Objectives

- Understand the impact of HF
- Understand current approach to diagnosis, management, and optimal care
- Understand current guidelines and recommendations
- Understand goals of therapy and current treatments
HF Statistics

• Prevalence: 6.6 million Americans (2010)
• Incidence: >700,000 new cases/year
• Responsible factors
  – older population
  – 10/1000 in those >65y/o
  – improved cardiac care
    • community defibrillators
    • coronary interventions, surgery

AHA Heart Disease, 2010
HF Mortality

• Causes or contributes to ~300,000 deaths/yr (USA)
  – Compared to 40,000 breast cancer
• In 2008, 1 in 9 death certificates mentioned HF
• 20% die in first year of diagnosis
  – 50% by 5 years
  – 80% by 10 years
HF Cost

- Most common cause of hospitalization in elderly
- >1 million admissions DRG 127
- >2 million secondary diagnosis admissions
- Avg LOS 5.6-8 days
- >15 million office visits
- Estimated direct and indirect cost of HF in the US for 2010: $39.2 billion
2010 HF Definition

• Syndrome caused by cardiac dysfunction, due to myocardial dysfunction
  – Neurohormonal and circulatory abnormalities
  – Pulmonary and systemic venous congestion and/or inadequate peripheral delivery
• Characteristic symptoms
• Usually progressive
  – Can be stabilized and dysfunction and remodeling may improve
**Definition of Heart Failure**

<table>
<thead>
<tr>
<th>Classification</th>
<th>Ejection Fraction</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Heart Failure with Reduced Ejection Fraction (HFrEF)</td>
<td>≤40%</td>
<td>Also referred to as systolic HF. Randomized clinical trials have mainly enrolled patients with HFrEF and it is only in these patients that efficacious therapies have been demonstrated to date.</td>
</tr>
<tr>
<td>II. Heart Failure with Preserved Ejection Fraction (HFpEF)</td>
<td>≥50%</td>
<td>Also referred to as diastolic HF. Several different criteria have been used to further define HFpEF. The diagnosis of HFpEF is challenging because it is largely one of excluding other potential noncardiac causes of symptoms suggestive of HF. To date, efficacious therapies have not been identified.</td>
</tr>
<tr>
<td>a. HFpEF, Borderline</td>
<td>41% to 49%</td>
<td>These patients fall into a borderline or intermediate group. Their characteristics, treatment patterns, and outcomes appear similar to those of patient with HFpEF.</td>
</tr>
<tr>
<td>b. HFpEF, Improved</td>
<td>&gt;40%</td>
<td>It has been recognized that a subset of patients with HFpEF previously had HFrEF. These patients with improvement or recovery in EF may be clinically distinct from those with persistently preserved or reduced EF. Further research is needed to better characterize these patients. Yancy et al. 2013</td>
</tr>
</tbody>
</table>
Systolic and Diastolic Heart Failure

**Normal Heart**
- Filling (Diastole): Right atrium flows into the right ventricle, which then fills up with blood.
- Ventricles relax and expand to fill with blood.

**Systolic Heart Failure**
- Right atrium flows into the right ventricle, which then fills up with blood.
- Enlarged ventricles fill with blood.

**Diastolic Heart Failure**
- Right atrium flows into the right ventricle, which then fills up with blood.
- Thickened and stiff ventricles fill with blood less than normal.

**Pumping (Systole)**
- Normal Heart: Ventricles contract and pump out between 50% and 60% of the blood.
- Systolic Heart Failure: Stretched ventricles are weaker, pumping out less blood than normal.
- Diastolic Heart Failure: Thickened ventricles contract normally, but have less blood to pump out.
Causes of Heart Failure

- CAD
- Valve disease
- Hypertension
- Congenital
- Toxic
- Cardiomyopathy
- Infection
- Myocarditis

