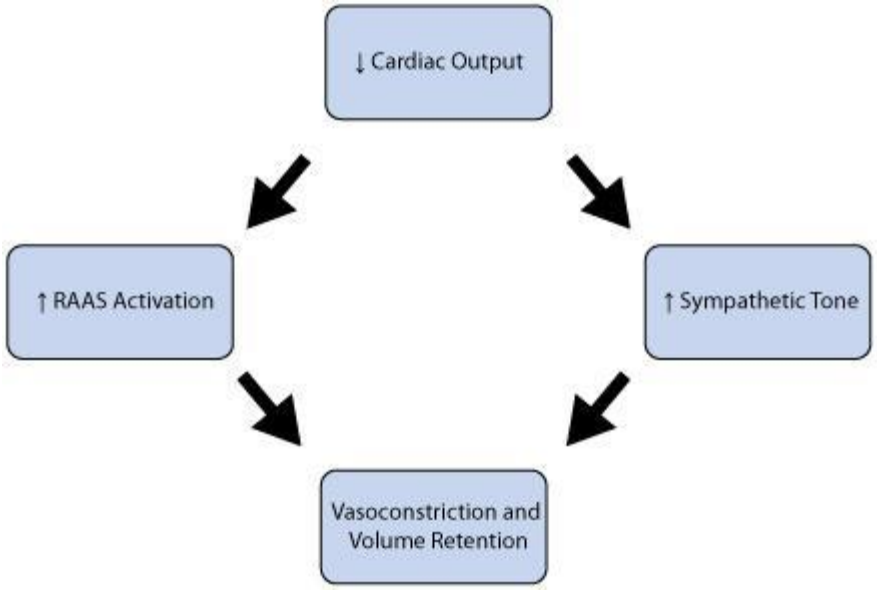
Heart Failure: Overview and Outpatient Management

- Scope of the Problem
    - 1-2% of general population; 10% of patients over 70
- Definition
    - CHF relies upon a syndromic definition of cardinal symptoms (dyspnea, fatigue) and often signs (peripheral or pulmonary edema)
        - LV dysfunction alone is not sufficient for a diagnosis of CHF, particularly on a one-time evaluation (e.g., you'll see a lot of sepsis cardiomyopathy in the ICU that resolves with treatment of the underlying disease process; these patients should not be labeled as having CHF after one TTE). Moreover, LV dysfunction not necessary for the diagnosis - more than 50% of CHF patients have a normal EF
    - CHF without LV dysfunction is not necessarily *diastolic* heart failure
        - Diastolic failure specifically implies an upward shift of the end-diastolic P/V curve (i.e., a decrease in ventricular capacitance and an increase in LVEDP, as is often seen with amyloidosis or other resrictive cardiomyopathies, while many of these patients actually have either no shift a rightward shift (similar to those with systolic dysfunction)
        - Those with an intermediate EF merit mention as these patients show evidence of eccentric ventricular remodeling and decreased contractility more in line with their < 40% counterparts than those > 55%
        - Appropriate paradigm is HFNEF (LVEF > 55%) / HFPEF (40-55%) / HFREF (< 40%)
    - NYHA *Class* quantitates real-time symptomatology; most of our trial data revolves around this
        - Class I: No symptoms with normal activity (can be symptomatic with vigorous activity, e.g. sports)
        - Class II: Symptoms with prolonged or moderate exertion (a flight of stairs, carrying something heavy)
        - Class III: Symptoms with ADL's (dyspneic after walking across room)
        - Class IV: Symptomatic at rest
    - ACC-AHA *Stage* documents the progression of the patient's disease, regardless of current symptoms
        - Stage A: At risk (No signs, symptoms, or functional abnormalities)
        - Stage B: Structural heart disease but no history of CHF signs / symptoms
        - Stage C: Structural heart disease with a history of signs / symptoms
        - Stage D: Advanced structural heart disease and a history marked symptoms at rest despite maximal medical therapy
- Etiologies
    - Main branch point is ischemic or non-ischemic; CAD is the most common etiology (> 60% in NHANES data); other significant causes include valvular disease (particularly in older patients), hypertension, infiltrative disease (sarcoidosis, amyloidosis), toxic cardiomyopathies (alcohol, cocaine), myocarditis (particularly viral)

