Heart Failure
Diagnosis and Management

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Objectives

- Heart Failure Diagnosis
  - Risk factors and HF classifications
  - Recognition of ED presentations of HF
  - General considerations
    - Low EF, Preserved EF
  - Acute decompensation

- Emergency Department management
  - Initial treatment
  - Optimization of chronic management
  - Treatment in acute decompensation
Introduction

- Increasing impact of Heart Failure
  - Over 5 million people living with heart failure
  - >550,000 new diagnoses each year
  - 3+ million hospital admissions each year
  - Incidence of 10 per 1000 by age 65
  - 80% of hospitalizations are in patients >65 years old
- Increasing total numbers, increasing numbers of deaths
  - Better acute care of ACS
    - Leads to higher numbers of people living with HF
  - Aging population increases numbers of people living with many chronic diseases of aging
HF Diagnosis: General Considerations

- HF is the **clinical syndrome** that results from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.

- Clinical diagnosis
  - No **single confirmatory test**
  - Testing can support diagnosis or reveal alternate diagnosis.
Introduction: Definitions

Heart Failure

- A clinical syndrome with specific symptoms
  - Primarily dyspnea and fatigue
- And specific signs
  - Edema and rales
- With evidence of impaired cardiac function
  - Primarily echocardiographic
- A clinical diagnosis with no single diagnostic test
- **Not synonymous with cardiomyopathy or LV dysfunction**
  - Both may be reasons or etiologies for heart failure
Introduction

Terms

- Heart Failure with Reduced Ejection Fraction
  - Heart failure with low EF
  - AKA: Systolic dysfunction
  - HFrEF
- Heart Failure with preserved Ejection Fraction
  - “diastolic dysfunction”
  - HFpEF

HF as a clinical diagnosis
- Symptoms and history are most important
There is poor correlation between cardiac performance and symptoms

- Patients may have very depressed LVEF and few symptoms, or may have mildly impaired EF and severe symptoms

Many factors affect symptoms

- Cardiac: Ventricular distensibility, valvular function, pericardial restraint, rhythm, conduction, and RV function
- Non-Cardiac: Peripheral vascular function, skeletal muscle physiology, pulmonary function, neurohormonal and sympathetic function, renal sodium handling
Progression of Disease

- Cardiac Remodeling
  - HTN, DM, CAD and Afib are all primary and independent causes of cardiac remodeling

- Additional increases in cardiac remodeling
  - Norepinephrine, Angiotensin II, Aldosterone, Endothelin, Vasopressin, Cytokines
    - Also lead to increased sodium retention and vasoconscription
    - Direct toxic effects on cardiac cells and increased myocardial fibrosis
AHA/ACC Guidelines

- **2005**: AHA/ACC Heart Failure Working Group published comprehensive guidelines for the diagnosis and management of heart failure
  - Focused on diagnosis, stages of disease, chronic and long term management
- **2009**: Focused update of the 2005 guidelines
  - Updated information on many topics
  - Added management of acute decompensation of the patient with HF
    - More useful for ED management
- **2013**: Comprehensive update of the guidelines
  - Evidence based, use Level I, IIa, IIb, III classification to guide recommendations
  - Comprehensive literature reviews
NYHA class vs. HF stage

- A single patient can move back and forth from one functional (NYHA) class to another
  - Improvement from higher class to lower often happens with initiation or improvement of treatment
  - Signifies a current functional assessment

- Heart failure stages are progressive and forward only
  - Once a patient reaches stage C (structural disease with prior or current symptoms), that patient does not go back to stage B, even if symptoms improve
    - Implications for treatment (once symptoms are present for any length of time, management should be more aggressive)
NYHA Functional Classification of Heart Failure

