DEFINITIONS:

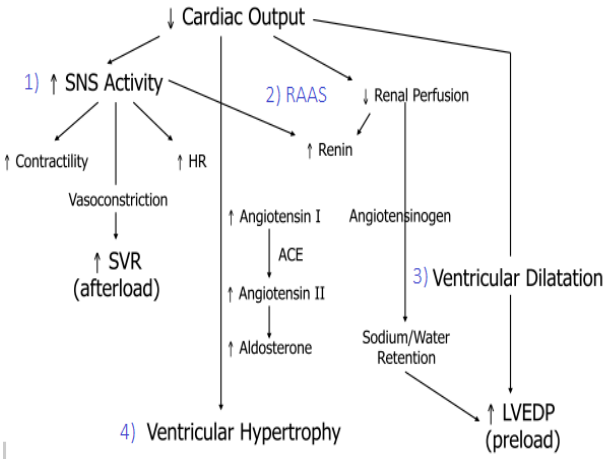
- Acute heart failure: onset of S/S of HF in a pt with no prior hx of HF
- Acute decompensated heart failure (ADHF): represents a new or worsening S/S of dyspnea, fatigue, or edema that results in hospitalization or unscheduled medical care that are consistent with an underlying worsening of LV function

CARDIAC OUTPUT: CO = HR x SV

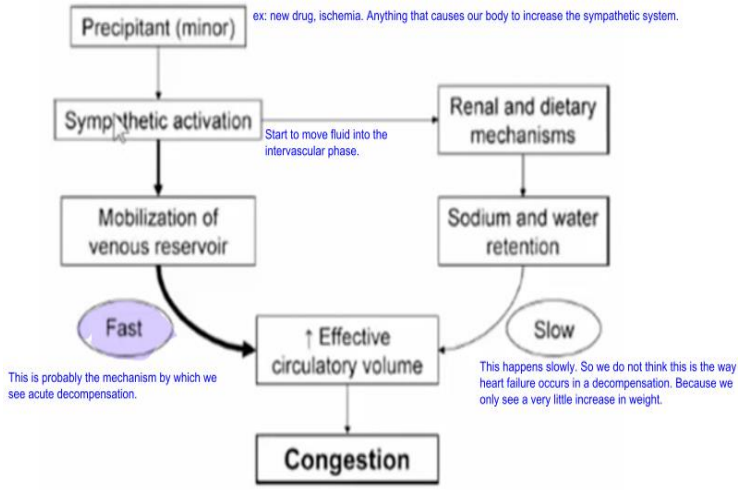
Stroke volume is affected by:

- Preload (venous return)
- Contractility
- Afterload (resistance to flow)

COMPENSATORY MECHANISMS:



MECHANISM OF CONGESTION:



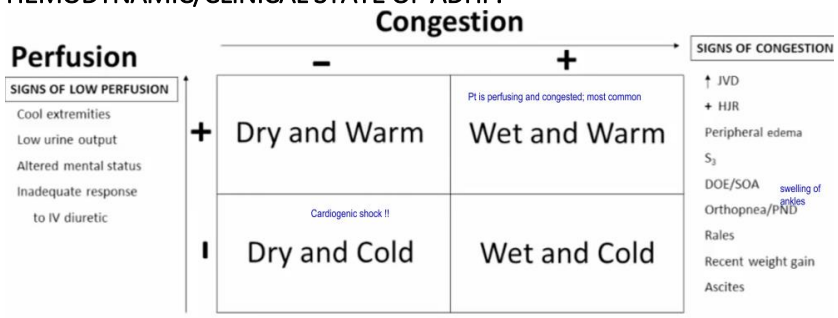
ETIOLOGY OF ADHF:

Primary Cardiac	• Progressive cardiomyopathy • Acute cardiomyopathy (myocarditis) • Myocardial ischemia • Arrhythmia • Valvular dysfunction • Pericardial syndrome
Pressure Overload	• Hypertensive urgency/emergency
Volume Overload	• Sodium and volume overload • Poor compliance with diuretics • Renal or hepatic dysfunction
High Output	• Shunt • Anemia • Septicemia • Thyroid disease
Other	• Inflammation of infection • Medication non-adherence • Major surgery • New medications • Substance abuse

CLINICAL PRESENTATION OF ADHF:

- Dyspnea on exertion
- Paroxysmal nocturnal dyspnea
- Orthopnea
- Fatigue
- Weakness
- Exercise intolerance
- Edema
- Cough
- Nocturia
- Weight gain
- Abdominal distention
- Cognitive impairment
- Nausea
- Abdominal discomfort
- Oliguria
- Anorexia
- Cyanosis

HEMODYNAMIC/CLINICAL STATE OF ADHF:



