Pathophysiology: Heart Failure

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Objectives

At the conclusion of this seminar, learners will be able to:
1. Define heart failure as a clinical syndrome
2. Define and employ the terms preload, afterload, contractility, remodeling, diastolic dysfunction, compliance, stiffness and capacitance.
3. Describe the classic pathophysiologic steps in the development of heart failure.
4. Delineate four basic mechanisms underlying the development of heart failure.
5. Interpret pressure volume loops / Starling curves and identify contributing mechanisms for heart failure state.
6. Understand the common methods employed for classifying patients with heart failure.
7. Employ the classes and stages of heart failure in describing a clinical scenario.
Heart Failure

• Not a disease
• A syndrome
  – From "syn" meaning "together" and "dromos" meaning "a running".
  – A group of signs and symptoms that occur together and characterize a particular abnormality.
• Diverse etiologies
• Several mechanisms

Heart Failure: Definitions

• An inability of the heart to pump blood at a sufficient rate to meet the metabolic demands of the body (e.g. oxygen and cell nutrients) at rest and during effort or to do so only if the cardiac filling pressures are abnormally high.
• A complex clinical syndrome characterized by abnormalities in cardiac function and neurohormonal regulation, which are accompanied by effort intolerance, fluid retention and a reduced longevity
• A complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.
Epidemiology Heart Failure: The Problem

- 3.5 million in 1991, 4.7 million in 2000, estimated 10 million in 2037
- Incidence: 550,000 new cases/year
- Prevalence: 1% ages 50--59, >10% over age 80
- More deaths from HF than from all forms of cancer combined
- Most common cause for hospitalization in age >65

Heart Failure Paradigms

1950 2000
Heart Failure: Classifications

- Right vs. Left Sided
- Cardiac vs. Non-cardiac
- Dilated vs. Hypertrophic vs. Restrictive
- Acute vs. Chronic
- Systolic vs. Diastolic
- Compensated vs. Decompensated
- High vs. Low Output
- Forward vs. Backward

Cardiac Muscle Function

**Preload**
- The length of a cardiac muscle fiber prior to the onset of contraction.
- Frank Starling

**Afterload**
- The force against which a cardiac muscle fiber must shorten.
- Isotonic Contraction

**Contractility**
- The force of contraction independent of preload and afterload.
- Inotropic State
From Muscle to Chamber

The Pressure Volume Loop
The Pressure Volume Loop

Compliance/Stiffness vs Capacitance
**Afterload (Arterial Properties)**

**Ea (Arterial Elastance)**
- If
  - TPR = \([\text{MAP} - \text{CVP}] / \text{CO}\), and
  - \(\text{CO} = \text{SV} \times \text{HR}\)
- Substituting the second equation into the first we obtain:
  - TPR = \([\text{MAP} - \text{CVP}] / (\text{SV} \times \text{HR})\)
- Making two simplifying assumptions.
  1. CVP is negligible compared to MAP.
  2. MAP is approximately equal to the end-systolic pressure in the ventricle (Pes).
- Then,
  - TPR = Pes / (SV \times HR)
- Which can be rearranged to:
  - Pes/SV \approx \text{TPR} \times \text{HR}.

---

**Cardiac Chamber Function**

**Preload**
- EDV
- EDP
- Wall stress at end diastole

**Afterload**
- Aortic Pressure
- Total peripheral resistance
- Arterial impedance
- Wall stress at end systole

**Contractility**
- Pressure generated at given volume.
- Inotropic State
Frank Starling Curves

Pathophysiology - PV Loop
Pathophysiology of Heart Failure

Myocardial Insult/Stimuli/Damage → Pump dysfunction → Activation of neurohormones
• Catecholamines
• Angiotensin II
• Cytokines

Remodeling
• Hypertrophy
• Fibrosis
• Apoptosis

Neurohormonal Activation in Heart Failure

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

Initial fall in LV performance, ↑ wall stress

Activation of RAS and SNS

Remodeling and progressive worsening of LV function

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

Peripheral vasoconstriction

Sodium retention

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.
Neurohormonal Activation in Heart Failure