Table 5: Causes of heart failure in population based studies

<table>
<thead>
<tr>
<th>Cause</th>
<th>Framingham heart study</th>
<th>Hillingdon heart failure study&lt;sup&gt;a&lt;/sup&gt;</th>
<th>Bromley heart failure study&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischaemic</td>
<td>59</td>
<td>36</td>
<td>52</td>
</tr>
<tr>
<td>Non-ischaemic:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>70</td>
<td>14</td>
<td>4</td>
</tr>
<tr>
<td>Valvular heart disease</td>
<td>22</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>5</td>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>Alcohol</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td>7</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Unknown</td>
<td>34</td>
<td>5</td>
<td>23</td>
</tr>
</tbody>
</table>

Because of rounding, the percentages do not always add up to 100. Framingham heart study: ischaemic heart disease and hypertension could be co-named as causing heart failure.
Types of Cardiomyopathy

- Cardiomyopathy-- impairment in heart function with pump failure and associated with ventricular dilatation and hypertrophy

- Primary cardiomyopathies
  - Genetic: hypertrophic, arrhythmogenic RV dysplasia, LV non-compaction
  - Mixed: dilated, restrictive
  - Acquired: myocarditis, stress (takotsubo), peripartum, tachycardia induced CM

- Secondary cardiomyopathies
  - Infiltrative (amyloidosis), hemachromatosis, toxicity (drugs, ETOH, chemotherapy), inflammatory (sarcoidosis, DM, hyperthyroidism, muscular dystrophy, autoimmune (lupus, scleroderma)
Dilated CM (DCM)

- Global enlargement
- Decreased pumping function
- EF< 40%
- Systolic dysfunction
Hypertrophic CM

- LV hypertrophy
  - early systolic
  - diastolic dysfunction
- Usually due to congenital defect
  - Most common 1:500 (AHA)
- Obstructive or non-obstructive
  - “IHSS”, “HOCM”
- SCD requiring ICD
Restrictive CM

- Least common CM
- Filling defects
  - Diastolic dysfunction
- Ventricle walls:
  - May thicken
  - Become stiff/rigid
  - Unable to relax

- Primary
  - Idiopathic
  - Loeffler’s CM
  - Endomyocardial fibrosis
- Secondary
  - Infiltrative
    - Amyloidosis
    - Sarcoidosis
    - S/p radiation
  - Storage
    - Hemochromatosis
    - Glycogen storage disease
HF Pathophysiology

- Heart Failure results in:
  - Increased cardiac workload
  - Decreased cardiac output
- Activation of compensatory mechanisms in an attempt to maintain normal cardiac pumping function
  - Hemodynamic changes
  - Neurohormonal response
  - Ventricular remodeling
Hemodynamic changes

- Frank-Starling law of the heart
  - Increased preload = increased contractility
  - Initially is a compensatory mechanism
- Normal ventricle can adjust
- Failing ventricle loses compensatory ability and cardiac output decreases
- Result: Pulmonary and systemic congestion
Neurohormonal Activation

- Primary myocardial injury

Secondary myocardial effects:
- LV remodelling
- Contractility
- Hypertrophy
- Apoptosis
- Cytokines
- Fibrosis
- NOS/ROS
- Electrophysiology

Neurohormones:
- ↑SNS activity
- ↑RAS
- ↑Endothelin
- ↑ANP/BNP
- ↑Cytokines

Endothelium:
- Vasoconstriction
- NOS/ROS
- Structural change
- Cytokines

CHF outcomes: sudden death, progressive pump failure, symptoms
Neurohormonal Response

- LV remodeling and cell hypertrophy
- Increased heart rate
- Vaso-constriction
- Sodium and water retention
- Cardiac cell death (apoptosis)
- Increased thirst
- Cardiac Cachexia

Detrimental Long Term Consequences
So now what.....
Goals of the Assessment of New Heart Failure

- Define symptoms, signs, and their severity
- Identify structural and functional abnormalities
- Determine etiology of HF if possible
- Evaluate risk for life-threatening arrhythmia
- Quantify the degree of functional limitation imposed by HF
- Guide therapy and tailor to individual goals of care
Steps in Evaluation of New HF

• History
• Physical Exam
• Diagnostic Labs and Tests
• Determine need for specialists
• Determine appropriate interval for f/u
The History is a Goldmine

