Trials

- if a pt is on lasix chronically and is becoming refractory then it is likely due to hypertrophy of DCT and thus give HCTZ
- NEJM article noted the effectiveness of **dronedarone** (a new anti-arrhythmic similar to amio but without common amio toxicities) for the Tx of chronic Afib per ATHENA Trial
- growing evidence that CCB cause various skin issues within days to even months from starting
- new med like Plavix called **prasugrel (Effient)** which according to TRITON trial showed it to be more effective but higher risk of bleeding compared to Plavix, hence use only in young pts w/ no bleeding RFs
- Merck is trying to make **Lipitor OTC**

ROS

- **General:** diaphoretic, fatigue, exercise tolerance
- **Pulm:** SOB, DOE, PND, orthopnea, cough
- **CV:** angina/pleuritic like cp, palpitations
- **GI:** nausea
- **MS**: claudication
- **Neuro:** syncope, dizzy, lightheaded
- **Lymph:** swelling

PEx

- **Neck:** JVD, JVP 7-9cm, carotid bruits
    - JVP: turn head away exposing right side of neck, shine light b/t heads of SCM, this is where the IJ lies and medial to it is CA, adjust angle of bed if necessary so that meniscus is in middle of neck, after you identify the Internal Jugular Vein "milk" the blood up to empty the vein and watch blood from right atrium fill up the vein, identify highest point of pulsation, find sternal angle aka Angle of Louis, measure vertical distance and add 5cm (distance from angle to atrium). Important: the angle of the patient is inconsequential when calculating pressure you always add 5cm regardless if pt is at 30 degrees or 90 degrees!!! 30 degrees is chosen b/c the meniscus in a normal pt lies in the middle of the neck
- **CV:** rate/rhythm, nl S1/2, M/G/E, PMI, Heave
    - M: 1 (faint), 2 (quiet), 3 (loud), 4 (loud+thrill), 5 (loud+thrill+stethescope partly off chest), 6 (loud+thrill+stethescope entirely off chest) NB always note grade, time in cycle, location, radiation
        - Aortic: lean forward, radiates to carotids
        - Mitral: turn to L decubitus, radiates to axilla
    - G: (S4S1 S2S3) S1/S2 (diaphragm) vs S3/S4 (bell)
        - physiologic splitting during inspiration (A2P2) (b/c expansion of lung sucks blood into RA therefore increasing RV ejection time)
        - wide splitting during inspiration (A2 P2) w/ RBBB
        - paradoxic splitting aka split during expiration (P2A2) and no split during inspiration (S2) w/ LBBB
        - fixed splitting during inspiration and expiration (A2P2) w/ ASD and RHF
    - E: extra sounds like rubs, ejection sounds, clicks, snaps, splitting, et al
    - PMI: normal is mid-clavicular 5th intercostal space
    - Heave: feel heart beat along left sternal border reflecting LVH
- **Ab:** AAA size and bruits
- **Ext:** venous stasis changes, edema, +2/symmetric (carotid, radial, femoral, DP, PT) pulses, <2sec cap refill, warm to touch extremities

Resuscitation (BCLS aka CPR)

- Call EMS and Get AED
- A (Airway) head tilt, chin lift, jaw thrust
- B (Breathing) look for chest rise and listen for air thru nose and feel for air thru mouth w/ cheek, if agonal then give 2 rescue breaths and each over 1sec and if normal pulse then give breath Q5sec, if pt is fine then move pt to lateral position
- C (Circulation) check pulse (feel Brachial/Femoral/Carotid if >80/70/60SBP) if no pulse then begin compressions 30:2
- D (Defibrillation) clear, analyze, if shockable then charge, everyone's clear I'm clear, shock, immediately resume CPR, check pulse at 2min

