Diagnosis and Management of Heart Failure
Northeast Regional Nurse Practitioner Conference 2016

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DISCLOSURES

None of the planners or presenters of this session have disclosed any conflict or commercial interest.
Diagnosis and Management of Heart Failure

OBJECTIVES:

1. Understand the epidemiology, diagnosis and pathophysiology of heart failure.

2. Describe current management including symptom recognition, medications, diet, exercise, and device based therapy.
Outline

- Definitions and scope of problem
- Diagnosing and classifying heart failure
- Approach to management of CHF
  - Oral drug therapy (ACE-I, ARB, betablockers, aldosterone blockade, digoxin) and newer treatments
  - Device therapy
    - ICD’s / Biventricular (BiV) pacers
    - Cardiac support systems
- Future directions and exciting developments
  - Remote PA sensor monitoring
What is Congestive Heart Failure?

- An inability of the heart to meet the metabolic demands of the body
- Etiology may be *either* systolic (i.e. squeezing) or diastolic (i.e. relaxing) dysfunction
- Can be categorized as forward or backward ventricular failure. Forward or LV failure is secondary to reduced forward flow into the aorta. Backward or RV failure is due to elevated systemic venous pressure
Types of Heart Failure

- **Systolic (or squeezing) heart failure**
  - HFrEF
  - Decreased pumping function of the heart, which results in fluid back up in the lungs and heart failure
  - Ejection Fraction (EF) 40% or less

- **Diastolic (or relaxation) heart failure**
  - HFpEF
  - Involves a thickened and stiff heart muscle
  - As a result, the heart does not fill with blood properly
  - This results in fluid backup in the lungs and heart failure
  - EF 40% or greater

*Low normal EF 40-50%*
US population with HF by 2030

Table 1

Projections of the US Population With HF From 2010 to 2030 for Different Age Groups

<table>
<thead>
<tr>
<th>Year</th>
<th>All</th>
<th>18–44 y</th>
<th>45–64 y</th>
<th>65–79 y</th>
<th>≥80 y</th>
</tr>
</thead>
<tbody>
<tr>
<td>2012</td>
<td>5,813,262</td>
<td>396,578</td>
<td>1,907,141</td>
<td>2,192,233</td>
<td>1,317,310</td>
</tr>
<tr>
<td>2015</td>
<td>6,190,606</td>
<td>402,926</td>
<td>1,949,669</td>
<td>2,483,853</td>
<td>1,354,158</td>
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<tr>
<td>2020</td>
<td>6,859,623</td>
<td>417,600</td>
<td>1,974,585</td>
<td>3,004,002</td>
<td>1,463,436</td>
</tr>
<tr>
<td>2025</td>
<td>7,644,674</td>
<td>434,635</td>
<td>1,969,852</td>
<td>3,526,347</td>
<td>1,713,840</td>
</tr>
<tr>
<td>2030</td>
<td>8,489,428</td>
<td>450,275</td>
<td>2,000,896</td>
<td>3,857,729</td>
<td>2,180,528</td>
</tr>
</tbody>
</table>

HF indicates heart failure.
Projected prevalence of HF from 2012 to 2030 is shown for men and women in the United States.

The table below depicts projections of the total cost of care in billions of dollars for HF for different age groups of the United States population:

<table>
<thead>
<tr>
<th>Year</th>
<th>All</th>
<th>18-44 Years</th>
<th>45-54 Years</th>
<th>65-79 Years</th>
<th>≥80 Years</th>
</tr>
</thead>
<tbody>
<tr>
<td>2012</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical</td>
<td>20.90</td>
<td>0.33</td>
<td>3.67</td>
<td>8.46</td>
<td>8.42</td>
</tr>
<tr>
<td>Indirect: Morbidity</td>
<td>5.42</td>
<td>0.52</td>
<td>1.92</td>
<td>2.05</td>
<td>0.93</td>
</tr>
<tr>
<td>Indirect: Mortality</td>
<td>4.35</td>
<td>0.66</td>
<td>2.53</td>
<td>0.98</td>
<td>0.18</td>
</tr>
<tr>
<td>Total</td>
<td>30.70</td>
<td>1.51</td>
<td>8.12</td>
<td>11.50</td>
<td>9.53</td>
</tr>
<tr>
<td>2020</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical</td>
<td>31.10</td>
<td>0.43</td>
<td>4.58</td>
<td>14.20</td>
<td>11.80</td>
</tr>
<tr>
<td>Indirect: Morbidity</td>
<td>7.09</td>
<td>0.66</td>
<td>2.20</td>
<td>3.11</td>
<td>1.12</td>
</tr>
<tr>
<td>Indirect: Mortality</td>
<td>5.30</td>
<td>0.79</td>
<td>2.89</td>
<td>1.49</td>
<td>0.22</td>
</tr>
<tr>
<td>Total</td>
<td>43.60</td>
<td>1.88</td>
<td>9.67</td>
<td>18.80</td>
<td>13.20</td>
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<tr>
<td>2030</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medical</td>
<td>53.10</td>
<td>0.59</td>
<td>5.86</td>
<td>23.30</td>
<td>23.40</td>
</tr>
<tr>
<td>Indirect: Morbidity</td>
<td>9.80</td>
<td>0.91</td>
<td>2.54</td>
<td>4.48</td>
<td>1.87</td>
</tr>
<tr>
<td>Indirect: Mortality</td>
<td>6.94</td>
<td>0.98</td>
<td>3.32</td>
<td>2.16</td>
<td>0.37</td>
</tr>
<tr>
<td>Total</td>
<td>69.70</td>
<td>2.48</td>
<td>11.70</td>
<td>29.90</td>
<td>25.60</td>
</tr>
</tbody>
</table>
The projected increase in direct and indirect costs attributable to HF from 2012 to 2030.

Epidemiology of Heart Failure in the US

- More deaths from heart failure than from all forms of cancer combined
- 550,000 new cases/year
- 4.7 million symptomatic patients; estimated 10 million in 2037

Heart Failure By State

Heart Failure Death Rates, 2011-2013
Adults, Ages 35+, by County

Rates are spatially smoothed to enhance the stability of rates in counties with small populations.

Data Source:
National Vital Statistics System
National Center for Health Statistics

CDC

Age-Adjusted Average Annual Rates per 100,000
- 54.6 - 154.5
- 154.6 - 178.9
- 177.0 - 198.0
- 198.1 - 227.9
- 228.0 - 709.2
- Insufficient Data
Population at Risk

Those who have or are at risk for:

- Ischemic Heart Disease
  - Diabetics, obesity, dyslipidemia, smokers
- Hypertension
- Infections e.g. viral myocarditis
- Issues with substance abuse or those receiving cytotoxic drugs (chemo)
- Valvular Disease
- Prolonged Arrhythmias
Downhill Cascade in Heart Failure

- Myocardial Insult
- Myocardial Dysfunction
- Reduced System Perfusion
- Inflammation
- Hemodynamic Defense Systems
- Altered Gene Expression
- Apoptosis
- Remodeling
Left Ventricular Dysfunction
Systolic and Diastolic

**Symptoms**
- Dyspnea on Exertion
- Paroxysmal Nocturnal Dyspnea
- Tachycardia
- Cough
- Hemoptysis

**Physical Signs**
- Basilar Rales
- Pulmonary Edema
- S3 Gallop
- Pleural Effusion
- Cheyne-Stokes Respiration
# Right Ventricular Failure

Systolic and Diastolic

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Physical Signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abdominal Pain</td>
<td>Peripheral Edema</td>
</tr>
<tr>
<td>Anorexia</td>
<td>Jugular Venous Distention</td>
</tr>
<tr>
<td>Nausea</td>
<td>Abdominal-Jugular Reflux</td>
</tr>
<tr>
<td>Bloating</td>
<td>Hepatomegaly</td>
</tr>
</tbody>
</table>
Peripheral Edema
JVP
Blood Flow Review
(Importance of knowing how the system works)
Lets go with the Flow......
Diagnostic Evaluation of New Onset Heart Failure

- Determine the type of cardiac dysfunction (systolic vs. diastolic)
- Determine Etiology
- Define prognosis
- Guide therapy
Diagnosis Evaluation of New Onset Heart Failure

Initial Work-up:

- ECG
- Chest x-ray
- Blood work
- Echocardiography
The key findings of **cardiogenic pulmonary edema**

- **Kerley B lines (septal lines)**
  - Seen at the lung bases, usually no more than 1 mm thick and 1 cm long, perpendicular to the pleural surface.