• Define patient’s presentation
• Assess clinical severity of symptoms
• Seek clues to etiology
• Identify exacerbating factors for HF
• Identify comorbidities
• Identify barriers to adherence
Three common HF presentations

• Decreased effort/exercise tolerance
  – Dyspnea, fatigue
  – May be mistakenly attributed to aging, deconditioning, or other medical disorders

• Fluid retention
  – Leg, abdominal swelling reason for seeking attention
  – Other symptoms may be subtle

• Incidental finding
  – No symptoms
  – Symptoms of another cardiac or noncardiac disorder
  – Abnormal test
Symptoms Suggestive of HF

### Table 4.3. Symptoms Suggesting the Diagnosis of HF

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Less specific presentations of HF</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyspnea at rest or on exertion</td>
<td>Early satiety, nausea and vomiting, abdominal discomfort</td>
</tr>
<tr>
<td>Reduction in exercise capacity</td>
<td>Wheezing or cough</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>Unexplained fatigue</td>
</tr>
<tr>
<td>Paroxysmal nocturnal dyspnea (PND) or nocturnal cough</td>
<td>Confusion/delirium</td>
</tr>
<tr>
<td>Edema</td>
<td>Depression/weakness (especially in the elderly)</td>
</tr>
<tr>
<td>Ascites or scrotal edema</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Coughing
- Shortness of breath
- Pulmonary edema (excess fluid in lungs)
- Pumping action of the heart grows weaker
- Pleural effusion (excess fluid around lungs)
- Swelling in abdomen (ascites)
- Swelling in ankles and legs
Symptom History

- Which HF sx predominate?
- Duration of symptoms
- Symptoms/illness that preceded HF
- Define current limitations
Medication History

• Current medication regimen
• Past medications
• OTC medications or herbals, now or ever
• NSAIDs
• Weight loss medications
• Chemotherapy
Social History

• Identify behaviors that could worsen HF
  – Substance use, diet history, noncompliance

• Identify barriers to adherence
  – Behavioral
  – Socioeconomic
  – Cognitive/learning
  – Medical literacy
  – Cultural differences/Health beliefs
Family History

• Explore at least three generations, and children
• Cardiovascular diseases, diagnosed or undiagnosed
• Sudden death and Sudden cardiac death
• Patient’s history will target more questions
• Noncardiac familial diseases matter
Comorbid Medical Conditions

- Allergies
- Other cardiovascular diseases, or symptoms of them
- Other medical conditions, ever
- Surgeries
- XRT
Physical Exam
(ACC/AHA Class I)

• Routine vital signs
• Orthostatics
• Weight
• Height
• Calculate BMI
## Assessment for Signs of HF

### Table 4.4. Signs to Evaluate in Patients Suspected of Having HF

<table>
<thead>
<tr>
<th>Cardiac Abnormality</th>
<th>Sign</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated cardiac filling pressures and fluid overload</td>
<td>Elevated jugular venous pressure, S3 gallop, Rales, Hepatojugular reflux, Ascites, Edema</td>
</tr>
<tr>
<td>Cardiac enlargement</td>
<td>Laterally displaced or prominent apical impulse, Murmurs suggesting valvular dysfunction</td>
</tr>
<tr>
<td>Reduced cardiac output</td>
<td>Narrow pulse pressure, Cool extremities, Tachycardia with pulsus alternans</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td>Irregular pulse suggestive of atrial fibrillation or frequent ectopy</td>
</tr>
</tbody>
</table>
Other pertinent physical findings

- Thyroid abnormalities
- Lymphadenopathy
- Cachexia
- Jaundice
- Liver or spleen enlargement
- Signs of connective tissue disease
- Slow mentation
- Observation of respiration pattern
Diagnostic Labs and Tests

- Evaluate for reversible causes of HF
- Evaluate for exacerbating conditions
- Evaluate for comorbid conditions
- Suggest severity of heart failure
Diagnostic Labs
(ACC/AHA Class I)

- CBC, Electrolytes, BUN, Creatinine
- Urinalysis
- Fasting blood glucose, HbA1C
- Fasting lipid profile
- Liver function tests
- TSH
Diagnostic Labs
(ACC/AHA Class IIa)

