Heart Failure Management

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Financial Disclosures

None
Outline

• Definition and classification of heart failure

• Pathophysiology of CHF

• Approach to the management of CHF
  • Drug therapy
  • Monitoring and Guiding Therapy

• New Developments
ACCF/AHA Practice Guideline

2013 ACCF/AHA Guideline for the Management of Heart Failure
A Report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines
## Types of Heart Failure

<table>
<thead>
<tr>
<th>Classification</th>
<th>EF (%)</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 controlled 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 patients 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.</td>
</tr>
</tbody>
</table>

Stages of Heart Failure

**At Risk for Heart Failure**

**STAGE A**
At high risk for HF but without structural heart disease or symptoms of HF

- e.g., Patients with:
  - HTN
  - Atherosclerotic disease
  - DM
  - Obesity
  - Metabolic syndrome
  - Patients Using cardiotoxins
  - With family history of cardiomyopathy

**STAGE B**
Structural heart disease but without signs or symptoms of HF

- e.g., Patients with:
  - Previous MI
  - LV remodeling including LVH and low EF
  - Asymptomatic valvular disease

**STAGE C**
Structural heart disease with prior or current symptoms of HF

- e.g., Patients with:
  - Known structural heart disease and HF signs and symptoms

**STAGE D**
Refractory HF

- e.g., Patients with:
  - Marked HF symptoms at rest
  - Recurrent hospitalizations despite GDMT

**Heart Failure**

**THERAPY**

**Goals**
- Control symptoms
- Improve HRQOL
- Prevent hospitalization
- Prevent mortality

**Strategies**
- Identification of comorbidities
- Treatment

- Diuresis to relieve symptoms of congestion
- Follow guideline driven indications for comorbidities, e.g., HTN, AF, CAD, DM
- Revascularization or valvular surgery as appropriate

**Patients with**:
- Known structural heart disease and HF signs and symptoms
- Asymptomatic valvular disease
- LV remodeling including LVH and low EF
- Patients Using cardiotoxins
- With family history of cardiomyopathy

**Heart healthy lifestyle**
- Prevent vascular, coronary disease
- Prevent LV structural abnormalities

**Drugs**
- ACEI or ARB as appropriate
- Beta blockers as appropriate
- Aldosterone antagonists
- Hydralazine/isosorbide dinitrate
- ACEI and ARB
- Digoxin
- CRT
- ICD
- Revascularization or valvular surgery as appropriate

**Development of symptoms of HF**

**Options**
- Advanced care measures
- Heart transplant
- Chronic inotropes
- Temporary or permanent MCS
- Experimental surgery or drugs
- Palliative care and hospice
- ICD deactivation

## Comparison Between ACC/AHA HF Stage and NYHA Functional Class

<table>
<thead>
<tr>
<th>ACC/AHA HF Stage¹</th>
<th>NYHA Functional Class²</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A</strong> At high risk for heart failure but without structural heart disease or symptoms of heart failure (eg, patients with hypertension or coronary artery disease)</td>
<td>I  Asymptomatic</td>
</tr>
<tr>
<td><strong>B</strong> Structural heart disease but without symptoms of heart failure</td>
<td>II  Symptomatic with moderate exertion</td>
</tr>
<tr>
<td><strong>C</strong> Structural heart disease with prior or current symptoms of heart failure</td>
<td>III  Symptomatic with minimal exertion</td>
</tr>
<tr>
<td><strong>D</strong> Refractory heart failure requiring specialized interventions</td>
<td>IV  Symptomatic at rest</td>
</tr>
</tbody>
</table>

Pressure Volume Loops

In systolic HF, there is decreased contractility and subsequent increase in LV volume/LVEDP, shifting the loop **DOWN** and to the **RIGHT**

In diastolic HF, there is an increase in LVEDP due to increased stiffness with minimal effect on contractility, shifting the loop **UP** and to the **LEFT**
Pathologic Progression of systolic CHF

Line N to A represents the initial reduction in cardiac output.