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
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</table>
| 1     | No limitation of physical activity  
       | Physical activity does not cause fatigue, palpitation or shortness of breath |
| 2     | Slight limitation of physical activity  
       | Comfortable at rest, but physical activity results in fatigue, palpitations or shortness of breath |
| 3-A   | Limitation of physical activity  
       | Comfortable at rest, but ordinary activity causes fatigue, palpitations or shortness of breath |
| 3-B   | Significant limitation of physical activity  
       | Comfortable at rest, but minimal activity causes fatigue, palpitation or shortness of breath |
| 4     | Unable to carry on any physical activity without discomfort  
       | Symptoms of heart failure at rest |
Mr. A is 65 years old with previous CAD s/p stent who presents with increasing DIB on exertion for the past week. He can usually go up 2 flights of stairs or walk 3 blocks before he gets short of breath, but over the past week, he has noticed that he is SOB going up the stairs in his house and walking into the grocery store from his car.

- What NYHA Functional Class is he?
- What Class was he before?
Figure 1. Stages in the development of heart failure/recommended therapy by stage. FHx CM indicates family history of cardiomyopathy; ACEI, angiotensin converting enzyme inhibitors; and ARB, angiotensin receptor blocker.
Heart Failure Stages

- **Stage A**
  - At risk, but no disease
    - Control risk factors

- **Stage B**
  - Structural Heart disease but no symptoms
    - CAD, s/p MI, Impaired EF, etc but no S/S
      - Start specific treatments (ACE-I, Bblockers)

- **Stage C**
  - Structural Disease with prior or current symptoms
    - HF specific treatments (diuretics, vasodilators, pacing, defibrillators, etc)

- **Stage D**
  - All of above with symptoms at rest despite medical treatment (End Stage CHF)
    - “Heroic Measures”, transplantation, palliative care
Patient #1

- What Heart Failure Stage is Mr. A?
- How might he change Stage?
## Modified Framingham Clinical Criteria for Diagnosis of HF

<table>
<thead>
<tr>
<th>Major</th>
<th>Minor</th>
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<tbody>
<tr>
<td>Paroxysmal nocturnal dyspnea</td>
<td>Bilateral leg edema</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>Nocturnal cough</td>
</tr>
<tr>
<td>Elevated JVP</td>
<td>Dyspnea with ordinary exertion</td>
</tr>
<tr>
<td>Pulmonary rales</td>
<td>Hepatomegaly</td>
</tr>
<tr>
<td>Third heart sound</td>
<td>Pleural effusion</td>
</tr>
<tr>
<td>Cardiomegaly on CXR</td>
<td>Tachycardia (&gt;120bpm)</td>
</tr>
<tr>
<td>Pulmonary edema on CXR</td>
<td>Weight loss of &gt;4.5kg in five days in response to treatment</td>
</tr>
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</table>
Mrs. B is 65 years old, and has a history of HTN, DM, and CAD. She had a NSTEMI about a year ago, had angioplasty with stenting done. She presents to the ED complaining of 3 weeks of progressive debilitating fatigue that is worse with exertion, nocturnal cough, and orthopnea. On CXR she has moderate cardiomegaly with mild pulmonary edema.

What work up needs to be done?
Making a HF diagnosis

- **Class I recommendations**
  - Complete H & P with particular focus on risk factors
  - History of alcohol, illicit drugs, alternative therapies, chemotherapy
  - Assessment of ability to perform ADLs (and desired ADLs)
  - Assessment of volume status, orthostatics, BMI
  - Lab studies
    - CBC, UA, Lytes, LFTs, lipids TSH
  - 12 lead EKG; CXR
  - Echocardiography
  - Coronary arteriography if angina or ischemia present
Making a HF Diagnosis

- **Class Ila Recommendations**
  - Coronary arteriography if chest pain present and CAD history unknown
    - Or if CAD present with no chest pain
  - Noninvasive imaging for detection of myocardial ischemia
  - Exercise testing
  - Screening for HIV, sleep apnea, hemachromatosis
  - Screening for rheumatologic disease, amyloidosis or pheochromocytosis
  - Endomyocardial biopsy if clinically indicated
  - Measurement of BNP in acute settings if clinical diagnosis uncertain
Patient #3

- Mr. C is 65 years old with history of HTN (poorly controlled) and alcoholism who presents with 2 weeks of progressive lower extremity edema, dyspnea (worse with exertion), abdominal discomfort and palpitations.