Hypertrophy, apoptosis, ischemia, arrhythmias, remodeling, fibrosis

Morbidity and Mortality

Adrenergic Pathway in Heart Failure Progression

β₁, β₂, α₁
Myocyte hypertrophy
Myocyte injury
Increased arrhythmias

β₁, α₁
Vasoconstriction

Activation of RAS
Sodium retention

Disease progression

↑ CNS sympathetic outflow

↑ Cardiac sympathetic activity

↑ Vascular sympathetic activity

↑ Renal sympathetic activity
Neurohormonal Balance in Heart Failure

Neurohormones in Heart Failure

Myocardial Injury → Fall in LV Performance

- Activation of RAAS and SNS (endothelin, AVP, cytokines)
- ANP
- BNP

- Myocardial Toxicity
- Change in Gene Expression
- Remodeling and Progressive Worsening of LV Function

→ Peripheral Vasoconstriction
- Sodium/Water Retention
- HF Symptoms

Morbidity and Mortality

Pathophysiology of myocardial remodeling:

**Insult / Remodeling Stimuli**
- **↑ Wall Stress**
- **Cytokines**
- **Neurohormones**
- **Oxidative stress**

**Increased Wall Stress**

**Myocyte Hypertrophy**

**Ventricular Enlargement**

**Altered interstitial matrix**

**Diastolic Dysfunction**

**Fetal Gene Expression**

**Systolic Dysfunction**

**Altered calcium handling proteins**

**Myocyte Death**

Acute and Chronic Responses – Benefits and Harm

<table>
<thead>
<tr>
<th>Response</th>
<th>Short-term Effects (mainly adaptive; hemorrhage, acute heart failure)</th>
<th>Long-term Effects (mainly deleterious; chronic heart failure)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salt and water retention</td>
<td>Augments preload</td>
<td>Pulmonary congestion, anasarca</td>
</tr>
<tr>
<td>Vasoconstriction</td>
<td>Maintains pressure for perfusion of vital organs (brain, heart)</td>
<td>Exacerbates pump dysfunction, increases cardiac energy expenditure</td>
</tr>
<tr>
<td>Sympathetic stimulation</td>
<td>Increases heart rate and ejection</td>
<td>Increases energy expenditure</td>
</tr>
<tr>
<td>Cytokine activation</td>
<td>Vasodilatation</td>
<td>Skeletal muscle catabolism, deterioration of endothelial function, impaired contraction, LV remodeling</td>
</tr>
<tr>
<td>Hypertrophy</td>
<td>Unloads individual muscle fibers</td>
<td>Deterioration and death of cardiac cells: cardiomyopathy of overload</td>
</tr>
<tr>
<td>Increased collagen</td>
<td>May reduce dilatation</td>
<td>Impairs relaxation</td>
</tr>
</tbody>
</table>
Laplace’s Law

Where $P = \text{ventricular pressure}$, $r = \text{ventricular chamber radius}$ and $h = \text{ventricular wall thickness}$

Remodeling – Concentric vs. Eccentric
Ventricular Remodeling

Pathophysiology of Heart Failure

Four Basic Mechanisms
1. Increased Blood Volume (Excessive Preload)
2. Increased Resistant to Blood Flow (Excessive Afterload)
3. Decreased contractility
4. Decreased Filling
Increased Blood Volume

**Etiologies**
- Mitral Regurgitation
- Aortic Regurgitation
- Volume Overload
- Left to Right Shunts
- Chronic Kidney Disease

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal</th>
<th>AI</th>
<th>AI + Neurohormones</th>
<th>AI + Remodeling</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP (mm Hg)</td>
<td>140/75</td>
<td>128/50</td>
<td>130/50</td>
<td>104/45</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>64</td>
<td>80</td>
<td>82</td>
<td>63</td>
</tr>
<tr>
<td>Cardiac Output (L/min)</td>
<td>3.8</td>
<td>3.0</td>
<td>4.3</td>
<td>2.6</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>10</td>
<td>15</td>
<td>25</td>
<td>20</td>
</tr>
</tbody>
</table>

Increased Afterload

**Etiologies**
- Aortic Stenosis
- Aortic Coarctation
- Hypertension

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal</th>
<th>HTN</th>
<th>HTN + DD</th>
<th>HTN + Heart failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP (mm Hg)</td>
<td>131/76</td>
<td>150/100</td>
<td>140/92</td>
<td>161/105</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>57</td>
<td>52</td>
<td>49</td>
<td>57</td>
</tr>
<tr>
<td>Cardiac Output (L/min)</td>
<td>4.0</td>
<td>3.6</td>
<td>3.4</td>
<td>4.0</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>10</td>
<td>10</td>
<td>13</td>
<td>23</td>
</tr>
</tbody>
</table>
### Decreased Contractility