Line A to B represents the mechanism of compensation; an increase in LVEDP needed to maintain cardiac output.
Activation of the SNS
Activation of the RAAS

Heart Rate and inotropy
Myocardial toxicity

Vasoconstriction - ↑ afterload
Hemodynamic alterations - ↑ preload

SNS Inhibited by:
Beta-blockers

RAAS = Renin Angiotensin Aldosterone System
SNS = Sympathetic Nervous System

Negative remodeling
Worsened LV Function

Symptoms of heart failure

RAAS Inhibited by:
ACE Inhibitors
Angiotensin receptor blockers
Aldosterone antagonists

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### Recommendations for Treatment

#### Stage A

**Class I**

1. Hypertension and lipid disorders should be controlled in accordance with contemporary guidelines to lower the risk of HF. (Level of Evidence: A)
2. Other conditions that may lead to or contribute to HF, such as obesity, diabetes mellitus, tobacco use, and known cardiotoxic agents, should be controlled or avoided. (Level of Evidence: C)

#### Stage B

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>In patients with a history of MI and reduced EF, ACE inhibitors or ARBs should be used to prevent HF</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>In patients with MI and reduced EF, evidence-based beta blockers should be used to prevent HF</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>In patients with MI, statins should be used to prevent HF</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Blood pressure should be controlled to prevent symptomatic HF</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>ACE inhibitors should be used in all patients with a reduced EF to prevent HF</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Beta blockers should be used in all patients with a reduced EF to prevent HF</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>An ICD is reasonable in patients with asymptomatic ischemic cardiomyopathy who are at least 40 d post-MI, have an LVEF ≤30%, and on GDMT</td>
<td>Ila</td>
<td>B</td>
</tr>
<tr>
<td>Nondihydropyridine calcium channel blockers may be harmful in patients with low LVEF</td>
<td>III: Harm</td>
<td>C</td>
</tr>
</tbody>
</table>

NYHA class II-IV and EF ≤ 35% OR EF ≤ 40% after an acute MI and either has sx’s of CHF or DM
<table>
<thead>
<tr>
<th>Drug</th>
<th>Initial Daily Dose(s)</th>
<th>Maximum Dose(s)</th>
<th>Mean Doses Achieved In Clinical Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE Inhibitors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Captopril</td>
<td>6.25 mg 3 times</td>
<td>50 mg 3 times</td>
<td>122.7 mg/d&lt;sup&gt;152&lt;/sup&gt;</td>
</tr>
<tr>
<td>Enalapril</td>
<td>2.5 mg twice</td>
<td>10 to 20 mg twice</td>
<td>16.6 mg/d&lt;sup&gt;153&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fosinopril</td>
<td>5 to 10 mg once</td>
<td>40 mg once</td>
<td>N/A</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>2.5 to 5 mg once</td>
<td>20 to 40 mg once</td>
<td>32.5 to 35.0 mg/d&lt;sup&gt;145&lt;/sup&gt;</td>
</tr>
<tr>
<td>Perindopril</td>
<td>2 mg once</td>
<td>8 to 16 mg once</td>
<td>N/A</td>
</tr>
<tr>
<td>Quinapril</td>
<td>5 mg twice</td>
<td>20 mg twice</td>
<td>N/A</td>
</tr>
<tr>
<td>Ramipril</td>
<td>1.25 to 2.5 mg once</td>
<td>10 mg once</td>
<td>N/A</td>
</tr>
<tr>
<td>Trandolapril</td>
<td>1 mg once</td>
<td>4 mg once</td>
<td>N/A</td>
</tr>
<tr>
<td>ARBs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Candesartan</td>
<td>4 to 8 mg once</td>
<td>32 mg once</td>
<td>24 mg/d&lt;sup&gt;149&lt;/sup&gt;</td>
</tr>
<tr>
<td>Losartan</td>
<td>25 to 50 mg once</td>
<td>50 to 150 mg once</td>
<td>129 mg/d&lt;sup&gt;151&lt;/sup&gt;</td>
</tr>
<tr>
<td>Valsartan</td>
<td>20 to 40 mg twice</td>
<td>160 mg twice</td>
<td>254 mg/d&lt;sup&gt;150&lt;/sup&gt;</td>
</tr>
<tr>
<td>Aldosterone antagonists</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spironolactone</td>
<td>12.5 to 25.0 mg once</td>
<td>25 mg once or twice</td>
<td>26 mg/d&lt;sup&gt;155&lt;/sup&gt;</td>
</tr>
<tr>
<td>Eplerenone</td>
<td>25 mg once</td>
<td>50 mg once</td>
<td>42.6 mg/d&lt;sup&gt;146&lt;/sup&gt;</td>
</tr>
<tr>
<td>Beta blockers</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bisoprolol</td>
<td>1.25 mg once</td>
<td>10 mg once</td>
<td>8.6 mg/d&lt;sup&gt;147&lt;/sup&gt;</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>3.125 mg twice</td>
<td>50 mg twice</td>
<td>37 mg/d&lt;sup&gt;147&lt;/sup&gt;</td>
</tr>
<tr>
<td>Carvedilol CR</td>
<td>10 mg once</td>
<td>80 mg once</td>
<td>N/A</td>
</tr>
<tr>
<td>Metoprolol succinate extended release (metoprolol CR/XL)</td>
<td>12.5 to 25 mg once</td>
<td>200 mg once</td>
<td>159 mg/d&lt;sup&gt;148&lt;/sup&gt;</td>
</tr>
<tr>
<td>Hydralazine and isosorbide dinitrate</td>
<td>Fixed-dose combination&lt;sup&gt;444&lt;/sup&gt;</td>
<td>37.5 mg hydralazine/20 mg isosorbide dinitrate 3 times daily</td>
<td>75 mg hydralazine/40 mg isosorbide dinitrate 3 times daily</td>
</tr>
<tr>
<td>Hydralazine and isosorbide dinitrate&lt;sup&gt;445&lt;/sup&gt;</td>
<td>Hydralazine: 25 to 50 mg, 3 or 4 times daily and isosorbide dinitrate: 20 to 30 mg 3 or 4 times daily</td>
<td>Hydralazine: 300 mg daily in divided doses and isosorbide dinitrate: 120 mg daily in divided doses</td>
<td>N/A</td>
</tr>
</tbody>
</table>