- **Pleural effusions**
  - Usually bilateral, frequently the right side being larger than the left.
  - If unilateral, more often on the right.

- **Fluid in the fissures**
  - Thickening of the major or minor fissure.

- **Peribronchial cuffing**
  - Visualization of small doughnut-shaped rings representing fluid in thickened bronchial walls occur. In addition, radiographic findings frequently persist for several days despite clinical recovery.

- Radiographic findings can lag behind physiologic changes.
A - Alveolar edema (bat's wing)
B - Kerley B line
C - Cardiomegaly
D - Dilated prominent upper lobe vessels
E - Pleural Effusion
BNP

- Elevated in accordance with severity of heart failure
- High negative predictive values
<table>
<thead>
<tr>
<th>CARDIAC ABNORMALITY</th>
<th>SIGN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase cardiac filling pressures and volume overload</td>
<td>▪ S3 Gallop</td>
</tr>
<tr>
<td></td>
<td>▪ Rales</td>
</tr>
<tr>
<td></td>
<td>▪ HJR</td>
</tr>
<tr>
<td></td>
<td>▪ Elevated JVD</td>
</tr>
<tr>
<td></td>
<td>▪ Ascites and edema</td>
</tr>
<tr>
<td>Cardiac Enlargement</td>
<td>▪ Murmurs suggesting valvular dysfunction</td>
</tr>
<tr>
<td></td>
<td>▪ Laterally displaced apical pulse</td>
</tr>
</tbody>
</table>
Mitral Regurgitation
SYMPTOMS OF HEART FAILURE

- Edema of the bronchial mucosa
- Pulmonary edema
- Cardiomegaly
- Edema in other body parts
- Tachycardia
- Coughing
- Dyspnea
- Fatigue
Symptoms: The Tip of the Congestion Iceberg in Heart Failure

- Systemic congestion (JVD, edema)
  - ↑ RV + RA pressure
  - Increase PA pressure
    - Increased PCWP (congestion)
      - ↑ LA and LV diastolic pressure
        - ↑ LVDP + Impaired volume regulation
          - Abnormal LV function (Sys and/or Dia)

- Abnormal lung function
  - Respiratory muscle dysfunction
  - Other factors

- Dyspnea
  - Alveolar edema
    - Redistribution in pulmonary vascular bed + Interstitial edema
  - Mitral Regurgitation

Gheorghiade et al. EJHF 2010
Classification of Heart Failure
<table>
<thead>
<tr>
<th>Class</th>
<th>Patient Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No limitation of physical activity</td>
</tr>
<tr>
<td></td>
<td>No undue fatigue, palpitation or dyspnea</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation of physical activity</td>
</tr>
<tr>
<td></td>
<td>Comfortable at rest</td>
</tr>
<tr>
<td></td>
<td>Less than ordinary activity results in fatigue, palpitation, or dyspnea</td>
</tr>
<tr>
<td>III</td>
<td>Marked limitation of physical activity</td>
</tr>
<tr>
<td></td>
<td>Comfortable at rest</td>
</tr>
<tr>
<td></td>
<td>Less than ordinary activity results in fatigue, palpitation, or dyspnea</td>
</tr>
<tr>
<td>IV</td>
<td>Unable to carry out any physical activity without discomfort</td>
</tr>
<tr>
<td></td>
<td>Symptoms of cardiac insufficiency at rest</td>
</tr>
<tr>
<td></td>
<td>Physical activity causes increased discomfort</td>
</tr>
</tbody>
</table>

Criteria Committee of the New York Heart Association, 1964.
Stages of Heart Failure

- Designed to emphasize preventability of HF
- Designed to recognize the progressive nature of LV dysfunction
Stages of Heart Failure

A
• No structural heart disease
• At risk population- HTN, DM, Cytotoxic drugs, Drug or Alcohol over use.