- Exam shows rales in the lungs, JVD, S3 heart sound, mild RUQ tenderness, and lower extremity edema
HF presentation: General

- Symptoms of excess fluid accumulation
  - Dyspnea, orthopnea, edema, abdominal pain (hepatic congestion, ascites)
  - Fluid retention is initiated by fall in cardiac output, then altered renal function
    - Activation of sodium retaining renin-angiotensin-aldosterone; activation of sympathetic nervous system

- Symptoms of decreased cardiac output
  - Fatigue, weakness
    - Especially exercise induced
HF Presentation: Chronic vs. Acute/Subacute

- Development over months:
  - Fatigue, anorexia, abdominal distension, peripheral edema may be more pronounced than dyspnea
  - May be more likely to have pleural effusions than pulmonary edema

- Development over days to weeks
  - Characterized primarily by shortness of breath
    - At rest and/or with exertion
  - Commonly present:
    - Orthopnea, PND, RUQ pain with right side failure
    - Palpitations if rhythm problems
Diagnosis of HF: Physical Exam

- Pulmonary congestion
  - Rales likely more prominent in acute to subacute presentation
  - Decreased breath sounds (pleural effusions) may be present in chronic
- Peripheral edema
  - Continued fluid retention preferentially accumulates in the periphery
  - Leg edema, ascites, hepatomegaly, splenomegaly
  - Hepatojugular reflux
- Elevated JVP
  - Usually present if peripheral edema is due to HF
- Pulsus alternans
  - Pathognomonic for severe LV dysfunction
S3 Heart Sound

- Occurs 0.12-0.18 seconds after S2
  - Changes “lub dub” sound to “lub dub ing”
- Lower frequency than S1 and S2
- May be due to atrial “kick”, or tensing of chordae tendinae due to fast ventricular filling
- Specific for increased LV filling pressures
- Wide variability in ability to detect
  - Not accounted for by clinical experience
HF Diagnosis: Physical Exam

- Advanced HF (evidence of decline in cardiac output)
  - Resting sinus tachycardia
  - Narrow pulse pressure
  - Diaphoresis
  - Peripheral vasoconstriction (cool extremities)
- Low output state with sympathetic compensation
Mrs. D is a 65 year old female with a history of hypertension and DM who presents with fatigue and dyspnea on exertion for the past few days.
HF Diagnosis: Testing

- EKG
  - Normal EKG makes systolic dysfunction unlikely
    - 98% negative predictive value
  - May show findings that favor CAD as underlying cause of heart failure
  - May favor a specific cause (afib or tachydysrhythmia)
  - Need to evaluate for current infarct or ischemia
Patient #4

- Mrs. D is a 65 year old female with a history of hypertension and DM who presents with fatigue and dyspnea on exertion for the past few days.
- CXR: Cardiomegaly
- Hemoglobin: 7g/dl
- BNP: 700
HF Diagnosis: Testing

- **CBC**
  - Anemia or infection can be exacerbating condition

- **Serum Lytes**
  - With BUN/Cr
  - Hyponatremia indicates severe HF (or excessive diuresis)
  - Renal function impacts fluid status, use and effectiveness of diuretics

- **LFTs**
  - Liver congestion
  - GGT >2 times the upper limit of normal

- **Fasting Blood Glucose**
  - Underlying DM as an independent risk factor
HF Diagnosis: Testing

- Chest Xray
  - Differentiation of HF from pulmonary disease
  - Findings of HF
    - Cardiomegaly
    - Cephalization of the pulmonary vessels
    - Kerley B-lines
    - Pleural effusions
  - Cephalization and cardiomegaly are best predictors
  - Alveolar edema, Interstitial edema, cephalization have specificity of >90%, but only cardiomegaly was >50% sensitive
HF Diagnosis: BNP Testing

