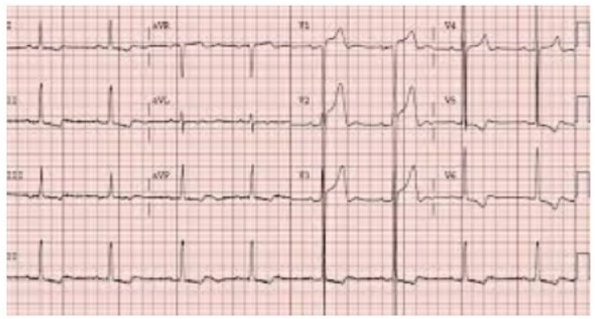


Untreated Hypertension

Complications

- Structural & functional changes in vasculature & heart
- Most adverse outcomes are due to thrombosis
- Excess morbidity/ mortality doubles for each 6mm Hg rise in diastolic pressure





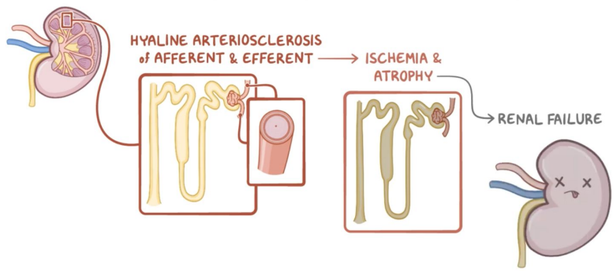
NOTE the ***tall QRS complexes that overlap with one another--*** this is due to high voltage from the hypertrophied ventricles

Prognosis

- Cardiac complications are major causes of morbidity/mortality in primary HTN
- LVH associated with increased cardiac risk

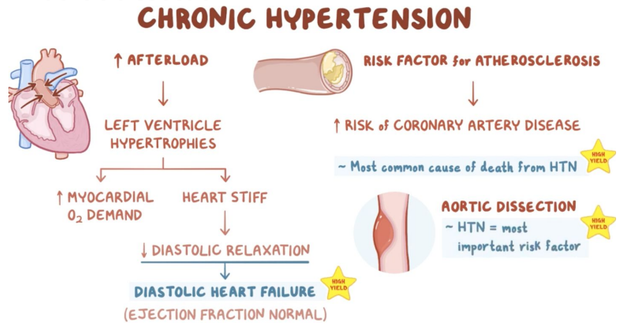
Hypertensive Kidney Disease

- Chronic HTN leads to vascular, glomerular and tubular conditions
- 25% of end-stage kidney disease caused by HTN



Aortic Dissection

- HTN is a contributing factor in the development of aortic dissection



Atherosclerotic Complications

- Most Americans with hypertension die of complications of atherosclerosis, but ***antihypertensive therapy seems to have a lesser impact on atherosclerotic complications***. Prevention of cardiovascular outcomes related to atherosclerosis probably requires control of multiple risk factors, of which hypertension is only one.

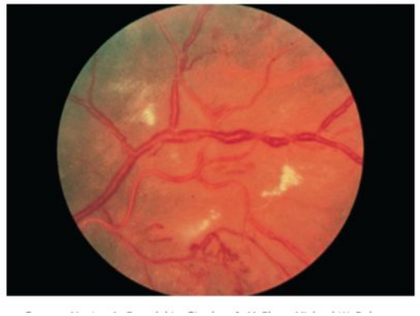
S/S

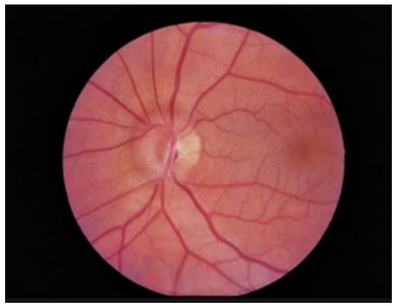
Symptoms

- Mild-Moderate HTN usually asymptomatic
- Most frequent symptom (headache) is very non-specific

Signs

- ***BP:*** Taken in both arms and/or legs
- ***Retinas***: Narrowing of arterial diameter, exudates, hemorrhages, papilledema associated with worse prognosis





- **Heart**: Left ventricular heave & aortic regurgitation murmur may be perceived
- **Pulses**: loss of peripheral pulses suggests ASHD and/or peripheral vascular disease

Labs/Diagnostic Testing

- **Recommended testing**: Hemoglobin, UA, creatinine, fasting blood sugar, plasma lipids, serum uric acid (relative contraindication to diuretic therapy if elevated because it can cause a gout attack), serum electrolytes
- **ECG**: ecg criteria are very specific but not very sensitive for LVH

Treatment (Start at @ 13:20)

Nonpharmacologic Therapy

- Lifestyle changes may affect morbidity/mortality. Overall, lifestyle modifications have only modest effects on BP.
- **Reduce**:
  - Weight, Alcohol use, smoking & salt intake
- **Increase**:
  - Gradual exercise in sedentary patients
  - Relaxation techniques and biofeedback

Pharmacologic Therapy

- Treatment should be offered to all patients with HTN regardless of their BP level: low risk of adverse effects
  - The BP level that should be treated has varied over time
  - Cardiovascular risk calculators are important (American College of Cardiology etc.)
- Treatment should be utilized at lower thresholds *if other cardiovascular risk factors are present or with* ***end-organ damage***