- Ferritin (Hemachromatosis)
- HIV
- ANA (rheumatologic diseases)
- SPEP/UPEP (amyloidosis)
- Metanephrines (pheochromocytoma)
- BNP or nt-proBNP
- Viral Serologies
- Genetic Testing

* Other labs directed by clinical suspicion
Diagnostic Tests (ACC/AHA Class I)

- **EKG**
  - Arrhythmia, BBB, atrial abnormalities

- **PA/Lat CXR**
  - Cardiomegaly, edema, congestion, effusions, enlarged PA
Diagnostic Tests

• Assessment of LVEF (Class I)
  – Echocardiogram
  – Radionuclide study
  – Cardiac MRI

• Screen for cardiac ischemia (Class I)
  – History/physical
    • Angina or atypical chest pain
    • Claudication or physical signs of peripheral vascular disease
  – Noninvasive (IIb)
    • HF with LV dysfunction and no CP/ischemia
    • Radionuclide study
    • Cardiac CTA
  – Invasive
    • Angiogram

• Assess for sleep disordered breathing (Class IIa)
• Wearable rhythm monitor (Class IIb)
2D Echocardiogram with Doppler

- **Everyone** (Class I)
- No contraindications!
- Assess LVEF, cardiac chamber sizes, wall thickness, wall motion, diastolic function, valves
- Marginal at assessing RV size and function
Cardiac Catheterization

- Coronary Angiogram
  - HF + angina/ischemia (I)
  - HF + chest pain (IIa)
  - HF + known/suspected CAD (IIa)

- Right Heart Cath
  - Right/left filling pressures
  - Cardiac output
  - Pulmonary arterial pressure

- Other studies as indicated
  - Valve studies
  - Shunt studies
  - Evaluation for constriction/restriction
Cardiac MRI

- Delineates structure
  - Myocardium, pericardium, valves, chamber sizes, other intra/extracardiac structures
- Precise LV/RV function
- Wall motion
- Viability, inflammation, infiltration, and scar
- Characterize cardiomyopathy
- Contraindications:
  - Indwelling ferromagnetic material
  - GFR<30 (gadolineum)
Endomyocardial Biopsy

• Should be done in a center with expertise in technique and tissue handling/interpretation.

• Technique:
  – RIJ or Femoral Vein
  – Flexible bioptome
  – 5-10 samples from RV septum

Table 1. Risks Associated With Endomyocardial Biopsy in 546 Procedures

<table>
<thead>
<tr>
<th>Complication</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall 33 complications (6%)</td>
<td></td>
</tr>
<tr>
<td>Sheath insertion 15 (2.7%)</td>
<td></td>
</tr>
<tr>
<td>12 (2.0%) arterial puncture during local anesthesia</td>
<td></td>
</tr>
<tr>
<td>2 (0.4%) vasovagal reaction</td>
<td></td>
</tr>
<tr>
<td>1 (0.2%) prolonged venous oozing after sheath removal</td>
<td></td>
</tr>
<tr>
<td>Biopsy procedure 18 (3.3%)</td>
<td></td>
</tr>
<tr>
<td>6 (1.1%) arrhythmia</td>
<td></td>
</tr>
<tr>
<td>5 (1.0%) conduction abnormalities</td>
<td></td>
</tr>
<tr>
<td>4 (0.7%) possible perforation (pain)</td>
<td></td>
</tr>
<tr>
<td>3 (0.5%) definite perforation (pericardial fluid)</td>
<td></td>
</tr>
<tr>
<td>2 of 3 patients with definite perforation died</td>
<td></td>
</tr>
</tbody>
</table>

Data derived from Deckers et al (20).
Classification of HF Severity

- Clinical course
- Diagnostic information
- ACC/AHA HF staging system
- Risk scoring systems
- NYHA functional classification
- Objective assessment of functional status
  - 6-Minute Walk Test
  - Cardiopulmonary Exercise Test
Staging of Heart Failure

End Stage HF
Marked Sx at rest
Max Med Tx

Symptomatic HF
Structural Heart Disease
SOB, Fatigue
Exercise Intolerance

Asymptomatic HF
LV Systolic Dysfunction
Previous MI
Asymptomatic Valvular Disease