ACE indicates angiotensin-converting enzyme; ARB, angiotensin-receptor blocker; CR, controlled release; CR/XL, controlled release/extended release; HF/EF, heart failure with reduced ejection fraction; and N/A, not applicable.
## Treatment for Stage C: HFpEF

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic and diastolic blood pressure should be controlled according to published clinical practice guidelines</td>
<td>I</td>
<td>B²⁷,⁹¹</td>
</tr>
<tr>
<td>Diuretics should be used for relief of symptoms due to volume overload.</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Coronary revascularization for patients with CAD in whom angina or demonstrable myocardial ischemia is present despite GDMT</td>
<td>Iia</td>
<td>C</td>
</tr>
<tr>
<td>Management of AF according to published clinical practice guidelines for HFpEF to improve symptomatic HF</td>
<td>Iia</td>
<td>C</td>
</tr>
<tr>
<td>Use of beta-blocking agents, ACE inhibitors, and ARBs for hypertension in HFpEF</td>
<td>Ila</td>
<td>C</td>
</tr>
<tr>
<td>ARBs might be considered to decrease hospitalizations in HFpEF</td>
<td>IIb</td>
<td>B⁵⁸⁶</td>
</tr>
<tr>
<td>Nutritional supplementation is not recommended in HFpEF</td>
<td>III: No Benefit</td>
<td>C</td>
</tr>
</tbody>
</table>
Stage D/Advanced HF

INTERMACS (The Interagency Registry for Mechanically Assisted Circulatory Support)

<table>
<thead>
<tr>
<th>Profile*</th>
<th>Profile Description</th>
<th>Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Critical cardiogenic shock (“Crash and burn”)</td>
<td>Life-threatening hypotension and rapidly escalating inotropic/pressor support, with critical organ hypoperfusion often confirmed by worsening acidosis and lactate levels.</td>
</tr>
<tr>
<td>2</td>
<td>Progressive decline (“Sliding fast” on inotropes)</td>
<td>“Dependent” on inotropic support but nonetheless shows signs of continuing deterioration in nutrition, renal function, fluid retention, or other major status indicator. Can also apply to a patient with refractory volume overload, perhaps with evidence of impaired perfusion, in whom inotropic infusions cannot be maintained due to tachyarrhythmias, clinical ischemia, or other intolerance.</td>
</tr>
<tr>
<td>3</td>
<td>Stable but inotrope dependent</td>
<td>Clinically stable on mild-moderate doses of intravenous inotropes (or has a temporary circulatory support device) after repeated documentation of failure to wean without symptomatic hypotension, worsening symptoms, or progressive organ dysfunction (usually renal).</td>
</tr>
<tr>
<td>4</td>
<td>Resting symptoms on oral therapy at home</td>
<td>Patient who is at home on oral therapy but frequently has symptoms of congestion at rest or with activities of daily living (dressing or bathing). He or she may have orthopnea, shortness of breath during dressing or bathing, gastrointestinal symptoms (abdominal discomfort, nausea, poor appetite), disabling ascites, or severe lower-extremity edema.</td>
</tr>
<tr>
<td>5</td>
<td>Exertion intolerant (“housebound”)</td>
<td>Patient who is comfortable at rest but unable to engage in any activity, living predominantly within the house or housebound.</td>
</tr>
<tr>
<td>6</td>
<td>Exertion limited (“walking wounded”)</td>
<td>Patient who is comfortable at rest without evidence of fluid overload but who is able to do some mild activity. Activities of daily living are comfortable and minor activities outside the home such as visiting friends or going to a restaurant can be performed, but fatigue results within a few minutes or with any meaningful physical exertion.</td>
</tr>
<tr>
<td>7</td>
<td>Advanced NYHA class III</td>
<td>Patient who is clinically stable with a reasonable level of comfortable activity, despite a history of previous decompensation that is not recent. This patient is usually able to walk more than a block. Any decompensation requiring intravenous diuretics or hospitalization within the previous month should make this person a Patient Profile 6 or lower.</td>
</tr>
</tbody>
</table>
Treatment

