Mechanism

- Decreased CO → Hypersympathetic State and RAAS Activation → Acute Compensation and Chronic Remodeling
- Markers of Poor Outcome: LVEF, hyponatremia, high BUN, hypokalemia (90% die from their CHF, 50% from progressive dz and 40% from sudden deathfrom VT/VF)
- CHF is not a disease but a manifestation of a disease hence you never say "pt has CHF" rather you always say "pt has CHF 2/2 MI" and when you describe CHF you characterize it in four ways
  - Low Output vs High Output
    - Low Output (refer below)
    - High Output
      - Decreased O2 Capacity (Anemia)
      - Increased Metabolic Demand (Pregnancy, HyperTH)
      - AV Fistulas (Fistula for HD: in arm, Paget's: in bone, Hemangiomatosis: in liver)
      - Vasodilation (Wet Beriberi, Shock)
  - Right vs Left
  - Systolic vs Diastolic
  - 2/2…
- Look at LVEDP
  - Low = High Output
  - High = Systolic/Diastolic then look at EF
    - Low = Systolic
    - High = Diastolic or Pericardial Dz

Causes of Systolic Dysfunction (EF<50%, increase in chamber size)	**Causes of Diastolic Dysfunction** (EF>50%, decrease in chamber size w/ hypertrophy, abnl MV inflow, tissue Doppler abnormalities, etc)
(1) Decreased Contractility Fxn of #/Mass of Myocytes • Ventricular MI (dead myocytes) • Ischemia (injured myocytes) • Restrictive Cardiomyopathies (injured myocytes) (2) Increased Afterload Laplace Eq = Afterload Stress = Pressure x Radius / 2 x Thickness • A/P Valve Stenosis (increased pressure) • Systemic/Pulmonary HTN (increased pressure) • A/P Valve Regurgitation (increased radius) • Dilated Cardiomyopathies (increased radius and decreased thickness) (3) Change in Preload Fxn of Volume of Blood • Low Venous Return (low preload) • Extreme Venous Return (very high preload) (4) Other • M/T Valve Regurgitation (less blood goes into systemic circulation) • VSD	(1) Abnormal Active Relaxation (occurs early diastole when Ca is pumped out of myocytes resulting in decreased cross-bridge pumping) • Ischemia (injured myocytes don't pump Ca out as well) (2) Abnormal Passive Filling (occurs middle diastole when mitral/tricuspid valves open 2/2 to change in pressure passively allowing blood flow into ventricles) • AS • HTN • Hypertrophic Cardiomyopathy (ventricles are more stiff) • Restrictive Cardiomyopathy (ventricles are more stiff) • Pericardial Dz • Systolic Dysfunction w/ Compensatory Hypertrophy (ventricles are more stiff) • T/M Stenosis (3) Abnormal Active Filling (occurs end diastole when atria contract to push the last but of blood into ventricles) • Atrial MI
Compensatory Mechanisms for Systolic Dysfunction	**Compensatory Mechanisms for Diastolic Dysfunction**
(1) Frank-Starling Mechanism (increased ESV and thus EDV leads to moderate increase in preload which leads to increase in # of actin-myosin cross-bridges which leads to increased contraction but at very high load the cross-bridges actually are less than normal) anyhow this compensation is acute with no true changes to the structure of the fibers but over time the following below occurs… (2) Hypertrophy (increase myocyte mass) (3) Adrenergic Stimulation (increase HR, vasoconstriction, etc)	None

- Stages of Progression of LV CHF
  - Stage I: = HR and =SV and thus =CO but ↑ filling pressure suggested by ↑ PCWP
  - Stage II: ↑HR and ↓SV and thus =CO but ↑↑ filling pressure suggested by ↑↑ PCWP
  - Stage III: ↑HR and ↓↓SV and thus ↓CO with ↑↑↑ filling pressure suggested by ↑↑↑ PCWP

- NB therefore =CO does NOT mean nl fxn therefore one must look at PCWP

- Reasons for Decompensation
  - Progressive of Initial Cause or Additional Cause for HF (refer above but esp Arrhythmias, HTN, MI, PO, new valve dz, excessive BB/CCB)
  - Noncompliance w/ Diet/Meds or Inappropriate Reduction of Therapy
  - Increased Venous Return
    - Increased Sympathetic Tone (parasympathetic neuropathy, caffeine, exercise, illicit drugs, infection, nicotine, pain/stress, adrenergic drugs)
    - Increased Extracellular Volume (pregnancy, RF, increased sodium intake, high fluid intake, renal failure)

S/S

- regardless of the etiology (systolic vs diastolic) S/Sx are based on left vs. right