Physiology

- MAP = COxSVR ("V = IR") = 1/3(S) + 2/3(D) = 65-100 mmHg and Pulse Pressure = S - D ~ SV via Baroreceptors/Chemoreceptors
- CO = HRxSV = (60-100)x(6-120) = 4-8 L/min and CI = CO/BSA = 2.6-4.2 L/min/m2 and EF = SV/EDV x100 > 50% (**~UOP, mental status, pH**) where SV is a fxn of...
    - Preload (=LVEDV~LVEDP~LAP~PCWP**~CXR~CVP**) diastolic fxn
        - Increase: venopressors (fluids, colloids) increase diastolic time (BB, CBB)
        - Decrease: venodilators (nitrates, diuretics) decrease diastolic time (inotropes)
        - NB preload is in turn a fxn of ventricular compliance and transmural pressure (difference in pressure b/t intracavitary and extracardiac)
        - NB Frank Starling Curve (X-preload vs Y-SV) CHF flattens the curve
    - Afterload (~MAP~SVR**~Cap Refill**) systolic fxn
        - Increase: vasopressors (refer)

        - Decrease: vasodilators (DRI/ACEI/ARB, CCB, Hydralazine, Minoxidil, BNP, alpha1 inhibitors, alpha2 stimulators)
    - Contractility (no direct measurement) systolic fxn
        - Increase: inotropes (refer)
        - Decrease: ?
    - Pressures/Waveforms
        - **C/JVP~RA** >6mmHg
            - a = atrial contraction
            - c = bulging of TV into RA
            - x = atrial relaxation
            - v = ventricular contraction
            - y = ventricular relaxation
        - **RV** 15-30/1-8mmHg
        - **Pulmonary Artery** 15-30/6-12mmHg
        - **Pulmonary Vein aka PCWP~LA** <12mmHg
        - **LV** ?mmHg
        - **Systemic Artery** 110-120/70-90mmHg

Shock	CVP	PCWP	SVR	CO	Tx
Hypovolemic	Dec	Dec	Inc	Dec	Colloids/Crystalloids
Cardiogenic	Inc	Inc	Inc (cool ext)	Dec (pulm S/S)	Inotropes IABP
Septic	Dec	Dec	Dec (warm ext)	Inc (tachy)	Vasoconstrictors
Neurogenic					
Anaphylactic				Dec (brady)	Epi Pen, Steroids, Benadryl

		Vasoconstrictive	Inotropic	Dosage
Vasoconstrictors	norepinephrine (Levophed)	+++ (alpha-1)	++ (beta-1)	16mcg/mL 0.1-3mcg/kg/min
	phenylephrine (Neosynephrine)	+++ (alpha-1)	+ (beta-1)	80mcg/mL 40-180mcg/min
	vasopressin (Pitressin)	++ (V2)	0	0.5U/mL 0.01-0.05 U/min • never use alone • only good if Levo present
Inotropes	dopamine (Intropin)	0 to +++ (dopamine)	+++ (beta-1)	1.6mg/mL 1-20mcg/kg/min • small doses (renal & mesenteric dilation) • med doses (inotropic) • large doses (vasoconstriction)
	dobutamine (Dobutrex)	Variable (?)	+++ (beta-1)	1mg/mL, 2-15mcg/kg/min
	milrinone (Primacore)	0	+++ (cAMP PDE Inhibitor)	200mcg/mL load 50mcg/kg over 10min then 0.375-0.75mcg/kg/min
	isoproterenol (Isuprel)	--- (beta-2)	+++ (beta-1)	0.1-10mcg/min • rarely used

Procedures

- **TTE**
    - Four Views: **parasternal long axis** (looks at LV, MV, AV), **parasternal short axis** (looks at a transverse section across LV), **4 chamber apical** (looks at all 4 chamber), **2 chamber apical** (looks at LV and RV) NB there are many other views like subcostal, etc
    - Look for wall motion abnl, valve dz, EF, RVSP nl <25 (calculated by plugging in TR velocity jet which almost everyone has into Bernoulli equation)
- **Left Heart Cath**
    - Coronary Angiography
    - EF or CO/CI
- **SC/IJ Central Line**
    - CVP
    - Central Venous O2Sat (ScvO2)
- **Right Heart Cath**
    - Same as a Central Line plus it measures RA/RV pressures