- Brain natriuretic peptide
  - Primarily released from ventricles
  - Pro-hormone (pro-BNP) is cleaved
    - BNP and NT-proBNP are both released into circulation
- Addition of BNP or NT-proBNP testing adds more to diagnosis of dyspnic patient with HF than any other test
  - Cannot stand alone, should only be done as confirmatory testing in appropriate clinical setting
BNP testing

- Levels <100
  - High negative predictive value for HF as cause of dyspnea

- Levels of 100-400
  - Indeterminate
  - Non sensitive or specific
  - Other diagnoses should be considered
  - Pulmonary embolus, LV dysfunction without exacerbation, cor pulmonale

- Levels >400
  - HF very likely
BNP Testing

- “False” elevations
  - Atrial fibrillation
    - Even in absence of HF
  - Increasing age
  - Women > men
  - Pulmonary hypertension
  - Renal Failure
  - Sepsis
- “False” decreases
  - Obesity
  - Diabetes?
BNP Testing: Limitations

- Multifactorial Dyspnea
  - HF patient with pneumonia or PE
- Chronic elevation
  - Some chronic HF patients will have persistent elevation of BNP
- Acute exacerbation (“Flash” pulmonary edema)
  - BNP may lag behind symptoms in the most acute settings
## Non-CHF Causes of elevated BNP

<table>
<thead>
<tr>
<th>Causes</th>
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<tbody>
<tr>
<td>Advanced Age</td>
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<tr>
<td>Renal Failure</td>
</tr>
<tr>
<td>Myocardial infarction</td>
</tr>
<tr>
<td>Acute Coronary Syndrome</td>
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<tr>
<td>Lung Disease with Right-Sided Failure</td>
</tr>
<tr>
<td>Acute Large P.E.</td>
</tr>
<tr>
<td>Cirrhosis</td>
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## Lower than expected BNP

<table>
<thead>
<tr>
<th>Causes</th>
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</thead>
<tbody>
<tr>
<td>Flash Pulmonary Edema</td>
</tr>
<tr>
<td>CHF 2° to Upstream Abnormality:</td>
</tr>
<tr>
<td>Acute Mitral Regurgitation</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
</tr>
<tr>
<td>Atrial Myxoma</td>
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</table>
Undifferntiated dyspnea and BNP

1. Patient presenting with dyspnea
   - Physical examination, chest x-ray, ECG, BNP level

2. BNP level categories:
   - BNP < 100 pg/mL
   - BNP 100–400 pg/mL
   - BNP > 400 pg/mL

3. Baseline LV dysfunction, underlying cor pulmonale or acute pulmonary embolism?
   - "Yes" or "No"

4. Outcomes:
   - CHF very unlikely (2%)
   - Possible exacerbation of CHF (25%)
   - CHF likely (75%)
   - CHF very likely (95%)
HF Diagnosis: Echocardiography

- Confirms presence of cardiac impairment and can identify cause
- Findings
  - Atrial and ventricular sizes
    - Identification of cause and chronicity of disease
  - Global RV and LV systolic function
  - Diastolic LV function
  - Regional wall motion abnormalities
  - Pericardial disease
  - Valvular disease
Ms. E is a 65 year old woman with a history of difficult to control HTN who presents with shortness of breath, tachycardia and hypertension. She has wheezing and rales on exam.

Bedside echo: LVH with normal EF
HFpEF

- Heart Failure with normal ejection fraction
  - Diastolic dysfunction
    - Abnormal LV filling, elevated LV filling pressure
  - Valvular disease
    - Stenosis or regurgitation
- Right heart failure
- Pericardial Disease
- Atrial myxoma
- Pulmonary vein stenosis
HFpEF

- Women > Men
- Increased at older ages
- Lower in hospital mortality
- Causes
  - Chronic HTN with LVH
    - Hypertrophy with LVEF > 75%
  - Hypertrophic cardiomyopathy
  - Aortic stenosis
  - CAD
  - Restrictive cardiomyopathy
HFpEF

- LV filling shifts toward end of LV diastole
  - Becomes more dependent on atrial contraction
    - **Patients with HFpEF tolerate loss of atrial contraction poorly**
      - Atrial fibrillation
      - Tachycardias

- Elevations in systemic blood pressure increase LV wall stress and can further impair LV relaxation
  - **HFpEF patients tolerate sudden BP elevations poorly**

- Acute exacerbations increase LA pressures, pulmonary venous pressure
  - Patients may have pulmonary symptoms out of proportion to expected
  - “Anginal equivalents” of SOB
  - “Cardiac Wheezing”
Mr. F is a 65 year old male with known HF who presents with acute onset of dyspnea today, 2 days after running out of his medications.