B
• Minimal structural heart disease
• No symptoms or signs of HF

C
• Moderately severe structural heart disease
• Previous or current symptoms

D
• Severe structural heart disease
• Refractory symptoms requiring special RX
Current Treatment of Heart Failure
Sympathetic Nervous System Response

Neurohormonal Activation in Heart Failure

Myocardial injury to the heart (CAD, HTN, CMP, Valvular disease)

Initial fall in LV performance, ↑ wall stress

Activation of RAAS and SNS

Remodeling and progressive worsening of LV function

Fibrosis, apoptosis, hypertrophy, cellular/molecular alterations, myotoxicity

Peripheral vasoconstriction
Hemodynamic alterations

Heart failure symptoms
Fatigue
Activity altered
Chest congestion
Edema
Shortness of breath

Morbidity and mortality
Arrhythmias
Pump failure

RAS, renin-angiotensin system; SNS, sympathetic nervous system.
Classification of Drugs to Treat Heart Failure

- Drugs that improve the force of cardiac contractility (positive inotrope)
- Drugs that improve compensatory stresses
Drugs that improve the force of cardiac contractility (positive inotrope)

- Cardiac glycosides (digoxin,)
- Phosphodiesterase inhibitors (Milrinone, Primacor)
- Beta I agonists (dobutamine)
Drugs that improve compensatory stresses upon cardiac performance

- Diuretics (Loop, Thaizides)
- Vasodilators (Hydralazine, isosorbide mononitrate)
- ACE Inhibitors (captopril, lisinopril)
- Angiotensin II receptor blockers (losartan, candesartan, valsartan)
- Beta-blockers (metoprolol succinate, carvedilol)
Standard Pharmacologic Therapy

- Beta Blockers
- Ace Inhibitors
- ARB’s
- Diuretics- For symptomatic relief
- Aldosterone Inhibitors
- Digoxin
Paradigm for Outpatient Management of Heart Failure with reduced LVEF

- **Control Volume**
  - Diuretic

- **Slow disease progression**
  - ACE-Inhibition + \( {\beta} \)-blockade
  - and/or ARB
  - Aldosterone Antagonists

- **Treat residual symptoms**
  - Digoxin
Inhibit the adverse effects of the sympathetic nervous system by:

- ↓ ventricular volumes and pressures by causing peripheral vasodilation
- ↓ norepinephrine release
- ↓ arrhythmias
- ↓ heart rate
3 Beta Blockers effective in chronic heart failure

- Bisoprolol
- Toprol XL (metoprolol succinate)
  Both of these drugs selectively block beta 1 receptors
- Carvedilol (Coreg)
  blocks alpha 1, beta 1 and beta 2 receptors.
Pharmacologic Management

**Beta-Blockers**

- Cardioprotective effects due to blockade of excessive SNS stimulation
- In the short-term, beta blocker decreases myocardial contractility; increase in EF after 1-3 months of use
- Long-term, placebo-controlled trials have shown symptomatic improvement in patients treated with certain beta-blockers
- When combined with conventional HF therapy, beta-blockers reduce the combined risk of morbidity and mortality, or disease progression

ACE INHIBITORS

- ↓ It and rt filling pressures
- ↓ MAP
- ↓ rt and lt end diastolic volume
- ↓ SVR
- ↓ LV remodeling
- ↓ fibrosis
- ↓ LV dilation
ACE Inhibitors

- Used in symptomatic and asymptomatic patients with a reduced EF <40%
- Interfere with the RAS by inhibiting the enzyme that is responsible for the conversion of angiotensin I to angiotensin II
- Stabilize LV remodeling, improve patient symptoms, prevent hospitalization, and prolong life
- Abrupt withdrawal avoided in the absence of life-threatening complications (e.g., angioedema, hyperkalemia).
- Side effects: decrease blood pressure, mild azotemia, nonproductive cough (10% to 15% of patients) and angioedema (1% of patients) hyperkalemia
Angiotensin Receptor Blockers (ARBs)