High Risk for Developing HF
HTN, CAD, DM
Family History of Cardiomyopathy
NYHA Functional Classification

- I: No limitation
- II: Mild symptoms, slight limitation with activity
- III: Marked symptoms and limitations with activity
- IV: Symptoms at rest
Cardiopulmonary Exercise Test (CPET, CMET, or CPX) (ACC/AHA Class IIa)

• Defines interaction of heart, lungs, cell metabolism, and circulation
• Gives functional and prognostic information
• Indications
  – Determine contribution of HF to exercise limitation
  – Identify high risk patients who are candidates for advanced cardiac therapies
Prognostic Value of CPX: Peak Oxygen Consumption

P<0.002 for pVO2<14 vs. >14
Heart Failure Risk Scoring Systems

- Heart Failure Survival Score

Table 1: The Heart Failure Survival Score

<table>
<thead>
<tr>
<th>Condition</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary artery disease (yes = 1, no = 0)</td>
<td>(……… × 0.6931) = +</td>
</tr>
<tr>
<td>Intraventricular conduction delay (yes = 1, no = 0)</td>
<td>(……… × 0.6083) = +</td>
</tr>
<tr>
<td>Left ventricular ejection fraction (%)</td>
<td>(……… × -0.0464) = +</td>
</tr>
<tr>
<td>Heart rate (b.p.m.)</td>
<td>(……… × 0.0216) = +</td>
</tr>
<tr>
<td>Na⁺ concentration (mmol/L)</td>
<td>(……… × -0.0470) = +</td>
</tr>
<tr>
<td>Mean arterial pressure (mmHg)</td>
<td>(……… × -0.0255) = +</td>
</tr>
<tr>
<td>Peak VO₂ (mL/min/kg)</td>
<td>(……… × -0.0546)</td>
</tr>
</tbody>
</table>

HFSS = ………

High risk < 7.19 (35%, 1-year survival), medium risk = 7.20-8.09 (60%, 1-year survival), and low risk > 8.10 (88%, 1-year survival).

- Seattle Heart Failure Model

http://meded.ucsd.edu/c clinicalmed/heart.htm
## Markers of Poor Prognosis

**Table 17 Conditions associated with a poor prognosis in heart failure**

<table>
<thead>
<tr>
<th>Demographics</th>
<th>Clinical</th>
<th>Electrophysiological</th>
<th>Functional/exertional</th>
<th>Laboratory</th>
<th>Imaging</th>
</tr>
</thead>
<tbody>
<tr>
<td>Advanced age*</td>
<td>Hypotension*</td>
<td>Tachycardia</td>
<td>Reduced work, low peak VO₂*</td>
<td>Marked elevation of BNP/NT-pro-BNP*</td>
<td>Low LVEF*</td>
</tr>
<tr>
<td>Ischaemic aetiology*</td>
<td>NYHA functional class III–IV*</td>
<td>Wide QRS*</td>
<td></td>
<td>Hyponatraemia*</td>
<td></td>
</tr>
<tr>
<td>Resuscitated sudden death*</td>
<td>Prior HF hospitalization*</td>
<td>LV hypertrophy</td>
<td>Elevated troponin*</td>
<td>Elevated biomarkers, neurohumoral activation*</td>
<td></td>
</tr>
<tr>
<td>Poor compliance</td>
<td>Tachycardia</td>
<td>Low heart rate variability</td>
<td>Poor 6 min walk distance</td>
<td>Elevated creatinine/BUN</td>
<td>Increased LV volumes</td>
</tr>
<tr>
<td>Renal dysfunction</td>
<td>Pulmonary rales</td>
<td>Atrial fibrillation</td>
<td>High VE/VO₂ slope</td>
<td>Elevated bilirubin Anaemia</td>
<td>Low cardiac index</td>
</tr>
<tr>
<td>Diabetes</td>
<td>Aortic stenosis</td>
<td>Low body mass index</td>
<td>Periodic breathing</td>
<td>Elevated uric acid</td>
<td></td>
</tr>
<tr>
<td>Anaemia</td>
<td></td>
<td></td>
<td></td>
<td>High LV filling pressure</td>
<td></td>
</tr>
<tr>
<td>COPD</td>
<td>Sleep-related breathing disorders</td>
<td></td>
<td></td>
<td>Restrictive mitral filling pattern, pulmonary hypertension</td>
<td></td>
</tr>
<tr>
<td>Depression</td>
<td></td>
<td></td>
<td></td>
<td>Impaired right ventricular function</td>
<td></td>
</tr>
</tbody>
</table>