	S/Sx of Left Sided HF	**S/Sx of Right Sided HF**
Heart Dysfunction	• HypoTN - b/c decreased CO • Tachy - compensatory mechanism • Conduction Disturbances - esp LBBB which delays impulse signaling contraction from getting to LV which causes further reduction in CO • Increase P of S2 - b/c of pulmonary congestion • Ventricular S3 Gallop "Kentucky" - reflects atrial blood hitting a pool of blood in ventricle blood during early diastole b/c of prior low EF aka systolic dysfunction - can be normally seen in young ("S3") and 3rd ("S3") trimester pregnant women - differentiate from fixed split S2, MS w/ opening snap, MVP w/ mid-systolic click • Atrial S4 Gallop "Tennessee" - reflects atrial blood unable to be forced into ventricle during late diastole b/c ventricle is too filled up with blood b/c of prior low EF aka systolic dysfunction OR the ventricle itself is not compliant enough to take on more blood aka diastolic dysfunction - can be normally seen in elderly ("S4") who do not have HF but just have a heart w/ decreased compliance and also seen in athletes • PMI shifted left and down - reflects CM 2/2 compensatory hypertrophy • B-type/Brain Natriuretic Peptide (BNP) 1000s pg/mcl - b/c LV has much more mass than RV - A,B,C = produced by atria, ventricle, vascular endothelium in response to increased wall stress (called Brain-B b/c in pigs it is found in brain but in humans it is found in heart) - NP actually suppresses RAS, decreases renal NaCl and water retention, and vasodilates but it does so only to a minor degree such that it cannot reverse the other stimulatory agents on these systems therefore its only clinical use is to help in diagnosis, monitor, quantify severity, and assess effectiveness of treatment - False +: RF, ACS - Can be used to predict r/o rehospitalization when measured b/f discharge	• S/Sx of Left Sided Heart + • RV Heave - reflects CM 2/2 compensatory hypertrophy • BNP 100s (if just true RHF) - b/c LV has much more mass than RV
Pulmonary / Body Congestion	*Cephalization of Pulmonary Veins* (vessels > bronchi in upper lobes) ↓ *Interstitial Ground-glass Markings (w/ air bronchogram, flattened diaphragm, Kerley B Lines aka enlarged lymphatics) aka Interstitial Edema (fluid goes from veins to interstium), peribronchial cuffing (fluid around bronchi when seen on end)* • tachypnea • dyspnea • paroxysmal nocturnal dyspnea - sudden intermittent dyspnea that awakens the pt at night forcing the pt sit upright in bed for 10-15min - much more common in CHF and NOT in primary pulm dz hence asked • orthopnea / nocturnal cough - constant persistent dyspnea that forces pt to sleep upright the entire night - much more common in CHF and NOT in primary pulm dz hence asked • accessory muscle use - SCM for inspiration and ab muscles for expiration	*Mild Congestion* • JVD • Kussmaul's Sign (JVD fails to decrease during inspiration but actually increases) • Hepatojugular Reflex (liver pressure raises JVP as blood has difficulty going into R heart) • Peripheral Pitting Pedal/Sacral Edema • R sided Pleural Effusion • Weight Gain • Liver Damage w/↑LFTs ↓

	• speech interrupted by inspiration (telegraphic speech) • pursed lip breathing ↓ *Alveolar Markings from Edema (fluid goes from interstitium into alveoli) often dependent/central sparing outer 1/3 aka "bat wing" appearance* • same + • crackles • couth with blood tinged or pink frothy sputum 2/2 alveolar hemorrhage • increased fremitus • dullness ↓ *Pulmonary Effusion (fluid goes from alveoli to parietal space)* • same but • decreased fremitus ↓ *Pulmonary Artery Congestion* • same + • RV heave • tricuspid regurgitation • increased P of S2 • RHF	*Moderate Congestion* • Anasarca • Ascites • Liver Damage w/ hepatomegaly and↑↑LFTs • RUQ tenderness if congestion is rapid enough ↓ *Severe Congestion* • Cardiac Cirrhosis w/ nl LFTs • TR
Body Hypoperfusion	*CNS* • AMS • memory problems • Cheyne-Stokes breathing • anorexia *Renal* • decreased EAV → decreased blood flow in afferent and decreased Na delivery → sympathetic stimulation and renin secretion • decreased BP → carotid/aortic arch baroreceptors stimulation → ADH release → increased distal water reabsorption • nocturia - during day decreased CO results in decreased renal perfusion and thus RAS activation but during the night b/c skeletal muscles are not being used at all enough blood is shifted to kidney that diuresis occurs *Muscles* • weakness/fatigue *Skin* • pale/cool	None