- Block $\text{AT}_1$ receptors, which bind circulating angiotensin II
- Examples: valsartan, candesartan, losartan
- Should not be considered equivalent or superior to ACE inhibitors
- Can be used in conjunction with ACE or in place of ACE in those who are intolerant.
Angiotensin Receptor Blockers

- Symptomatic and asymptomatic patients with an EF less than 40% who are ACE-intolerant for reasons other than hyperkalemia or renal insufficiency

- ARBs block the effects of angiotensin II on the angiotensin type 1 receptor, the receptor subtype responsible for almost all the adverse biologic effects relevant to angiotensin II on cardiac remodeling

- Side effects: hypotension, azotemia, and hyperkalemia
Entresto® - sacubitril/valsartan

- Indications:
  - Neprilysin inhibitor and angiotensin II receptor blocker combination to reduce the risk of cardiovascular death and hospitalization for heart failure in patients with chronic heart failure (NYHA Class II-IV) and reduced ejection fraction
  - Place in therapy:
    - Patient who have progressed in severity of their heart failure on optimum ACE inhibitor therapy
Entresto® - sacubitril/valsartan

• **Indications:**
  
  • Neprilysin inhibitor and angiotensin II receptor blocker combination to reduce the risk of cardiovascular death and hospitalization for heart failure in patients with chronic heart failure (NYHA Class II-IV) and reduced ejection fraction

• **Place in therapy:**
  
  • Patient who have progressed in severity of their heart failure on optimum ACE inhibitor therapy
Entresto® - sacubitril/valsartan

Drug Facts

- Pharmacology:
  - Sacubitril - inhibits neprilysin
    - Neprilisyn – neutral endopeptidase
    - Leads to increase in level of peptides, including natriuretic peptides
  - Valsartan – blocks the angiotensin II type-1 (AT1) receptor
Entresto® - sacubitril/valsartan

Prescription Information

• If switching from ACE-I to Entresto, 36 hour washout period is recommended

• Cost – Source: NY Times; Accessed 8/21/15
  • $4,500/year
  • Novartis offers free 30-day supply and $10 co-pay cards
Entresto® - sacubitril/valsartan

Literature Review

• Conclusions
  • Entresto’s dual inhibition was more effective in reducing the risk of death from cardiovascular causes or hospitalization for HF than ACE inhibition with enalapril
  • The only significant side effect was symptomatic hypotension, though this did not increase the rate of discontinuation

Indications:

- To reduce the risk of hospitalization for worsening heart failure in patients with stable, symptomatic chronic heart failure with left ventricular ejection fraction ≤ 35%, who are in sinus rhythm with resting heart rate ≥ 70 beats per minute and either are on maximally tolerated doses of beta-blockers or have a contraindication to beta-blocker use.
Corlanor® - Ivabradine

Clinical Application

• **Contraindications:**
  - Acute decompensated heart failure
  - Blood pressure less than 90/50 mmHg
  - Sick sinus syndrome, sinoatrial block or 3rd degree AV block, unless a functioning demand pacemaker is present
  - Resting heart rate less than 60 bpm prior to treatment
  - Severe hepatic impairment
  - Pacemaker dependence (heart rate maintained exclusively by the pacemaker)
Corlanor® - Ivabradine

Prescription Information

**Recommended starting dose**

<table>
<thead>
<tr>
<th>Dose Option</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>5mg tablet</td>
<td>5mg tablet by mouth twice daily with meals</td>
</tr>
<tr>
<td>or</td>
<td>2.5mg tablet by mouth twice daily for patients in whom bradycardia could lead to hemodynamic compromise or with a history of conduction defects</td>
</tr>
</tbody>
</table>

**After 2 weeks, check resting heart rate**

<table>
<thead>
<tr>
<th>Heart Rate</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt;60 bpm</td>
<td>Increase dose by 2.5mg twice daily up to max of 7.5mg twice daily</td>
</tr>
<tr>
<td>50-60 bpm</td>
<td>Maintain dose</td>
</tr>
<tr>
<td>&lt;50 bpm</td>
<td>Decrease dose by 2.5mg twice daily *Discontinue therapy if current dose is 2.5mg twice daily</td>
</tr>
</tbody>
</table>
Corlanor® - Ivabradine

Prescription Information

- **Cost:**
  - $450 (AWP) for 30 day supply (60 tabs) of either 5mg or 7.5mg (McKesson)
Corlanor® - Ivabradine

Conclusion

- These results show that ivabradine substantially and significantly reduced major risks associated with heart failure when added to standard of care treatment.