Vitals: HR 105, RR 28, BP 190/110, SpO2 88%

Exam: Lung exam w rales in lower 1/3, +JVD, minimal LE edema
Acute Decompensation in HF

Clinical Syndrome

- Acute dyspnea
  - Associated with: Rapid accumulation of fluid within the lung’s interstitial and alveolar spaces
  - Resulting from: Elevated cardiac filling pressures
  - Resulting in: Cardiogenic pulmonary edema

- Other causes of acute pulmonary edema (non HF)
  - Primary fluid overload (iatrogenic, renal disease)
  - Severe hypertension

“Flash” pulmonary edema

- Any cause (cardiogenic or other) of pulmonary edema that occurs in faster than usual fashion
  - Etiology, diagnosis and treatment are similar
  - Rapid onset necessitates more rapid management
ADHF Clinical Signs and Symptoms

- Presentation with dyspnea, cough and fatigue, +/- chest discomfort
  - Progressive worsening of symptoms
- Tachypnea, use of accessory respiratory muscles
- Tachycardia
- Hypertension or hypotension
  - Hypotension may indicate severe LV dysfunction and impending cardiogenic shock
- Lung Exam:
  - Crackles (interstitial pulmonary edema)
  - Wheezing (cardiac asthma)
    - Present in as many as 1/3 elderly HF patients with dyspnea
    - Associated with increased hypercapnia but similar mortality
- Presence of S3 and/or S4
- Elevated JVP
ADHF: Precipitating Factors

- Dietary or medication non-compliance
- Iatrogenic volume overload
- Drug interactions
  - Addition of negative inotropes
    - Verapamil, Nifedipine, Beta blockers, NSAIDs
- New MI or ACS
- Progression of underlying disease
- Arrhythmia (new afib, SVT, VT)
- Cardiotoxins (ETOH, cocaine, chemotherapy)
- Severe hypertension
- Renal failure
- Pulmonary embolism
- Sepsis
- Anemia
- Thyroid disorders
ADHF Diagnosis

- Search for proximate cause
- EKG, blood work (including troponin, ABG)
- BNP
- CXR
- Range of findings from isolated cardiomegaly with mild redistribution to bilateral alveolar edema
- Pleural effusions likely not present in acute exacerbation
- **Normal CXR does NOT rule out ADHF**
Treatment Overview for HF

Pharmacologic:
- Diuretics - preload reduction: thiazide, loop etc
- Vasodilators (nonspecific) Hydralazine, Nitrates
- Afterload reduction: ACEi, ARBs
- Inotropic agents - to improve contractility
- Beta-adrenergic blockade - improves remodeling
- Natriuretic Peptide?

Adjunctive Therapies:
- Patient positioning
- Oxygen and Noninvasive ventilation
- IABP
Treatment of HF: Chronic

- Treatment as a chronic disease
- Management:
  - Decrease morbidity (including hospital admission and readmission)
  - Decrease symptoms
  - Increase functional status
  - Decrease progression
- Treatment of underlying cause
  - Management of HTN to include B blocker, ACE inhibitor or ARB and aldosterone antagonist (spironolactone)
  - Renovascular disease
  - Coronary artery disease
- AICD, Cardiac resynchronization therapy (biventricular pacing)
Chronic Treatment of HF