* = powerful predictors.
Your Assessment Will Guide The Therapy
Standard Treatment

- Pharmacologic therapy
- Non-pharmacologic therapy
- Device therapy
Treatment Goals

• Improve survival
• Improve QOL
• Improve, recognize and manage sx
• Avoid precipitating factors
• Decrease hospital stays
Pharmacological Therapy

- ACE Inhibitors or Angiotensin Receptor Blockers (ARB’s)
- Beta-blockers
- Diuretics for volume
- Digoxin
- Aldosterone antagonists
- Additional vasodilators
ACE-Inhibitors

• High risk and LVEF<40% with symptoms or not
• Blocks Angiotensin I from converting to angiotensin II
• Start small doses and titrate when euvoletic to wet

• Reduce total mortality rate by 23%
• Lower risk of death and lower risk for hospitalization for HF

• Side effects:
  – Cough—dry, hacky, related to bradykinin not broken down by angiotensin II
  – Angioedema
  – Hyperkalemia– less angiotensin II → less aldosterone
  – Hypotension
  – Renal insufficiency

• Contraindications
  – Pregnancy
  – Cr > 3.0
  – Renal stenosis
  – Hyperkalemia
Guidelines: ARB

• Alternative to ACE-I
• Cough less common
  – Angiotensin II forms and breaks down bradykinin then blocked at the receptor
• If not tolerated: Nitrate/Hydralazine combination
Guidelines: β-blocker

- High risk post MI, LVEF <= 40% with symptoms, asymptomatic (expert opinion)
- Block sympathetic response
- Reverse cardiac remodeling
- Improve EF
- Reduce all cause mortality by ~32%

- Start when euvolemic on stable dose of ACE-I and diuretics
- Start low and increase every 2 weeks
- Monitor HR and BP
- Monitor daily weigh and signs of congestion
β-blockers

• Caution
  – Diabetes with recurrent hypoglycemia
  – HR <55-60/min, second or third degree HB
  – Hypotension, SPB < 80-5mm Hg

• Avoid
  – Asthma with active bronchospasm
  – Initiation/dose increase when fluid overloaded
  – Abrupt discontinuation, reflex tachycardia
Guidelines: Aldosterone Antagonists

• Functional class III-IV symptoms
  – On maximal HF therapy

• Side effects
  – Hyperkalemia
  – Impotence/ menstrual changes
  – Gynecomastia
    • Eplerenone: more aldosterone selective

• Contraindicated
  – Cr> 2.5 (or >2 in women)
  – K> 5.0

• Check K and renal function at 3 days or 1 week, monthly x3 and q 3mo
Other HF meds.....

• Additional vasodilators
  – Hydralazine
    • Initial 37.5 qid, target 75mg qid
  – Isosorbide dinitrate
    • Initial 20mg qid, target 40mg qid
• Digoxin: Aim for level < 1.0, interaction with Amiodarone
• Statins: decrease LDL
• Anticoagulation: Afib or thrombus
• Antiarrhythmics: Amiodarone and dofetilide
What about Diuretics.....
## Diuretics