- In patients treated with ivabradine, the relative risk of the primary endpoint fell by 18% compared to placebo.

- Heart rate modulation plays an important part in the pathophysiology of heart failure and the progression of the disease.

- These findings reflect the good tolerability of ivabradine in patients with chronic heart failure.

Corlanor® - Ivabradine

Summary

• First in-class Hyperpolarizing-activated Cyclic Nucleotide (HCN) channel blocker that lowers heart rate, indicated for patients with stable chronic HFref and heart rate ≥ 70 bpm

• Use in patients who are already on maximum tolerated dose of beta-blockers or are unable to use beta blockers

• Ivabradine reduces heart rate without reducing the heart’s contractility (no negative inotropic effects)

• No clinical benefit in the treatment of atrial fibrillation

• Potential drug interactions w/ CYP3A4 inhibitors/inducers

• Bradycardia is the most common adverse effect
Our Most Important Outpatient Tool
Diuretics

- Controls symptoms of fluid retention
- Facilitate the use of other drugs indicated for heart failure
- Patients can be taught to adjust their diuretic dose based on changes in body weight
- Electrolyte depletion a frequent complication
- Should never be used alone to treat heart failure
- Higher doses of diuretics are associated with increased mortality
**Loop Diuretics**

- Titrate to achieve dry weight
- ↓ Dose when fluid is controlled
- Combine to overcome resistance

<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Dose</th>
<th>Maximum Dose</th>
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</thead>
<tbody>
<tr>
<td>Bumetanide (Bumex)</td>
<td>0.5-1.0/12-24h</td>
<td>10mg/day</td>
</tr>
<tr>
<td>Furosemide (Lasix)</td>
<td>20-40/12-24h</td>
<td>400mg/day</td>
</tr>
<tr>
<td>Torsemide (Demadex)</td>
<td>10-20/12-24h</td>
<td>200mg/day</td>
</tr>
</tbody>
</table>

AHA/ACC HF Guidelines 2001
Thiazide Diuretics

Synergy between loop and DCT diuretic

- Metolazone po (2.5-10 mg) 20 minutes prior to loop diuretic
  - Long duration of action (days)
  - Follow K, volume status closely
  - Often limited to several days of rx; usually not used chronically
Aldosterone Antagonists

- Generally well-tolerated
- Shown to reduce heart failure-related morbidity and mortality
- Generally reserved for patients with NYHA Class III-IV HF
- Side effects include hyperkalemia and gynecomastia. Potassium and creatinine levels should be closely monitored
Diuretic effects on the Nephron
Diuretic Resistance

- When moderate doses of a loop diuretic do not achieve the desired reduction of the extracellular fluid volume
- Potential delay in their rate of absorption
- Post diuretic NaCl retention
- Loss of renal responsiveness to endogenous natriuretic peptides as HF advances
- Diuretics increase solute delivery to distal segments of the nephron, causing epithelial cells to undergo hypertrophy and hyperplasia
Management of Diuretic Resistance

- Treat the diuretic-resistant patient with administration of two classes of diuretic concurrently
  - I.e. Addition of Metolazone because its half-life is longer, works at a different level of the nephron.
- Change the type of diuretic i.e. Furosemide to Torsemide due to the bioavailability of the drug
- Transition to Intravenous Diuretics
Out Patient IV Diuretics

Weight gain and HF symptoms despite uptitration of oral diuretics seen in HF clinic. If ADHF confirmed and patient reports PND and signs of heart failure.

