



Essential Learning

Acute Decompensated Heart Failure

- **Differential for Acute SOB**
 - Pulmonary: asthma, COPD, PE, pneumothorax, pulmonary infections, airway obstructions (angioedema, aspiration)
 - Cardiac: ACS, HF, dysrhythmia, tamponade, valve dysfunction
 - Other: anemia,
- **What are the general classifications for heart failure?**
 - HF = heart's inability to meet the body's metabolic demands due to impaired filling or ejection of blood
 - Right-sided
 - Common causes include L-sided failure, PE, lung disease, pulm HTN
 - Signs and symptoms include JVD, edema, hepatic congestion, sob
 - Left-sided
 - Common causes include ischemia, HTN, valvular disease
 - Signs and symptoms include fatigue, SOB, DOE, PND, S3 on auscultation
- **What is the difference between HFrEF and HFpEF?**
 - Heart failure with reduced ejection fraction (HFrEF)
 - Impaired contraction and forward flow
 - Left ventricular ejection fraction (LVEF) $\leq 40\%$ (norm 50-70%, mildly reduced 41-49%)
 - Usually related to HTN, ischemia, valve disease
 - Afterload sensitive
 - Heart failure with preserved ejection fraction (HFpEF)
 - Impaired filling
 - LVEF $\geq 50\%$
 - Usually related to HTN, scarring
 - Preload dependent and higher risk for hypotension with diuresis
- **What history findings are associated with heart failure exacerbations?**
 - Commonly, weight gain (increased from "dry weight") or decreased urine output despite taking diuretics, orthopnea, worsening LE edema
 - Highest sensitivity - DOE
 - Highest specificity - orthopnea, PND, edema
- **What is the workup for acute heart failure exacerbations?**

- Ultimately, this is a clinical diagnosis; no single test is diagnostic for acute HF.
- Labs: BMP, CBC, magnesium, BNP, troponin (if indicated)
- Diagnostics: ECG, POCUS, CXR (not 100% sensitive as up to 20% of patients with CHF will have an unremarkable CXR)
- **What is the role of BNP in heart failure?**
 - BNP < 100 pg/mL has a NPV of 90%
 - BNP > 500 pg/mL has a PPV of 90%
 - Causes of elevated BNP- acute decompensated heart failure, renal failure, PE, lung disease with right-sided heart failure
 - Cause of normal BNP in CHF - obesity, mitral stenosis/regurgitation, flash pulmonary edema (if measured < 1-2 hours after onset as BNP can take that long to rise)
- **What is the management for acute heart failure exacerbations?**
 - General
 - Goal is to decrease LVEDV, which improves SV and CO (Frank-Starling Curve)
 - Treatment includes:
 - Supplemental oxygen, BiPAP or CPAP
 - Decrease preload/afterload with high dose nitroglycerin (>100-250 µg/min) if hypertensive
 - Loop diuretics IF the patient is hypervolemic
 - General guidelines (ideal dosing is controversial)
 - Furosemide 20-40 mg IV (or equivalent) if naive
 - IV dose of normal PO dose (some give up to 2.5x home dose)
 - Higher doses are required for patients with CKD
 - Gentle IV fluids if the patient is hypovolemic
 - Inotropes for cardiogenic shock
 - If the patient has remarkably LOW blood pressure -> consider cardiogenic shock
 - Positive pressure ventilation
 - Provide blood pressure support
 - Give small IV fluid challenges (250cc, repeat per clinical effect) if the patient is hypovolemic.
 - Start pressors/inotropes as needed (norepinephrine, epinephrine, dobutamine, milrinone)
 - Consider triggers for cardiogenic shock, including acute ischemia and valvular dysfunction.
 - Consult cardiology/heart failure, ICU or cardiothoracic surgery for patients in extremis who might benefit from a balloon pump, LVAD or ECMO.
 - If the patient has remarkably HIGH blood pressure -> consider sympathetic crashing acute pulmonary edema (SCAPE)

- **What is Sympathetic Crashing Acute Pulmonary Edema (SCAPE)?**
 - Acute onset heart failure associated with severe hypertension
 - Also called flash pulmonary edema
 - A rapid increase in LV end diastolic filling pressures → interstitial and alveolar edema → hypoxia → catecholamine surge → increased SVR and further LV dysfunction → worsening pulmonary edema
- **What are the signs and symptoms seen with SCAPE?**
 - Rapid progression of dyspnea, cough productive of pink and/or frothy sputum
 - Tachycardia, hypertension, hypoxia, rales or wheezes, S3, JVD, peripheral edema
- **What is the management for SCAPE?**
 - Goal is to decrease LVEDV which improves SV and CO (Frank-Starling curve)
 - Treatment includes:
 - Decrease preload/afterload (nitroglycerin +/- diuretics)
 - BiPAP or CPAP
 - Inotropes for cardiogenic shock (norepinephrine, dobutamine, milrinone)
- **What medications should be used in treating SCAPE?**
 - While IV access is being obtained, start with 0.4 mg SL nitroglycerin tablets every 3-5 minutes (up to three doses)
 - 1 tablet of 0.4 mg SL nitroglycerin every 3-5 minutes = 80-133 mcg/min-keep this in mind when starting nitroglycerin gtt
 - Nitroglycerin bolus 400mcg-1000mcg depending on the BP followed by an infusion
 - Nitroglycerin infusion at 100-200 mcg/min (titrate by 20-40 mcg/min q3-5 min)
 - In higher doses, nitroglycerin reduces both preload and afterload
 - Some advocate for starting rates at 400 mcg/min and titrating as needed
 - Further afterload reduction with ACE-Inhibitors
 - Consider captopril 12.5 mg SL or 0.635 mg enalapril IV
 - Morphine has historically been used to decrease dyspnea but has been associated with increased mortality and should generally be avoided in this population
- **What is the role of diuretics in treating SCAPE?**
 - Historically, diuresis with loop diuretics has been standard of care
 - Not all patients with SCAPE are volume overloaded (more volume shifted into the lungs) so treatment with diuretics should be based on the patient's history and exam
 - Patients who are volume overloaded should be given loop diuretics, generally the IV equivalent of their home dose (or up to 2.5 the home daily PO dose if clinically indicated)

- **POCUS Pearls**

- POCUS can help determine the cause of acute dyspnea - it is faster and more sensitive than CXR for pulmonary edema
- Patients with pulmonary edema will typically have B-lines on ultrasound bilaterally
 - B-lines are reverberation artifacts that arise from the pleural line and extend the depth of the screen without fading
 - They move with ventilation and lung sliding
 - > 3 B-lines in > 2 lung fields is diagnostic of interstitial edema
- In contrast, patients with COPD, asthma, or normal lungs will have an A-line pattern of repeating horizontal reverberation artifacts
 - B-lines obscure A-lines
- Not all B-lines occur secondary to edema as anything that causes thickening of the interlobular septae (i.e., increased lung density) causes B-lines
 - Examples include pulmonary fibrosis, ARDS, early pneumonia, pulmonary contusion
 - Combining thoracic ultrasound results with POC echo and clinical history is helpful for interpreting your scan

- **Attributions**

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