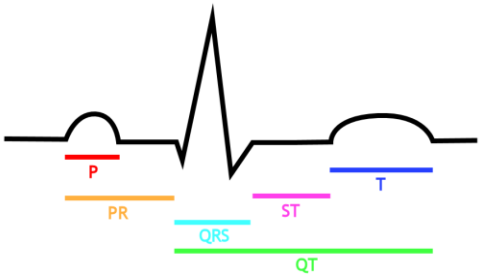
- **Swan Ganz Catheter**
  - Same as a Right Heart Cath plus it measures PCWP, CO, Mixed Venous O2Sat (SmvO2) ~70% (blood obtained from pulm artery and thus it is the mixture of systemic central venous blood AND coronary venous blood but in general systemic central venous blood is close enough)
  - Use: differentiate b/t cardiogenic vs distributive shock and then tailor therapy based on PCWP and SmvO2
  - Placement: place large sheath thru RIJ/LSC using a kit just like a central line kit, under fluoro inflate balloon tip of catheter which floats it down pulmonary artery wedging itself, a column of blood extends from catheter tip thru pulm circulation to LA, if there is no flow then PCWP~LAP~LVEDP~LVEDV~Preload, measurements are taken at end expiration, at 20cm you should hit RV, at 40cm you should hit the PA, at 60cm you should be wedged

Pre-Syncope/Syncope (sensation of fainting)

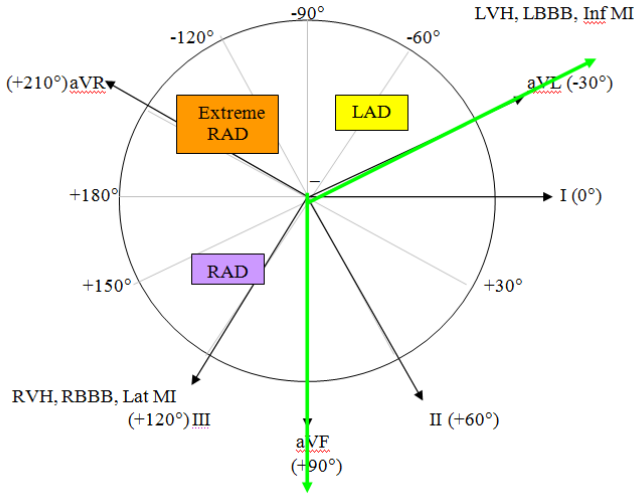
- Rule Out Vertigo (sensation of rotation/movement)
  - Peripheral Vestibular Dysfxn (sudden onset, severe Sx, few other neuro Sx)
    - BPV: repetitive brief (sec) episodes, 2/2 changes in position resulting in movement of otolith, Tx: Dix-Hallpike Maneuver to identify affected ear, Tx: Epley Maneuver to remove otolith from SCC
    - Labyrinthitis or Vestibular Neuronitis: single long (days) episode, 2/2 viral infection
    - Otitis Media (refer)
    - Meniere's (refer)
    - Ototoxic Drugs esp aminoglycosides, vancomycin, diuretics, NSAIDs, chemo, etc
  - Central (gradual onset, mild Sx, other neuro Sx)
    - CVA of the Posterior Circulation
    - Tumor esp Acoustic Neuroma
    - MS
    - Migraine
  - Tx: anticholinergics (scopolamine), antihistamines (meclizine, dimenhydramine), benzodiazepines (diazepam), neuroleptics (promethazine)
- Rule Out Disequilibrium (sensation of unsteadiness)
  - Seen in older pts w/ multiple problems including vision problems, arthritis, neuropathies, CVA deficits, dementia, etc
  - Seen in pts w/ cerebellar dz
- Etiology
  - **Unknown (30%)**
  - **Neurocardiogenic aka Vasovagal (25%)** cough/sneeze, deglutition of very cold liquids, defecation/micturition, after exercise, scare, sight of blood, pain, fatigue, prolonged still standing, warm environment, etc > increased sympathetic tone > vigorous contraction of LV > mechanoreceptors in LV trigger increased vagal tone aka hyperactive Bezold-Jarisch reflex > decrease BP
  - **Orthostatic Hypotension (15%)** hypovolemia (diuretics, bleed, adrenal insufficiency, etc) and/or vasodilation (BB/CCB/ACE-I, autonomic neuropathy from DM, EtOH, etc)
  - **Cardiovascular (10%)** carotid sinus syncope (pressure on carotid sinus from head turning, shaving, tight collar, etc) brady/tachyarrhythmia, valve dz, CAD, subclavian steal, etc
  - **Neurologic (10%)** TIA/CVA, seizure, migraine, etc
  - **Other (10%)** hypoglycemia, anemia, psychogenic, cataplexy, etc
- Dx: review in detail Hx/PMHx/Meds/FHx/PEx, orthostatics (supine after 5min then standing after 3min w/ + if >20 SBP or >10 DBP or Sx), carotid massage (monitor EKG/BP and + if pause >3sec, >50SBP, or Sx), Tilt Table Testing EKG/Tele/Holter/Event Recorder/EP eval, TTE, cardiac biomarkers, CT±A Neck/Head w/Contrast, EEG, prolactin level, UTox
- Tx: treat underlying problem, if vasovagal then try meds (fludricortisone, midodrine, disopyramide, anticholinergics) or drink water before inciting event, if orthostatic then use same meds, rise slowly, use Jobst compressive stockings
- Prognosis: 25% overall recurrence rate if not Dx and Tx
- Consider driving restrictions