Diuretics

- Most extensively studied treatment group
- **MOA**
  - They decreased intravascular volume initially
  - They then decrease peripheral vascular resistance
  - Biochemical & metabolic effects are dose related
- **Positives**:
  - Diuretics are more potent in blacks, older people, smokers and obese patients
  - They mitigate bone loss in older women at risk for osteoporosis
  - Effective alone or in combination with other drugs
- **Negatives**:
  - May increase serum uric acid & precipitate gout attack

Beta Blockers

- **MOA**
  - Decrease heart rate, cardiac output & renin release
- **Positives**:
  - Beneficial in patients with angina, previous MI & stable heart failure
  - Useful in migraine & people with anxiety
- **Negatives**:
  - Side effects include bronchospasm, sinus node dysfunction, AV node depression, nasal congestion, Raynaud phenomenon, nightmares, depression
  - May cause fatigue, lethargy, erectile dysfunction

Renin Inhibitors

- **Positives**:
  - Most efficient treatment of elevated renin states
- **MOA:**
  - Aliskiren reduces levels of Angiotensin I and II
  - Aliskiren effectively lowers BP, reduces albuminuria, limits LVH but not yet a first line drug in HTN treatment

Angiotensin-Converting Enzyme Inhibitors

- Commonly used as initial medication in mild-moderate HTN
- Work best in combination with diuretics
- MOA:
  - Inhibits renin-angiotensin-aldosterone system
- **Positives**:
  - May be more effective in younger, white patients
  - Agents of choice in **diabetics**: they delay progression to end-stage kidney failure
- **Negatives**:
  - Less effective in black & older people

Angiotensin II Receptor Blocker (ARBs)

- **Positives**:
  - ARBs improve cardiovascular outcomes in HTN, CHF & diabetics with nephropathy
  - In diabetics, death & MI were reduced with ARBs
  - Unlike ACEI, ARBs rarely cause cough, rashes or angioedema:
  - ARBs (and ACEI) are less likely to cause depression than calcium channel blockers and beta blockers

Aldosterone Receptor Blocker

- **MOA**
  - Spironolactone is a natriuretic but only weakly in HTN; it has re-emerged in HTN treatment especially in resistant patients
- **Positives**:
  - Effective at lowering BP in all patients with HTN and are useful in blacks
- **Negatives**:
  - Can cause breast pain & gynecomastia by activity at the progesterone receptor

Calcium Channel Blockers

Alpha-Adrenoceptor Antagonists (Prazosin, Terazosin, and *Doxazosin*)

- **MOA**
  - These drugs cause peripheral vasodilation
- **Positives**:
  - Effective as single-drug therapy in 60% of people in all demographic groups
  - May be preferable in blacks and elderly patients
- **Negatives**:
  - Most common side effects: headache, peripheral edema, bradycardia, constipation

- **MOA**
  - Block postsynaptic alpha-receptors, relax smooth muscle, lower peripheral vascular resistance
- **Positives**:
  - Effective as single-drug therapy in some people
  - No adverse effects on serum lipid levels
- **Negatives**:
  - Side effects common (hypotension following 1st dose, palpitations, headache, nervousness)
  - Generally not used as initial therapy in HTN

AA = African Americans

	Affect RAAS?	Increase PVR?	Decrease PVR?	Better for AA?	First Line?	Notable Characteristics:
Diuretics			✔	✔✔✔	✔ for AA	May increase serum uric acid & precipitate gout attack
Beta Blockers	✔					Beneficial in patients with angina, previous MI & stable heart failure
Renin Inhibitors	✔					Most efficient treatment of elevated renin states
ACE Inhibitors	✔			Less effective; combine with diuretics	✔	Agents of choice in **diabetics**: they delay progression to end-stage kidney failure
ARBs	✔				✔	ARBs (and ACEI) are less likely to cause depression than calcium channel blockers and beta blockers
Aldosterone Receptor Blocker	✔			✔		It has re-emerged in HTN treatment especially in resistant patients
Ca2+ Blockers		✔		✔✔	✔	Effective as single-drug therapy in 60% of people in all demographic groups
Alpha-Adrenoceptor Antagonists			✔			Generally not used as initial therapy in HTN; Effective as single-drug therapy in some people

Developing an Antihypertensive Regimen

Regimen

- Antihypertensive therapy with diuretics & beta blockers have beneficial effect in majority of patients
- ACE inhibitors, and to a lesser extent ARBs, reduce adverse cardiovascular outcomes in other related populations (diabetics, heart failure, post MI)
- Clinical trials that have compared outcomes in relatively unselected patients have failed to show a difference between newer agents—such as ACE inhibitors, calcium channel blockers, and ARBs
- ACE inhibitors, ARBs, and calcium channel blockers useful as first line treatment
- *Thiazide diuretics best reserved for elderly and blacks* as first line therapy

Note about Beta Blockers

Beta blockers **not ideal** initial treatment

Drugs divided into 3 complementary groups: A, C, D

A: Interrupt renin-angiotensin system (ACE, ARB, Renin Inhibitors)
C: Calcium Channel Blockers
D: Thiazide Diuretics

Combinations of these groups are more potent in lowering BP

"**A**" drugs more effective in young, white patients	"**C**" & "**D**" drugs better in black & elderly patients	Most patients require **2 or more** drugs for BP control