Class I Recommendations
- Diuretics and salt restriction
- ACE inhibitor
- Beta blockers
  - 3 have proven mortality reduction (bisoprolol, carvedilol, and sustained release metoprolol succinate)
- ARBs in patients who cannot tolerate ACE-Is
- Avoidance of drugs with adverse effects
  - NSAIDs, many antiarrhythmics, calcium channel blockers
- Introduction of exercise training
- AICD when indicated
  - Vent arrhythmia, EF<35% with NYHA class II or III symptoms
- Resynchronization therapy (if QRS >120)
- Aldosterone antagonist (watch renal function and K level)
- Combination of hydralazine and nitrates
Chronic Treatment of HF

- Class IIa Recommendations
  - Afib treatment: conversion or rate control
  - Maximal exercise testing
  - ARBs instead of ACE-I as first line therapy
  - Digitalis to prevent morbidity
  - Cardiac resynchronization therapy and/or AICD in broader indications
Chronic HF Treatment

- Class III (not recommended)
  - Routine use of calcium channel blockers
  - Long term infusion of positive inotrope
    - Except in end-stage and palliative care
  - Intermittent outpatient use of IV inotropes
  - Nutritional supplements
  - Hormonal therapies without documented hormonal deficiency
Chronic Heart Failure Treatment

Order of therapy may be important

1. Loop diuretics for fluid control and subsequent symptom relief
2. ACE inhibitors (ARB if not tolerated) which provide rapid hemodynamic benefit without exacerbation of HF
3. Beta blockers started at low dose and titrated upwards
4. Addition of aldosterone antagonist especially in DM or post MI
5. Combined hydralazine and nitrate
6. Digoxin
Order of ACE-I vs Beta Blocker

- ACE-I have a rapid achievement of hemodynamic benefit
  - No negative inotropic effect, can be started soon after acute phase

- Beta blockers
  - Hemodynamic benefit is delayed (weeks to months)
  - May have transient worsening of cardiac function at initiation
  - Dose related effect (but benefit still present in patients who cannot tolerate full dose)
Diuretics

- Useful for acute therapy and symptom relief, no evidence to support decreased mortality
- Generally loop diuretics used, thiazide may be used for mild CHF
- Combination with nitroglycerin
  - High dose NTG + Low dose diuretic better than High dose diuretic + Low dose NTG
ACE inhibitors/ Angiotensin II Receptor antagonists

- ACEi most efficacious agent for symptomatic and asymptomatic CHF
- Enalapril appears to lower mortality more than hydralazine-nitrate combination
- ARBs recommended for ACEi intolerance
- Overall reduction in mortality 30-35%
- HOPE trial showed decrease mortality, CVAs, MIs in patients with vascular disease or DM
Digoxin

- Not for acute management
- Digitalis decreases CHF-specific mortality when combined with diuretics in Class III/IV CHF
- DIG trial showed decreased hospitalizations but no overall decrease mortality
- Avoid in extremely elderly, renal insufficiency, patients on quinidine or diastolic dysfunction
Beta-Blockers

- Not for acute management of CHF
- Carvedilol first approved BB with clear decrease in mortality and morbidity
- Carvedilol improves EF, exercise tolerance and symptoms (long term)
- Low dose Metoprolol may prolong survival in patients with EF <40%
- Reduces toxic effects on myocytes, halts remodeling
Spironolactone

- Previously presumed ACEi would curtail or suppress aldosterone formation
- Evidence points only to transient suppression of aldosterone by ACEi
- Addition of spironolactone to regimen of diuretic and ACEi decreased mortality in patients with Class III, IV CHF (RALLES)
Patient #7

- Mr. G is a 65 year old male with known HF with EF=30% who is compliant with medications (carvedilol, Lisinopril, Lasix) who presents with palpitations and dyspnea.
- Vitals: HR 125, RR28, BP 160/90, SpO2 92%
- EKG: Afib w RVR
- CXR: Cardiomegaly with mild congestion

What interventions should be done?
ADHF Treatment Goals

- Symptom improvement
- Normalization of oxygenation
- Optimize volume status
- Identify etiology/precipitating factors
- Initiate/optimize oral therapy
- Identify patients who would benefit from revascularization
- Identify risk of thromboembolism and treat
- Identify patients who would benefit from device therapy
- Patient education
Approach to ADHF