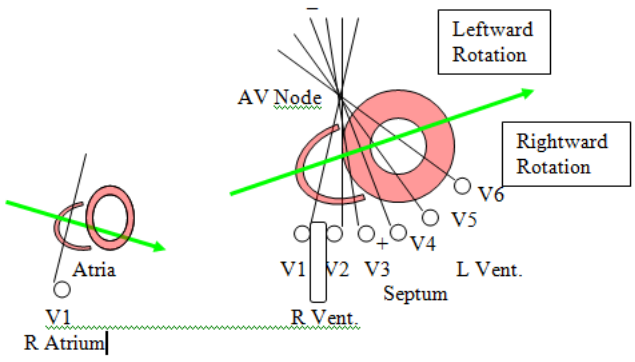
EKG

- General
  - when the direction of depolarization (due to movement of positive sodium ions in) is towards a positive electrode there is a positive deflection on the EKG for that lead
  - atrial AND ventricular depolarization moves from endocardium to epicardium while atrial repolarization moves from endocardium to epicardium and ventricular repolarization moves from epicardium to endocardium hence the T wave is a positive deflection
  - most EKGs are now10 Lead EKGs (precordials + RL + aVR, aVL, aVF) with last four leads creating a rectangle with aVR (right shoulder), aVL (left shoulder), RL (right leg), and aVF (left leg)
  - small box: 1mm/0.1mV x 1mm/40msec large box: 1mm/0.5mV x 1mm/200msec



I Normal	aVR Completely opposite w/ inverted P and QRS	V1 No P Small R Big S	V4 Small P Isoelectric
II Normal	aVL Normal but smaller	V2	V5
III Isoelectric	aVF Normal but smaller	V3 Small P Isoelectric	V6 Big P





- Ventricular Rate
  - 300,150,100,75,60,50, # of beats/6sec strip x10
  - SA 100-60, A 80-60, AV 60-40, V 40-20
  - Brady: Dz of SA Node (Sinus, SSS), Dz of AV Nodes (AV Blocks), Escape
  - Tachy: Narrow vs Wide Complex
- Rhythm
  - Sinus (upright P in I and aVF and negative in aVL suggesting that the direction of atrial depolarization is from SA to AV node, there is a P wave before each QRS, the P wave marches out w/ calipers, the P wave has the same morphology, negative in aVR)

- Atrial (+ P wave and narrow QRS)
- Junctional (no P wave but still narrow QRS)
- Ventricular (no P wave and QRS is wide)

- Axis
  - Nl (-30 to +90) (upright QRS in I and aVF but if negative in aVF then look at II and if + then b/t 0 and -30) "two thumbs up"
  - RAD (+90 to +180)
  - LAD (-30 to -120)
  - Extreme RAD (-120 to +180)
- Waves
  - P (atrial depolarization, V1 is perpendicular to the mean atrial vector resulting in a isoelectric biphasic P wave with equal up wave and down wave while II is parallel resulting in a monophasic P wave (<2.5mm wide and <3mm tall) that is the fusion of two waves)