- Diagnosis
  - History
    - Establish chronicity and pattern of symptoms
    - Screen for confounding conditions (COPD, etc.) and assess exercise tolerance / functional status
    - Left-sided (orthopnea, PND) vs. right-sided (peripheral edema, early satiety or abdominal pain/distension) symptoms
  - Physical
    - Key physical exam characteristics, +/- Mcgee / Sapira data
    - Left vs right
  - Laboratory
    - Elevated BNP (Breathing Not Properly trial) can be useful (particularly in difficult historians) to distinguish between pulmonary and cardiac etiologies of dyspnea
    - Common associated nonspecific abnormalities include hyponatremia, elevated BUN / creatinine, congestive hepatopathy (indirect hyperbilirubinemia), anemia
  - CXR
    - Cardiomegaly
    - Pulmonary edema
  - EKG
    - Look for evidence of ischemic heart disease (old Q waves), systemic hypertension (LVH suggested by increased voltage, left axis deviation, left atrial enlargement, etc.), pulmonary hypertension (right ventricular / atrial hypertrophy), infiltrative disease (low voltage, QRS fractionation)
  - TTE
    - In addition to quantifying EF, can see evidence of diastolic dysfunction (speckling)
  - RHC
    - Discussed elsewhere, but characteristically see elevated PCWP



- Pathophysiology
- Management
    - Risk Factor Modification
        - All patients (including asymptomatic) Stage A / B patients require risk management of comorbidities and risk factors
        - Smoking / EtOH cessation, treat hypertension (amlodipine safe in CHF patients per PRAISE trial), treat hyperlipidemia / metabolic syndrome
    - Symptomatic Pharmacotherapy
        - Loop diuretics are the backbone of symptomatic management; ideally these should be implemented in a weight-responsive regimen, though this requires a health literate and proactive patient
        - Digoxin shown to decrease hospitalizations, though not mortality. Subgroup analysis from DIG trial showed most benefit at lower serum levels (0.5-0.8 ng/ml), where the neurohormonal blockade effects predominate over the inotropic effects
        - IV iron (FAIR-HF) shown to improve NYHA functional class and quality of life in iron deficient patients (ferritin < 100 mcg/L or 100-299 with T-sat < 20%) even without frank anemia (included Hgb 9.5-12.5 g/dL)
    - Mortality-reducing Pharmacotherapy
        - Sympathetic Tone:
            - Beta blockade (Class I-IV)
                - Carvedilol (CAPRICORN) or metoprolol succinate (MERIT-HF); metoprol tartrate shown to be inferior in COMET. No head-to-head of carvedilol and long-acting metoprolol performed. Bisoprolol (CIBIS-II) also an option, though rarely used.
                - Monitor for bronchospasm if history of COPD / asthma
        - RAAS
            - ACE-I / ARB (Class I-IV; Cr > 2.5 usually excluded from trials)

        - Essentially equivalent, though ACE-I better studied (and overall preferred as first line in patients with no history of ACE-I cough or angioedema)
        - Combination is of unclear benefit; CHARM-ADDED showed a reduction in hospitalizations but VALIANT showed no benefit and an increase in adverse events. Per current AHA guidelines can be considered for patients with persistent symptoms despite otherwise maximal therapy.
    - Aldosterone blockade (Class III-IV, Cr < 2.5 in men or 2.0 in women, K < 5.0)
        - Spironolactone (RALES) and eplerenone (EPHESUS / EMPHASIS) both effective; eplerenone more expensive but associated with fewer side effects, e.g., gynecomastia / reduced libido
  - Vasoconstriction
    - ISDN / hydralazine (Class III-IV, self-reported African American) shown in A-HeFT significant reduction in both all-cause mortality and hospitalizations when added to a stable CHF regimen
  - Additional pharmacotherapies

- Device Therapy
  - ICD: Indicated in NYHA II-III patients at least 40 days out from any myocardial infarction with an EF < 35%, despite 3 months of therapy; 23% reduction in ACM at approximately 4 year mark in SCD-HeFT trial
  - Bi-V indications and outcomes
    - Indications: Per current AHA guidelines, recommended for patients with EF < 35% despite optimal medical therapy and a QRS duration >.12 seconds (though most benefit shown in those with >.15 seconds) with class III or ambulatory class IV symptoms. Notably, these guidelines do not reflect the most recent data (RAFT) which demonstrated a benefit in Class II symptoms; these patients likely should also be referred to electrophysiology.

- Prognostication
  - Useful calculator based on your patient's characteristics (also an iPhone version): http://depts.washington.edu/shfm/app.php
- HFNEF / HFPEF Caveat
  - Essentially all of the trials supporting the above interventions required a reduced EF in their inclusion criteria; we have markedly less data regarding patients with a normal or preserved EF. For further reading, see the below-referenced review by Drs. Bernard and Maurer, however, despite the comparative dearth of evidence current management revolves still revolves around beta blockade, RAAS inhibition, and management of comorbidities, particularly hypertension.
- Figures:

- HFNEF / HFREF PV loops
- CHF pathophysiology and pharmacology targets

Systolic HF (NEJM clinical practice) - http://www.nejm.org/doi/full/10.1056/NEJMcp0909392
HFPEF (Maurer / Bernard review) - http://www.ncbi.nlm.nih.gov/pubmed/22660923