**Etiologies**
- Ischemic Cardiomyopathy
  - Myocardial Infarction
  - Myocardial Ischemia
- Myocarditis
- Toxins
  - Anthracycline
  - Alcohol
  - Cocaine

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal</th>
<th>MI</th>
<th>MI + Neurohormones</th>
<th>MI + Remodeling</th>
</tr>
</thead>
<tbody>
<tr>
<td>BP (mm Hg)</td>
<td>124/81</td>
<td>80/40</td>
<td>90/54</td>
<td>87/44</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>60</td>
<td>42</td>
<td>46</td>
<td>46</td>
</tr>
<tr>
<td>Cardiac Output (L/min)</td>
<td>4.2</td>
<td>3.0</td>
<td>3.2</td>
<td>3.2</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>12</td>
<td>17</td>
<td>25</td>
<td>23</td>
</tr>
</tbody>
</table>

### Decreased Filling

**Etiologies**
- Mitral Stenosis
- Constriction
- Restrictive Cardiomyopathy
- Cardiac Tamponade
- Hypertrophic Cardiomyopathy
- Infiltrative Cardiomyopathy

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal</th>
<th>HCM</th>
<th>HCM + HF</th>
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</thead>
<tbody>
<tr>
<td>BP (mm Hg)</td>
<td>124/81</td>
<td>95/47</td>
<td>105/53</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>63</td>
<td>50</td>
<td>55</td>
</tr>
<tr>
<td>Cardiac Output (L/min)</td>
<td>4.4</td>
<td>3.5</td>
<td>3.8</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>10</td>
<td>17</td>
<td>26</td>
</tr>
</tbody>
</table>
Part II

Heart Failure: Classifications

- Right vs. Left Sided
- Cardiac vs. Non-cardiac
- Dilated vs. Hypertrophic vs. Restrictive
- Acute vs. Chronic
- Forward vs. Backward
- Compensated vs. Decompensated
- High vs. Low Output
- Systolic vs. Diastolic
Types of Heart Failure

<table>
<thead>
<tr>
<th></th>
<th>SHF</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathophysiology</td>
<td>Impaired Contraction</td>
<td>Impaired filling</td>
</tr>
<tr>
<td>Demographics</td>
<td>All ages</td>
<td>&gt; 60 years</td>
</tr>
<tr>
<td>1° Cause</td>
<td>Coronary Artery Disease</td>
<td>Hypertension</td>
</tr>
</tbody>
</table>

Systolic Versus Diastolic Failure

- **Systolic Dysfunction**: 
  - Pressure
  - Volume
  - ↓ Contractility

- **Normal**: 
  - Pressure
  - Volume

- **Diastolic Dysfunction**: 
  - Pressure
  - Volume
  - ↓ Capacitance
# Systolic Versus Diastolic Failure

## Heart Failure: Classifications

- Right vs. Left Sided
- Cardiac vs. Non-cardiac
- Systolic vs. Diastolic
- Dilated vs. Hypertrophic vs. Restrictive
- Acute vs. Chronic
- Forward vs. Backward
- High vs. Low Output
- COMPENSATED vs. DECOMPENSATED

## Table 2. Characteristics of Patients with Diastolic Heart Failure and Patients with Systolic Heart Failure

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Diastolic Heart Failure</th>
<th>Systolic Heart Failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Frequently female</td>
<td>All ages, typically 50–70 y.</td>
</tr>
<tr>
<td>Sex</td>
<td>Preserved or normal, approximately 40% or higher</td>
<td>More often male</td>
</tr>
<tr>
<td>Left ventricular ejection fraction</td>
<td>Preserved or normal, approximately 40% or higher</td>
<td>Depressed, approximately 40% or lower</td>
</tr>
<tr>
<td>Left ventricular cavity size</td>
<td>Usually normal, often with symmetric left ventricular hypertrophy</td>
<td>Usually dilated</td>
</tr>
<tr>
<td>Left ventricular hypertrophy on echocardiography</td>
<td>Usually present</td>
<td>Sometimes present</td>
</tr>
<tr>
<td>Chest radiography</td>
<td>Congestion with or without cardiomegaly</td>
<td>Congestion and cardiomegaly</td>
</tr>
<tr>
<td>Gallbladder present</td>
<td>Fourth heart sound</td>
<td>Third heart sound</td>
</tr>
<tr>
<td>Compromising conditions</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>+</td>
<td>+++</td>
</tr>
<tr>
<td>Obesity</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Chronic lung disease</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>Sleep apnea</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Long-term dialysis</td>
<td>++</td>
<td>0</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