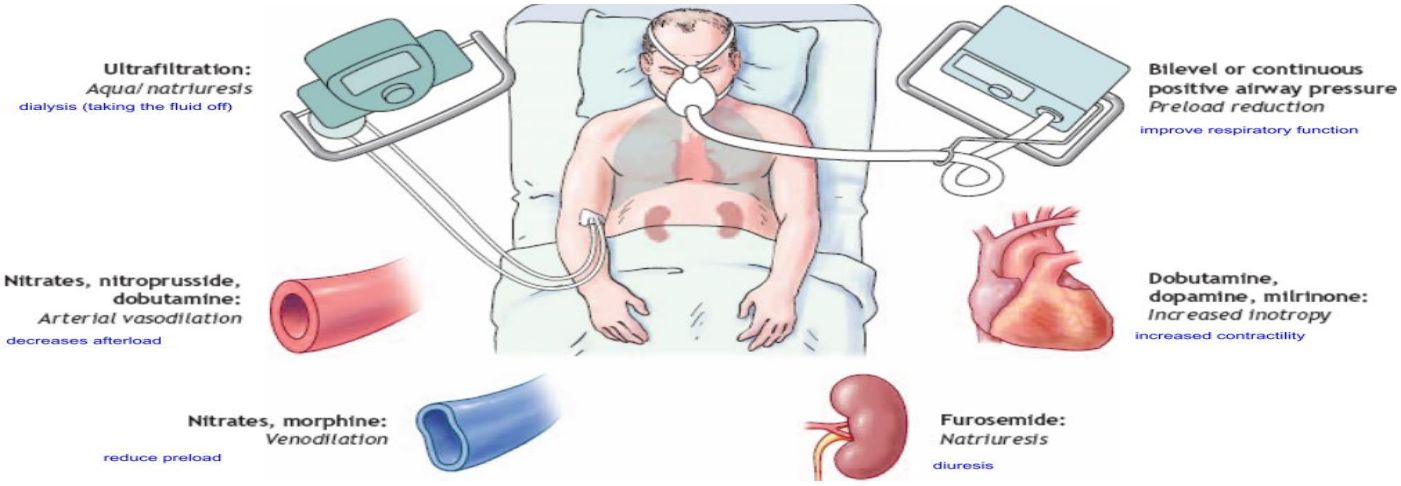
DRUG-INDUCED HEART FAILURE:

Beta-blocker	Negative inotrope (initially)
CCB (verapamil, diltiazem)	Negative inotrope
Anti-arrythmics (propafenone, flecainide, sotalol)	
NSAIDs	Fluid retention
Thiazolidinediones (glitazones)	
Doxorubicin	Direct Toxic EFfect

GOALS OF THERAPY:

- Improve symptoms
- Optimize volume status
- Identify etiology
- Identify precipitating factors
- Optimize chronic oral therapy
- Minimize AEs of therapy
- Identify patients that will benefit from revascularization
- Education
- Consider disease management program

TREATMENT OPTIONS:



DIURETICS:

USE IN HF:

- Elevated filling pressure is ultimately responsible for most of the S/S of ADHF
- A loop diuretic (furosemide) is mainstay of therapy due to ability to produce significantly more natriuresis than other classes of diuretics
- Loop diuretics will increase urine output by excretion of sodium and water → reducing plasma volume, total body/water sodium → reduce RV and LV filling pressures, peripheral and pulmonary edema

DOSE:

- High dose (vs low): better diuresis (reduced congestion) but no mortality or hospitalization benefit
- Bolus IV dose (vs. continuous IV): no difference but more practical/faster

MONITORING:

Signs	Edema, ascites, rales, hepatomegaly, JVP, HJR, liver tenderness	2-3x daily
Sx	Orthopnea, PND, nocturnal cough, dyspnea, fatigue	2-3x daily
Vitals	HR, BP, O_2 sat	2-3x daily
Weight	After voiding in AM	Daily
Urine output	> 500 mL in first 2h (> 250 mL if SCr > 220 umol/L)	2-3 x daily
Lab	Na, K, Mg, BUN/SCr	Daily

DIURETIC RESISTANCE: failure to reduce volume despite liberal diuretic dose

Why?	Options
• Inadequate furosemide dose • Reduced GI absorption • Compensatory sodium Na retention	• Increase furosemide dose • Addition of a 2nd diuretic (metolazone) • Addition of a vasodilator (nitroglycerin) • Ultrafiltration

VASODILATORS:

- Vasodilators will reduce preload, reduce congestion and minimize cardiac oxygen demand
- Vasodilators can also decrease SVR (afterload), decreased ventricular workload, increase SV and improve CO
  - Causes reflex tachycardia
- May be considered in the absence of hypotension in pts with ADHF who have persistent symptoms despite aggressive treatment with diuretics (i.e. if pts are not hypotensive and not responsive to diuretic)
- Frequent dose titration often necessary: 10-20 mcg/min IV, increase by 5-20 mcg/min q5min PRN
- Contraindications: tachyphylaxis, hypotension

INOTROPES:

- Short-term infusions remains controversial with limited RCT data on the efficacy and safety
- May be considered to relieve symptoms and improve end organ function in patients with advanced HF and diminished peripheral perfusion (ex// low cardiac output) but patients monitored in CCU/ICU
- May also be used in those with marginal or symptomatic BP (< 90 mmHg) despite adequate filling pressures or are unresponsive or intolerant to vasodilators

DOBUTAMINE:

- Synthetic catecholamine with primarily B1 receptor agonist
- Some B2 receptor activity
- 3-5 days infusion in ADHF has improved symptoms for up to 30 days
- Intermittent therapy in severe HF results in worse outcomes

MILRINONE:

- Phosphodiesterase inhibitor
- Activity not mediated by stimulation of B-receptors, thus not diminished by concomitant administration of B-blockers

CONCLUSIONS:

- Intravenous loop diuretics should be given to all patients with ADHF
- Vasodilators should be considered in the absence of hypotension in patients with ADHF who have persistent symptoms despite aggressive treatment with diuretics
- Inotropes should generally be avoided but may be considered to relieve symptoms and improve end organ function in patients with advanced HF and diminished peripheral perfusion (ex// low cardiac output) despite adequate filling pressures or in patients unresponsive or intolerant to vasodilators