	Tx of S/Sx of Left Sided HF
Pulmonary Body Congestion	**"LMNOP"** (1) **L**oop Diuretics • diuretic resistance occurs and if this occurs then change route (PO to IV), change Hz (single to multiple), use more powerful diuretics • remember that in the very acute severe setting diuretics are actually harmful • no mortality benefit just symptom relief • decreases preload • immediate effect which is not 2/2 diuretic effect but rather lasix increased PG secretion from kidney which vasodilates vessels in lung (2) **M**orphine • decrease Sx of dyspnea/anxiety • venodilator thereby decreasing preload **M**onitoring • if severe exact knowledge of intracardiac filling pressures and volumes thru Swan-Catheter is helpful • <2g/d salt intake • limit fluid intake (1.5L/d) • BNP • volume status w/ I/Os, daily weight, etc (3) **N**itrovasodilators • venodilator thereby decreasing preload (4) **O**xygen • BiPAP to intubation w/ MV (5) **P**osition

	• sit up w/ feet dangling over side of bed
Body Pulmonary Hypoperfusion	Increase Contractility if Hypotensive (1) Inoptropes (2) Ventricular Assist Devices (LVAD) Decrease Afterload if Hypertensive (1) Vasodilators (2) Intra Aortic Balloon Pump (IABP) Decrease Preload (1) Venodilators Other: nesiritide (Primacor) • used for short term treatment in acute HF as has mild but good hemodynamic effects but long term M/M has not been established • Endogenous: A,B,C = produced by atria, ventricle, vascular endothelium • VERY MILD renin inhibition • VERY MILD vasodilation • VERY MILD inhibition of proximal Na reabsorption

Systolic Dysfunction Treatment Scheme

- Prolong Survival: ACEI/ARB, H/N, BB, Aldactone/Eplerenone
- Do Not Prolong Survival but Tx Sx: Loop Diuretics, Digoxin, Antiarrhythmics, Inotropes

ACC/AHA Stage	NYHA Class	Treatment
A + High Risk NO Structural Dz NO Symptoms	NYHA I Asymptomatic During Exertion	Treat RFs ACE-I/ARB • V-HeFT II: ACE-I vs hydralazine/nitrates resulted in better ↓M/M • SAVE: ↓M/M in NYHA I pts, SOLVD-T: ↓M/M in NYHA II/III pts, CONSENSUS: ↓M/M in NYHA IV pts, ATLAS: high dose >30mg better than low dose <5mg • HOPE: ramepril is the best ACE-I • ELITE, OPTIMAL, VALIANT, CHARM: ARB same as ACE-I in ↓M/M and can actually increase benefit of ACE-I when added on, consider in pt who cannot tolerate ACE-I or as an add on if you want more ↓M/M
B + High Risk + Structural Dz NO Symptoms		Same + BB • US Carvedilol, MERIT, CIBIS-II, COPERNICUS, CAPRICORN: ↓M/M in NYHA II/III/IV • COMET: carvedilol better than extended release metoprolol b/c of alpha2 blocking and antioxidant effects • Bisoprolol • Avoid in NYHA IV AICD • to help prevent risk of VT/Vfib and thus sudden death which is the most common cause of death in CHF • Indication: (refer to EKG notes) • NB if pt cannot get an AICD then start amio for the time being
C + High Risk + Structural Dz + Symptoms	NYHA II Symptomatic During Exertion NYHA III Symptomatic During Normal Daily Activities	Same + Chronic Symptomatic Tx w/ "LMNOP" Salt Restriction Aldactone/Eplerenone • RALES: ↓M/M in NYHA III/IV pts and EF<35% • If Cr<2.5 Digoxin • Controversial, consider if EF <35% Hydralazine+Nitrates • V-HeFT I: placebo vs hydralizine/nitrates resulted in ↓M/M but V-HeFT II showed that ACE-I is superior to H/N • A-HeFT (African American Heart Failure Trial): this is Dr. Yancy's work on pharmacogenomics in treating AA with HF, increased NO and decreased nitric oxidants in white and vice versa in AA, there apparently is a different polymorphism is NOS explaining this difference, long acting nitrates and hydralazine (combo called BiDil) are good in AA HF pts, Nebivolol (beta-blocker that has other properties that increase NO) • Consider in pt who cannot tolerate ACE-I/ARB or in AA with NYHA III/IV AC • if LV thrombus, large akinetic LV segment or EF<30% BiVentricular PPM

		• helps resynchronize the heart so that it pumps as effectively as possible • Indication: symptomatic despite medical Tx + LBBB + LVEF <35% + LVED >55mm
D + High Risk + Structural Dz + Symptoms Refractory to Meds	NYHA IV Symptomatic During Rest	Same + Chronic Inotropes LVADs Transplant (50% survival at 10yrs) Hospice

Diastolic Dysfunction Treatment Scheme

- eliminate the underlying etiology
- lengthen diastole as much as possible by slowing the heart through BB
- similar symptomatic Tx as above but no inotropes b/c there is no systolic dysfunction
- milrinone and nitroglycerine have lusitropic effects (promote ventricular relaxation)