	II	V1
LAE	Two Peaks Wide >3mm	More Negative Peak
RAE	Tall >2.5mm	More Positive Peak

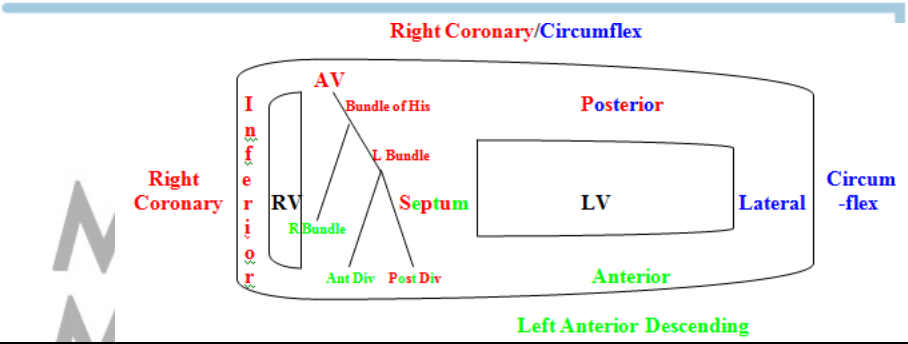
  - QRS (R = depolarization of L ventricle in V6→5→4 and R ventricle in V1→2→3 while S = depolarization of L ventricle in V1→2→3 and R ventricle in V4→5→6)
    i. check good R wave progression if not then "Regression" DDx: RVH, dextrocardia, L ventricle infarct, etc
    ii. check BBB in which the blocked branch is unable to conduct depolarization to its ventricle and is delayed but when it finally does it results in two out of phase QRS which is seen as wide with two R peaks (complete, incomplete, hemiblock)

	QRS	V1/2	V5/6	I,aVL
LBBB (L Heart Dz)	>120msec if < then "incomplete"	Deep S	RSR'	
RBBB (R Heart Dz)	>120msec if < then "incomplete"	RSR'	Deep S	Terminal Slurring of S
L Anterior Fascicular Block (LAFB) aka Hemiblock LAD + <120msec + qR in I/aVL and rS in II/III/aVF				
L Posterior Fascicular Block (LAFB) aka Hemiblock LAD + <120msec + rS in I/aVL and qR in II/III/aVF				

    iii. check VH
      1. Normal: LV is much bigger than RV and thus the R wave reflects the LV, since V6 is in the same direction as the mean ventricular vector it would be the most positive aka biggest R while V1 is roughly opposite the mean vector and thus it would be the most negative aka biggest S [V1 S>>>>R – V6 R>>>>S]
      2. LVH: "kissing waves", S in V1/2 + R in V5/6 >35, R in aVL + S in V3 >20(F)>28(F), R in aVL/I >11, along w/ LAD
      3. RVH: R>S or R>7 in V1/2, S>R or S>7 in V5/6, along w/ RAD
  - S (atrial repolarization)
  - T (ventricular repolarization)
- Intervals (interval = wave + segment)
  - PR (atrial contraction, beginning of P to **beginning** of R)
    i. 1 AV
    ii. 2 AV Mobitz Type I (Wackebach)_ __ ___ ____ X vs Mobitz Type II __ __ __ __ X
    iii. 3 AV
  - QTc (ventricular contraction, beginning of Q to **end** of T) corrected QT = QTc (Bazett's Formula) = QT/square root of RR (done b/c increase in HR causes decrease in QT), ~<1/2RR, 430msec (male) – 450msec (female), increased QT results in increased "R on T phenomenon" and b/c of increased time of repolarization there is increased chance of PVC and Torsades
    i. Ischemia (don't forget!!!)
    ii. Electrolyte: low K, Mg, Ca
    iii. Endocrine: hypothyroid
    iv. Drugs: psych (neuroleptics, SSRIs, TCAs), anti-biotics (quinolones, macrolides, bactrim), anti-arrhythmics, anti-fungals, toxins, etc
    v. Long QT Syndrome (channelopathy, ~15yo, SCD after unique events like swimming, abrupt arousal, etc)
    vi. NB Short QT (hypercalcemia, hyperTH, digitalis, increased sympathetic tone)
- Signs of CAD (remember continuous leads (V1 - V6, aVR - aVL - I - II - aVF - III) and reciprocal changes)
  - 1st: Is there evidence of a prior infarction?
    i. Q Waves