Lab review

Outpatient IV diuresis based on protocol

Outpatient follow up in 24 hours
Banana vs. K Supplement

To get the same amount of K from a banana that a 40 meq supplement provides, the patient would have to eat a banana that is 4 feet long!
**Digoxin**

- Enhances inotropy of cardiac muscle
- Reduces activation of SNS and RAAS
- Controlled trials have shown long-term digoxin therapy:
  - Reduces symptoms
  - Increases exercise tolerance
  - Improves hemodynamics
  - Decreases risk of HF progression
  - Reduces hospitalization rates for decompensated HF
  - Does not improve survival

**Beware of Digoxin toxicity**

- Depletion of serum potassium level
- Concomitant use of drugs
- Presence of renal failure
- Hypothyroidism
- Old age
Algorithm for Treatment of CHF

Diagnosis of HF confirmed

Assess for fluid retention

Fluid retention
- Diuretic

No fluid retention
- ACE inhibitor*
- Beta blocker

ICD if NYHA class II–III

CRT if NYHA class III–IV and QRS >120 ms

Persistent symptoms or special populations

*ARB if ACE-intolerant

ARB
- Aldosterone antagonist
- Hydralazine/isosorbide
- digoxin
Management of HF with a Preserved Ejection Fraction (>40–50%)

No proven therapy

- Treatment efforts should be focused on the underlying disease process (e.g., myocardial ischemia, hypertension).

- Precipitating factors such as tachycardia and AF should be treated.

- Dyspnea may be treated by reducing total blood volume (dietary sodium restriction and diuretics), decreasing central blood volume (nitrates), or blunting neurohormonal activation with ACE inhibitors, ARBs, and/or beta blockers.
New Therapy
The pulmonary artery pressure sensor is implanted via a right heart catheterization procedure via femoral vein approach.

Target location for pulmonary artery pressure sensor
CardioMEMS™ HF System

- Pulmonary Artery Pressure Sensor
- Patient Electronics System
- CardioMEMS™ HF System Website
What to do with those patients who are Refractory to Standard Medical Treatment

- **Device Therapy**
  - Internal Cardiac Defibrillators (ICD’s)
  - Bi-Ventricular Pacemakers

- **Cardiac Replacement Therapy:**
  - Ventricular Assist Device
  - Cardiac Transplantation

- **Home Inotropic Therapy**
  - Palliative Treatment: May Increase Mortality

- **Hospice**
# Sudden cardiac death (SCD)

## ICD

**High risk in heart failure patients**

- Heart failure patients experience SCD at six to nine times the rate of the general population.

- Sudden death is the predominant mode of death in mild to moderate heart failure.

## CRT-D

- Patients who are at high risk of sudden cardiac death due to ventricular arrhythmias.

- Moderate to severe heart failure, NYHA Class III/IV.

- Left ventricular dysfunction, EF ≤ 35%.

- QRS duration ≥ 120 ms.

- Symptomatic despite stable, optimal heart failure drug therapy.

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CRT/ICD

Inside

Out
Current Generation Left Ventricular Assist
The HeartMate II®

- A surgically implanted, rotary continuous-flow device in parallel with the native left ventricle
  - Left ventricle to ascending aorta
  - Percutaneous driveline
- Electrically powered
  - Batteries and line power
- Fixed speed operating mode
- Home discharge
Left Ventricular Assist Devices
Worldwide Heart Transplants
Post Transplant Survival

As of June 5, 2009:

Survival rates

1 year : 88.0% (males), 86.2% (females)

3 years: 79.3% (males), 77.2% (females)

5 years: 73.2% (males), 69.0% (females)

April 3 2012
The Future?
Take Home Points

- Heart failure is a chronic, progressive disease that is generally not curable, but treatable.

- Most recent guidelines promote lifestyle modifications and medical management with ACE inhibitors, beta blockers, digoxin, aldosterone antagonists and diuretics.

- Close follow-up of the heart failure patient is essential, with necessary adjustments in medical management. This is where a specialized Cardiomyopathy Clinic plays an important role.
Thank You!

THINK SUMMER!!!!