*Consists of 2 parts*

- Support (ie- inotropes) until definitive therapy implemented

- Definitive therapy
  - medications/procedures (ie- revascularization, valve surgery)
  - MCS/Transplant
  - palliative care
<table>
<thead>
<tr>
<th>Inotropic Agent</th>
<th>Dose (mcg/kg)</th>
<th>Drug Kinetics and Metabolism</th>
<th>Effects</th>
<th>Adverse Effects</th>
<th>Special Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Bolus Infusion (/min)</td>
<td>t₁/₂: 2 to 20 min</td>
<td>CO HR SVR PVR</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adrenergic agonists</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>N/A  5 to 10</td>
<td>2 to 3 min R,H,P</td>
<td>↑ ↑ ↔ ↔</td>
<td>T HA, N, tissue necrosis</td>
<td>Caution: MAO-I</td>
</tr>
<tr>
<td>N/A  10 to 15</td>
<td>↑ ↑ ↑ ↑</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>N/A  2.5 to 5</td>
<td>2 to 3 min H</td>
<td>↑ ↑ ↓ ↔</td>
<td>↑/↓ BP, HA, T N, F, hypersensitivity</td>
<td>Caution: MAO-I; Cl: sulfite allergy</td>
</tr>
<tr>
<td>N/A  5 to 20</td>
<td>↑ ↑ ↑ ↔</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PDE inhibitor</td>
<td>N/R  0.125 to 0.75</td>
<td>2.5 h H</td>
<td>↑ ↑ ↓</td>
<td></td>
<td>T, ↓ BP</td>
</tr>
</tbody>
</table>

T= tachyarrhythmia's
“OK, the old one’s in my right hand, the donor’s in my left. Right?”
• An estimated 5.7 million Americans ≥20 years of age have HF
• Projections show that the prevalence of HF will increase 46% from 2012 to 2030, resulting in >8 million people ≥18 years of age with HF

LVAD for Destination Therapy

### Summary of LVAD trials demonstrating ongoing survival improvements

<table>
<thead>
<tr>
<th>Author, reference</th>
<th>Year</th>
<th>Device</th>
<th>Number of patients</th>
<th>1 year survival (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rose <em>et al.</em> (5)</td>
<td>2001</td>
<td>Pulsatile Heartmate</td>
<td>68</td>
<td>52</td>
</tr>
<tr>
<td>Miller <em>et al.</em> (11)</td>
<td>2007</td>
<td>Heartmate II</td>
<td>133</td>
<td>68</td>
</tr>
<tr>
<td>Pagani <em>et al.</em> (12)</td>
<td>2009</td>
<td>Heartmate II</td>
<td>281</td>
<td>73</td>
</tr>
<tr>
<td>Slaughter <em>et al.</em> (15)</td>
<td>2009</td>
<td>Heartmate II</td>
<td>134</td>
<td>68</td>
</tr>
<tr>
<td>John <em>et al.</em> (14)</td>
<td>2011</td>
<td>Heartmate II</td>
<td>1,496</td>
<td>85</td>
</tr>
<tr>
<td>Starling <em>et al.</em> (13)</td>
<td>2011</td>
<td>Heartmate II</td>
<td>169</td>
<td>85</td>
</tr>
<tr>
<td>Aaronson <em>et al.</em> (17)</td>
<td>2012</td>
<td>Heartware HVAD</td>
<td>140</td>
<td>86</td>
</tr>
<tr>
<td>Slaughter <em>et al.</em> (31)</td>
<td>2013</td>
<td>Heartware HVAD</td>
<td>332</td>
<td>84</td>
</tr>
<tr>
<td>Strueber <em>et al.</em> (16)</td>
<td>2014</td>
<td>Heartware HVAD</td>
<td>254</td>
<td>85</td>
</tr>
</tbody>
</table>