<table>
<thead>
<tr>
<th>Diuretic</th>
<th>Dosage (mg/day)</th>
<th>Onset of Action</th>
<th>Action Duration</th>
<th>Peak Oral Effect (Hours)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Oral (Hours)</td>
<td>IV (Minutes)</td>
<td>Oral (Hours)</td>
<td>IV (Hours)</td>
</tr>
<tr>
<td><strong>Ascending Loop of Henle</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>40-400</td>
<td>1</td>
<td>5</td>
<td>6</td>
<td>2-3</td>
</tr>
<tr>
<td>Bumetanide</td>
<td>1-5</td>
<td>0.5</td>
<td>5</td>
<td>6</td>
<td>2-3</td>
</tr>
<tr>
<td>Torsemide</td>
<td>10-200</td>
<td>1</td>
<td>10</td>
<td>6-8</td>
<td>6-8</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Early Distal Tubule</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Metolazone</td>
<td>2.5-20</td>
<td>1</td>
<td>—</td>
<td>12-24</td>
<td>2-4</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>High potential for K+ loss</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>25-100</td>
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<td>—</td>
<td>12</td>
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<td>Ineffective if GFR &lt;30</td>
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<tr>
<td>Chlorothiazide</td>
<td>500-1000</td>
<td>1</td>
<td>15-30</td>
<td>8</td>
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<td>Ineffective if GFR &lt;30</td>
</tr>
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</table>

Mann, D. *Heart Failure 2nd ed.* 2011
Diuretic Risks

- Hypokalemia
  - Arrhythmia
  - Muscle aches
- Bicarbonate reabsorption $\rightarrow$ metabolic alkalosis
- Decreased serum uric acid excretion $\rightarrow$ gout
- Hyponatremia
- Further neurohormonal activation
- Stop diuretics when possible......
Consider Admission for…..

- Signs and symptoms of pulmonary or systemic congestion
  - Even in the absence of weight gain
- Major electrolyte disturbance
- Associated comorbid conditions
- Pneumonia
- Pulmonary embolus
- Diabetic ketoacidosis
- Symptoms suggestive of transient ischemic accident or stroke
- Repeated ICD firings
- Previously undiagnosed HF with signs and symptoms of systemic or pulmonary congestion
Advanced HF Therapy

- Inotropes
- Implantable Cardiac Defibrillators
- Bi-ventricular pacemakers
- Left Ventricular Assist Devices
- Cardiac Transplant
- Investigational medicine and devices
- Palliative medicine consult/hospice at EOL
Inotropic Therapy

• Milrinone or Dobutamine
  – Routine scheduled infusions
    • Evidence does not support efficacy
  – Continuous infusions
    • Can lead to increased mortality
      (Hunt et al., 2005)

• Medicare requirements
  – Failure of maximum oral therapy
  – 20% improvement of cardiac index or wedge pressure
  – Symptoms at rest
  – Completion of Medicare form
    (Option Care, 2005)
Balancing the Effects of Inotropes

• Benefits
  – Increase cardiac contractility
  – Dilate peripheral vasculature
  – Increase C.O. and C.I.

• Negative Side Effects
  – ↑ myocardial O2 consumption
  – Hypotension
  – Tachycardia
  – Arrhythmias
  – Worsening HF
Device Therapy- Rhythm Support

• Pacemakers
  – For bradyarrhythmias or after ablation

• Implantable Cardiac Defibrillators
  – Reduces mortality in both ischemic and non-ischemic patients

• Bi-ventricular pacemakers or defibrillators
  – Reduced mortality compared to medical management
  – Improves functional class, 6-minute walking distance, quality of life, maximum oxygen consumption, exercise time, ejection fraction, and LV diastolic and systolic diameters after 6 months
    (Wilkoff, 2005)
HF Disease Trajectory

Goodlin SJ, J Card Fail. 2004;10(3).
Non-pharmacologic Therapy

- Patient education
- Home monitoring
- Symptom management
- Dietary
- Lifestyle modifications
- Exercise
General Education

• Psychological responses
  – Depression and anxiety common
  – Treatment is necessary if persists
• Immunizations needed
  – Flu and pneumococcal vaccines
• Prognosis
• Goals of care: early and repeated
• Advanced directives
Conclusions

• HF remains a leading cause of morbidity and mortality.

• Multiple etiologies but all lead to neurohormonal activation. Therapies are aimed at the etiology of HF and the modulation of the neurohormonal axis.

• Treatment goals are individualized and can incorporate pharmacologic, surgical, mechanical options.

• Goals of care and advanced directives are key interventions.