ACUTE DECOMPENSATED HEART FAILURE

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Acute Decompensated HF

- Defined as sudden worsening of HF symptoms
- Generally requires hospitalization for management
- BNP can help discern if dyspnea is cardiogenic
- Terminology used to describe pts:
  - Wet: fluid up, needs diuresis
  - Dry: euvolemic
  - Warm: well perfused
  - Cold: poor perfusion

Acute Decompensated HF
Common Causes

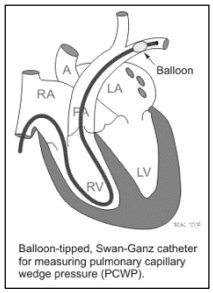
- ACS
- AFib/arrhythmias
- Concurrent infections
- Endocrine abnormalities (thyroid, DM)
- Pulmonary embolus (PE)
- Other acute CV disorder (valve disease, endocarditis, etc.)
- Uncontrolled HTN

Acute Decompensated HF
Common Causes

- Meds
  - Non-compliance
  - Recent addition of negative inotropic agent
  - Na-retaining meds (NSAIDS, TZDs, steroids..)
- Na/H_2O retention
- Excessive alcohol/drug use

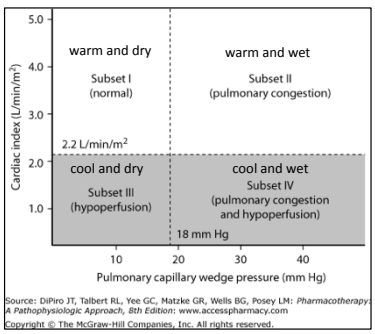
Pulmonary Artery Catheter

•Invasive hemodynamic monitoring helps evaluate volume and perfusion status, but is not required
•Pulmonary Artery Catheter (PAC) placement
•PCWP- measures fluid status, cut-off is 18 mmHg
•CI- measures CO, cut-off is 2.2 L/ min/m^2



Balloon-tipped, Swan-Ganz catheter for measuring pulmonary capillary wedge pressure (PCWP).

Acute HF Subsets



Acute HF Subsets

- Subset I (warm and dry)
  - CI > 2.2, PCWP < 18
  - Well perfused without congestion
  - Likely well compensated and only needs fine-tuning oral meds and monitoring
- Subset II (warm and wet)
  - CI > 2.2, PCWP > 18
  - Well perfused with congestion
  - Needs IV diuretics and vasodilators

Acute HF Subsets

- Subset III (cool and dry)
  - CI < 2.2, PCWP < 18
  - Inadequate perfusion without congestion
  - High mortality
  - Treatment focuses on increasing CO with a combination of positive inotropes, *very cautious* fluid replacement, and vasodilators
  - Therapy will need to be tailored to each pt

Acute HF Subsets

- Subset IV (cool and wet)
  - CI < 2.2, PCWP > 18
  - Inadequate perfusion with congestion
  - Most complicated presentation for AHF with worst prognosis
  - Therapy is highly individualized to diurese and improve CO while maintaining adequate MAP
  - Agents used include inotropes, vasodilators, and diuretics

Acute Decompensated HF
Goals of Therapy

- Correct underlying factors and prevent further episodes
- Relieve symptoms
- Improve hemodynamics
- Optimize chronic oral medications before D/C
- Educate pt to reinforce importance of compliance with meds and lifestyle changes

Acute Decompensated HF
Medications: Diuretics

- Loops are first line diuretics in acute HF
  - Furosemide, torsemide, bumetanide
- Reduce preload and PCWP, no effect on CO
- Usually IV boluses or continuous infusion
- Titrate to U/O, PCWP, congestion, BP
- For maximal effect metolazone may be added
- See previous notes for details on diuretics

Acute Decompensated HF
Medications: Vasodilators

- Nitroglycerin (NTG), niotroprusside, nesiritide
- Help relieve s/s of congestion
- Have not been shown to have any benefit on outcomes
- Reduce preload and afterload, reduce PCWP, reduce SVR, increase CO

Acute Decompensated HF
Medications: Vasodilators

- Nitroglycerin
  - Source of NO
  - Given as continuous IV infusion
  - Venodilation at low doses (reduces preload), veno- and arteriodilation at higher doses (reduces preload and afterload)
  - Typically used at low doses
  - Causes coronary dilation, ideal in CAD and myocardial ischemia
  - Tachyphylaxis develops within 24h

Acute Decompensated HF
Medications: Vasodilators

- Sodium Nitroprusside
  - Source of NO
  - Given as continuous IV infusion
  - Venodilation AND arteriodilation at any dose
  - Reduces both preload, afterload, PCWP, SVR, BP, increases CO and HR
  - More potent in lowering BP than NTG
  - Can worsen myocardial ischemia
  - Cyanide and/or thiocyanate toxicity with hepatic or renal insufficiency

Acute Decompensated HF
Medications: Vasodilators

- Nesiritide
  - Recombinant Human BNP
  - Causes venous and arterial dilation and natriuresis
  - Reduces PCWP, preload, afterload, SVR, BP, increases CO, no effect on HR
  - No tachyphylaxis
  - Longer half life than NTG or nitroprusside
  - Place in therapy is not well defined but effective and has unique mechanism

Acute Decompensated HF
Medications: Inotropic Agents

- Dobutamine, milrinone, dopamine
- All increase intracellular cAMP levels which leads to increased contractility
- Used to help perfuse vital organs
- They increase cardiac workload/ischemia
- ALL inotropes are associated with increased risk for arrhythmias. Should be used as last resort in select populations

Acute Decompensated HF
Medications: Inotropic Agents

- Dobutamine
  - Inotrope of choice for AHF
  - Primarily β_1 agonist with minor β_2 and α_1
  - Improves contractility and CO
  - Increased CO causes a reflex decrease in SVR
  - Reduces PCWP- useful with congestion
  - Can cause tachycardia, little effect on BP
  - Causes increase in oxygen consumption
  - Avoid use if pt already on β-blocker

Acute Decompensated HF
Medications: Inotropic Agents

- Milrinone
  - Phosphodiesterase Inhibitors (PDEI)
  - Works through a different mechanism than β-receptors
  - Positive inotrope, vasodilator for mostly veins and pulmonary vessels
  - Causes increase in SV, CO, reduction in PCWP, with minimal changes to HR and BP
  - Useful with congestion
  - Ideal for use in β-blocked pts

Acute Decompensated HF
Medications: Inotropic Agents

- Dopamine
  - β_1, β_2, α_1, D_1 agonist
  - At lower doses (3-10 mcg/kg/min) it activates β_1, β_2, D_1 receptors, increasing inotropy, SV, HR, CO
  - At higher doses (> 10 micg/kg/min) activates α_1 and increases chronotropy and arrhythmias
  - Increases BP, CO, PCWP, coronary ischemia
  - Highly proarrhythmic, should be reserved for pts with HoTN and near cardiogenic shock

Acute Decompensated HF
General Monitoring Parameters

- Fluids in and out
- Vital signs
- Daily body wt.
- Daily serum electrolytes, BUN, Cr
- Clinical s/s perfusion and congestion