- ABCs
- Cardiac monitor, vitals
- IV access
- Upright patient positioning
- Diuretic therapy
  - Loop diuretic administered IV
- Vasodilator therapy
  - If not hypotensive
- Urinary output measurements
Approach to ADHF

- Initial goal is rapid correction of hemodynamic and intravascular volume abnormalities
- Tailor therapy to situation
  - Hypertensive emergency
    - Aggressive vasodilation
  - Normotension
    - Balanced diuresis and vasodilation
  - Hypotension
    - “Gentle” diuresis =/- inotropes
Management of Atrial Fibrillation in ADHF

Chicken vs Egg

- HF increases risk for AFib
- Afib (esp new onset or with RVR) increases risk for HF exacerbation
- Need to control rhythm (or at least control rate) to maximize cardiac function
- Common treatments (beta blocker, calcium channel blocker) cause myocardial depression
- Since acute HF can precipitate afib, early cardioversion may lead to recurrence of Afib
Afib and ADHF

- Short acting IV beta blockers or calcium channel blockers are used to control rate with least amount of negative inotropic effect
- Digoxin is traditional choice, but even in IV administration, onset is hours
  - Significant toxicity concerns
- Amiodarone may be used
  - May cardiovert and/or control rate
  - Consider initiation of heparin therapy first
- Cardioversion
  - If cardiogenic shock present or impending
  - If Afib is the definite proximal cause of the HF
  - Start heparin first if possible
Mrs. G is a 65 year old woman with a history of known severe HF. She has recent echo with EF = 20%, presents with severe dyspnea.

- HR 120, BP 165/100, RR 36, SpO2 85%
- CXR with moderate pulmonary edema
- Supplemental O2 does not improve status

What intervention should be done?
Ventilatory Support in ADHF

- Supplemental oxygen administration
  - Initial high flow O2, titrate down to pulse ox
- Early NPPV (non-invasive positive pressure ventilation)
  - Indications:
    - Respiratory distress
    - Respiratory acidosis
    - Persistent hypoxia
  - Improves respiratory parameters
    - Dyspnea, hypercapnia, acidosis, heart rate
  - Decreases need for intubation
  - Assists patient by decreasing the work of breathing, enhances O₂/CO₂ exchange and increases cardiac output
Noninvasive Ventilatory Support

- Decreased work of breathing:
  - Alveoli may fill with water diluting surfactant, making lungs stiff
  - Positive pressure prevents collapse at end-exhalation

- Enhanced O₂/CO₂ Exchange:
  - By keeping alveoli open longer increases exchange time

- Increasing Cardiac Output:
  - By providing positive pressure for entire thorax creates a pressure differential favoring cardiac output and cardiac index
| **Table 2. Selecting Patients for Noninvasive Ventilation.** |

**Inclusion criteria**
- Acute or chronic respiratory failure
- Acute pulmonary edema
- Chronic congestive heart failure with sleep-related breathing disorder

**Relative contraindications**
- Failure of prior attempts at noninvasive ventilation
- Hemodynamic instability or life-threatening arrhythmias
- High risk of aspiration
- Impaired mental status
- Inability to use nasal or face mask
- Life-threatening refractory hypoxemia ($\text{PaO}_2 < 60 \text{ mm Hg with } 1.0 \text{ FIO}_2$)*

*PaO\(_2\) denotes partial pressure of oxygen, and FIO\(_2\) fraction of inspired oxygen.
Mr. I is a 65 year old man who presents with acute dyspnea and a medication list consistent with HTN and HF. He states he is out of his medications, and presents with the following vitals:

- BP 240/130, HR 132, RR32, SpO2 87%
- What intervention should be started?
ADHF Treatment: Vasodilators

- IV nitroglycerin is first line for symptom control
  - Acute pulmonary edema or severe hypertension
  - May add to diuretic therapy if normotensive to decrease symptoms
  - Primary action is venodilation and decreased preload
  - Higher doses have variable lowering of afterload and increased stroke volume and cardiac output
- Nitroprusside if severe hypertension not responding to nitroglycerin
- Careful titration to avoid hypotension
ADHF treatment: Nesiritide