**1 year transplant survival rate** 87.8%
CHF Management
The Acute Setting

A: Management to prevent disease progression
B: Diuretics and vasodilators/afterload reduction (ie- HDLZ/NTG, ACEi)
C: Inotropic agents (ie- Dobutamine/milrinone) and diuretics
L: ?adjust outpatient meds, inotropes

Evidence for Congestion (Elevated Filling Pressure)
- Orthopnea
- High Jugular Venous Pressure
- Increasing S₃
- Loud P₂
- Edema
- Ascites
- Rales (Uncommon)
- Abdominoljugular Reflux

Evidence for Low Perfusion
- Narrow Pulse Pressure
- Pulsus Alternations
- Cool Forearms and Legs
- May Be Sleepy, Obtunded
- ACE Inhibitor–Related Symptomatic Hypotension
- Declining Serum Sodium Level
- Worsening Renal Function

Congest? 

Rest?

No         Yes

Warm and Dry  A          Warm and Wet  B

Cold and Dry  L

Cold and Wet  C

Medical Management of Advanced Heart Failure. JAMA. 2002;287:628-640

www.southcoast.org
Diuretic therapy

• Dose Equivalents

<table>
<thead>
<tr>
<th></th>
<th>Furosemide</th>
<th>Torsemide</th>
<th>Bumetanide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dose</td>
<td>40 mg</td>
<td>20 mg</td>
<td>1 mg</td>
</tr>
</tbody>
</table>

• Bolus vs Infusion, ideal dose for acute CHF?

DOSE Trial...
• prospective, double-blind, randomized trial

• 308 patients with acute decompensated heart failure to receive furosemide IV either as a bolus every 12 hours or continuous infusion AND at either a low dose (equivalent to the patient’s previous oral dose) or a high dose (2.5 times the previous oral dose)

• 2 coprimary end points after 72hrs: patient’s global assessment of symptoms (VAS), change in Cr
Secondary Endpoints

*Bolus vs. Continuous*  
*Low-dose vs. High-dose*

<table>
<thead>
<tr>
<th>Endpoint</th>
<th>Bolus vs. Continuous</th>
<th>P-value</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>No congestion at 72 hours</td>
<td>14% vs. 15%</td>
<td>0.78</td>
<td>0.09</td>
</tr>
<tr>
<td></td>
<td>11% vs. 18%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Weight change at 72 hours</td>
<td>-6.8 vs. -8.1 lbs</td>
<td>0.20</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-6.1 vs. -8.7 lbs</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Net fluids at 72 hours</td>
<td>-4,237 vs. -4,249 mL</td>
<td>0.89</td>
<td></td>
</tr>
<tr>
<td></td>
<td>-3,575 vs. -4,899 mL</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Persistent or worsening HF</td>
<td>25% vs. 23%</td>
<td>0.78</td>
<td></td>
</tr>
<tr>
<td></td>
<td>26% vs. 22%</td>
<td>0.40</td>
<td></td>
</tr>
<tr>
<td>Hospital stay</td>
<td>5 vs. 5 days</td>
<td>0.97</td>
<td></td>
</tr>
<tr>
<td></td>
<td>6 vs. 5 days</td>
<td>0.55</td>
<td></td>
</tr>
<tr>
<td>All-cause mortality, rehospitalization, or ED visit</td>
<td>HR for continuous infusion 1.15 (95% CI 0.83-1.60; P=0.41)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HR for high-dose 0.83 (95% CI 0.60-1.16; P=0.28)</td>
<td></td>
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</tr>
</tbody>
</table>
Monitoring and Guiding Therapy
One of the Best Devices for Monitoring Heart Failure
NP Guided Therapy

<table>
<thead>
<tr>
<th>BIOMARKERS</th>
<th>CLASS OF RECOMMENDATION</th>
<th>LEVEL OF EVIDENCE</th>
</tr>
</thead>
<tbody>
<tr>
<td>BNP or NT-proBNP</td>
<td>Diagnosis</td>
<td>I</td>
</tr>
<tr>
<td></td>
<td>Prognosis</td>
<td>I</td>
</tr>
<tr>
<td>Guided-therapy (chronic HF)</td>
<td>IIa</td>
<td>A</td>
</tr>
<tr>
<td>Guided-therapy (acute HF)</td>
<td>IIb</td>
<td>B</td>
</tr>
</tbody>
</table>