1. DDx of Q: Nl Qs (in aVR, III) vs Abnl Qs (in other leads and >1mm or >1/4 R or >1/3 QRS of that lead in >2 contiguous leads)

- 2nd: Is there evidence of active ischemia/infarction right now?
  - i. 1. Short-Lived/Hyperacute Peaked Ts
    1. DDx of Peaked T: hyperK, normal variant
  - ii. 2. Mild STE
    1. DDx of STE: Concave "Happy" STE (early repol which is seen in young pts and those w/ LVH, pericarditis, myocarditis, spasm) vs Abnl Convex "Sad" STE aka "Tomb-stoning" (>1-2/limb-precordial mm in >2 continuous leads w/ reciprocal changes)
  - iii. 3. Significant STE and Flattened Ts
  - iv. 4. Mild STE and TWI and Mild Qs
    1. DDx of TWI: abnl repol, post-tachy, post-pacing, intracranial bleed, strain)
  - v. Also check for LBBB
  - vi. When do you see STD? suggests subendocardial ischemia as seen in UA/NSTEMI
    1. DDx: digitalis, hypoK, abnl repol, strain
- 3rd: Where is the lesion located?
  - i. L Common = L Main gives off LAD, Ramus Intermedius, Cx.... LAD gives off a few left medium diagonal arteries and a few small septal perforators while the Cx gives off a medium Obtuse Marginals and large PDA
  - ii. R Common = R Main gives off Small Conus Artery (Conus Arteriosus), Right Diagonal Artery (RV/A), Right Marginal Artery (RV/A), SA Nodal Artery (SA Node), AV Nodal Artery (AV Node), Posterior Interventricular Artery aka PIVA, large PDA
  - iii. NB PDA 60% of time comes from R Main vs 40% comes from Cx
  - iv. NB think of it as two systems: LAD/PDA and Cx/PIVA



	Direct Changes (STE, Q)	Reciprocal Changes (STD, Big R)
Inferior LV or RV (R Main) NB suspect RV involvement (30% of the time) if pt has hypotension, signs of R-CHF, sinus/AV node changes, clear CXR etc therefore confirm w/ right precordial lead EKG, when this happens you can drive venous flow w/ fluids and pulm art dilators like NO/oxygen	II, III, aVF	I, aVL
Lateral LV (Cx)	I, aVL	II, III, aVF
Septal (Prox LAD & R Main)	V1-2	X
Anterior LV (Mid LAD)	V4-5	X
Apical/Apex LV (Dist LAD)	V5-6	X
Posterior LV (PDA)	X	V

- Other
  - Low Voltage aka <5 limb and <10 precordial (COPD, big breasts, effusions, infiltrative dz, myxedema, obesity, RCM/ICM, diffuse CAD, hypothyroidism)
  - U Wave (LVH, hypoK, anti-arrhythmics)
  - Brugada Syndrome (mutation of Na+ channel = decreased Na+ current = deadly arrhythmias while sleeping, 30yo Southeast Asian, EKG: ST elevation in V1-3, RBBB)
  - Hypothermia (Osborn Wave = small wave at J point)
  - Pulmonary Embolism (normal, sinus tachycardia, R heart strain signified by RBBB/RAD/RVH/RAE, wide S in I, large Q in III, inverted T in III/V, STD in II)
  - Pericarditis (PR depression in II, STE in V1-6, RR Elevation in aVR)
  - COPD (R hear strain like above + Low Voltage of Precordial Leads only, Prominent P Waves)
  - Digitalis (therapeutic: slopping downward ST depression) vs (Excess: conduction block esp AV block) vs (Toxicity: increased automaticity esp AT w/ 2:1 block, JT, PVCs)
  - Stroke (peaked/inverted Ts