- Recombinant BNP
- Vasodilator
- Clinical trials with mixed results
  - Trend toward improvement in dyspnea symptoms
  - Hypotension as adverse effect
  - No mortality difference (early trials thought to show increase in mortality, later did not)
  - No significant effect on renal function
- No treatment benefit in comparison to standard vasodilator therapy
Patient #10

- Mrs. J is a 65 year old woman, brought to the ED by her daughter for weakness and fatigue for the past few days, associated with increasing orthopnea and dyspnea. She has a known history of HF with EF 25%.
- Vitals: HR 90 BP 80/60 RR 28 SpO2 85%
- CXR with mild pulmonary congestion
- How should her ADHF be managed?
ADHF: Inotropes

- Useful in patients with severe LV dysfunction and low output syndrome
  - Diminished peripheral perfusion or end organ dysfunction
  - Marginal blood pressure or hypotension
  - Inadequate response to vasodilator and diuretic therapy, or worsening renal function
  - Not indicated in preserved systolic function
- May increase heart rate, increase myocardial oxygen consumption, increase arrhythmias
Milrinone

- Phosphodiesterase inhibitor
  - Increases myocardial inotropy by inhibiting degradation of cAMP
  - Reduces systemic and pulmonary vascular resistance
  - Improves LV compliance
  - Increased cardiac index
  - Decreased LV afterload
  - Decreased LV filling pressure
- Loading dose 50mcg/kg; 0.375-0.750mcg/kg/min
- Does not act on beta receptors, not affected by beta blocker use
Dobutamine

- Acts on beta 1 adrenergic receptors
  - Increased stroke volume
  - Increased cardiac output
  - Modest decrease in systemic vascular resistance and pulmonary capillary wedge pressure
  - Useful in hypotension without shock
- Effect diminished by beta blockade
Treatment of Acute Decompensated Heart Failure (ADHF)

Criteria for Hospitalization

- Severe decompensation
  - Hypotension
  - Worsening renal function
  - Altered mentation
- Dyspnea at rest
  - Tachpnea > decreased 02 saturation
- Hemodynamically significant arrhythmia (inc. new afib)
- Acute coronary syndromes

Consideration of hospitalization

- Worsened congestive symptoms (even in absence of dyspnea or weight gain)
- Major electrolyte disturbance
- Associated diagnosis (infection, PE, DKA, CVA)
- New HF diagnosis
Disposition

- Admission for all patients with new onset CHF (particularly if moderate to severe)
- Patients with recurrent heart failure complicated by acute life threatening events:
  - recent myocardial infarction
  - pulmonary edema
  - hypotension
  - pulmonary embolism
  - symptomatic arrhythmia
  - other severe medical illness
  - hypoxia
Patients on renal replacement therapy who are deemed as needing admission may be best served on nephrologic service. This decision can be made in consultation with nephrologist.

Verify all electrolyte abnormalities and medications requiring a therapeutic level are corrected.
Summary

- Therapy for Acute Congestive Heart failure:
  - Upright position
  - Oxygen
  - Nitrates (up to 0.8 mg SL), then IV
  - Furosemide (40-80mg IVP)
  - If hypotensive consider inotropic support
  - If hypertensive add Nitroglycerin, Nitroprusside IV, or Hydralazine IV
  - If in respiratory distress
    - Evaluate for CPAP or BiPAP
  - Verify Urine output (foley catheter)
Heart Failure is best treated as chronic disease with focus on long term management.

Diagnosis of chronic heart failure and acute exacerbations are approached in a similar fashion:
- Primarily clinical diagnosis with consistent test results
- Use of BNP, echocardiography for diagnosis

Treatment mainstays are control of underlying disease, diuretics, vasodilation:
- Chronic disease management with ACE-I, beta blockers
- Acute disease management with IV vasodilators, diuretics

Acute decompensation may require ventilatory or inotropic support.