Diagnosis For HF

NT-proBNP versus clinical judgment, $P = 0.006$
Combined versus NT-proBNP, $P = 0.04$
Combined versus clinical judgment $P < 0.001$

- Combined, AUC = 0.96
- NT-proBNP, AUC = 0.94
- Clinical judgment, AUC = 0.90

Heart Failure: A Companion to Braunwald's Heart Disease.
3rd Edition. Mann and Felker
Meta-Analytic Analysis of Trials Evaluating NP-Guided Therapy

All Cause Mortality

NP Guided Rx


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N-Terminal–Pro-Brain Natriuretic Peptide Predicts Outcome After Hospital Discharge in Heart Failure Patients

Paulo Bettencourt, PhD; Ana Azevedo, MD; Joana Pimenta, MD; Fernando Friões, MD; Susana Ferreira, MD; António Ferreira, PhD
• Evaluate dynamic changes in renal function (sWRF: absolute increase in serum Cr level of >0.5 mg/dl in combination with >25% increase in serum Cr level) compared to dynamic changes in Pro-BNP

• 1,232 pts hospitalized for ADHF (74% HFrEF, 26% HFpEF)

• Endpoints were all-cause mortality and the composite of all-cause mortality and/or readmission for a cardiovascular reason within 180 days after discharge
Key objective in CHF treatment: relieve congestion and achieve euvolemia

≥ 30% BNP reduction ~ 15% in 180 mortality

...BNP is stronger predictor of outcomes vs renal fnc
OptiVol in Patients with CRT/ICD Devices

- Measures intrathoracic impedance, which is inversely related to PCWP
- The OptiVol fluid index will rise as intrathoracic fluid level increases
Upon Discharge

Clinical Status Goals
Achievement of dry weight
Definition of optimal blood pressure range
Walking without dyspnea or dizziness

Stability Goals
Twenty-four hours without changes in oral heart failure regimen
At least 48 hours off intravenous inotropic agents, if used
Fluid balance even on oral diuretics
Renal function stable or improving

Home Maintenance Plan
Patient and family education about
Sodium restriction
Fluid limitation
Medication schedule
Medication effects
Exercise prescription
Flexible diuretic plan
Scheduled call to patient within 3 days
Indications for when to call nurse, physician, or 911
Clinic appointment within 5 to 10 days

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Medical Management of Advanced Heart Failure. JAMA. 2002;287:628-640
New Developments
Prospective, randomized double blind trial

8442 pts with EF ≤ 40%, NYHA II-IV

LCZ696 (ARB valsartan + neprilysin inhibitor) vs. enalapril (mean dose 18.9mg)

Primary Endpoint: Death from cardiovascular causes or hospitalization for heart failure
NNT to prevent one primary event and one death from CV causes = 21 and 32
• Double-blinded multi-center RCT

• Ivabradine vs placebo

• 6558 pts with EF ≤ 35% AND
  • Sinus rhythm
  • HR ≥ 70
  • Symptomatic, hospitalized for CHF within past year
  • On stable chronic therapy, including BB

• Primary Endpoint: Composite of cardiovascular death or hospital admission for worsening heart failure

Lancet. 2010 ;376:875-85
Mainly driven by reduction in HF hospitalizations
CardioMEMS

Inserted via RHC
Placed in branch of PA
Measures PAP (PASP, PADP, mean)
Cumulative HF Hospitalizations Reduced
At 6 Months and Full Duration

≤ 6 Months
28% RRR, p = 0.0002
> 6 Months
45% RRR, p < 0.0001

Study Duration
37% RRR, p < 0.0001

Days from Implant
Cumulative Number of HF Hospitalizations
0 20 40 60 80 100 120 140 160 180 200 220 240 260 280
0 90 180 270 360 450 540 630 720 810 900

Red: Treatment (158 HF Hospitalizations)
Blue: Control (264 HF Hospitalizations)
Take Home Points

• Treatment based on Type/Stage of CHF

• Systolic (HFrEF) vs Diastolic (HFpEF) CHF: different pathophysiology

• Evaluation of CHF pt: congestion and perfusion

• Key is to relieve congestion
Thank You