* A single plus sign denotes “occasionally associated with.” Two plus signs “usually associated with.” Three plus signs “usually associated with,” and a zero “not associated with.”
Decompensated Heart Failure

Heart Failure: Classifications

- Right vs. Left Sided
- Cardiac vs. Non-cardiac
- Systolic vs. Diastolic
- Compensated vs. Decompensated
- Dilated vs. Hypertrophic vs. Restrictive
- Acute vs. Chronic
- Forward vs. Backward
- High vs. Low Output
**High vs. Low Output Failure**

- Causes:
  - Anemia
  - Systemic arteriovenous fistulas
  - Hyperthyroidism
  - Beriberi heart disease
  - Paget disease of bone
  - Glomerulonephritis
  - Polycythemia vera
  - Carcinoid syndrome
  - Obesity
  - Anemia
  - Multiple myeloma
  - Pregnancy
  - Cor pulmonale
  - Polycythemia vera

**Heart Failure: Classifications**

- Right vs. Left Sided
- Systolic vs. Diastolic
- Compensated vs. Decompensated
- High vs. Low Output
- Cardiac vs. Non-cardiac
- Acute vs. Chronic
- Forward vs. Backward
- Dilated vs. Hypertrophic vs. Restrictive
# Dilated vs. Hypertrophic vs. Restrictive

<table>
<thead>
<tr>
<th>Type</th>
<th>Definition</th>
<th>Sample Etiologies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dilated</td>
<td>Dilated left/both ventricle(s) with impaired contraction</td>
<td>Ischemic, idiopathic, familial, viral, alcoholic, toxic, valvular</td>
</tr>
<tr>
<td>Hypertrophic</td>
<td>Left and/or right ventricular hypertrophy</td>
<td>Familial with autosomal dominant inheritance</td>
</tr>
<tr>
<td>Restrictive</td>
<td>Restrictive filling and reduced diastolic filling of one/both ventricles, Normal/near normal systolic function</td>
<td>Idiopathic, amyloidosis, endomyocardial fibrosis</td>
</tr>
</tbody>
</table>

**Sample Etiologies**

- **Ischemic**
- **Idiopathic**
- **Familial**
- **Viral**
- **Alcoholic**
- **Toxic**
- **Valvular**

### Diagrams

**Normal Heart vs. Heart with Dilated Cardiomyopathy**
- **Normal Heart**: Heart chambers relax and fill, then contract and pump.
- **Heart with Dilated Cardiomyopathy**: Muscle fibers have stretched. Heart chamber enlarged.

**Heart with Hypertrophic Cardiomyopathy vs. Heart with Restrictive Cardiomyopathy**
- **Heart with Hypertrophic Cardiomyopathy**: Growth and arrangement of muscle fibers are abnormal. Heart walls thicken, especially in the left ventricle.
- **Heart with Restrictive Cardiomyopathy**: Ventricle walls stiffen and lose flexibility.
Clinical Manifestations

**Symptoms**
- Reduced exercise tolerance
- Shortness of breath
- Congestion
- Fluid retention
- Difficulty in sleeping
- Weight loss

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sensitivity</th>
<th>Specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hx of HF</td>
<td>62</td>
<td>94</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>56</td>
<td>53</td>
</tr>
<tr>
<td>Orthopnea</td>
<td>47</td>
<td>88</td>
</tr>
<tr>
<td>Rales</td>
<td>56</td>
<td>80</td>
</tr>
<tr>
<td>S3</td>
<td>20</td>
<td>99</td>
</tr>
<tr>
<td>JVD</td>
<td>39</td>
<td>94</td>
</tr>
<tr>
<td>Edema</td>
<td>67</td>
<td>68</td>
</tr>
</tbody>
</table>

Diagnosis of heart failure

- Physical examination
- Chest X ray
- EKG
- Echocardiogram
- Blood tests: Na, BUN, Creatinine, BNP
- Exercise test
- MRI
- Cardiac catheterization
NYHA Classification

<table>
<thead>
<tr>
<th>Class</th>
<th>Patient Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Mild</td>
</tr>
<tr>
<td></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>Mild</td>
</tr>
<tr>
<td></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>Moderate</td>
</tr>
<tr>
<td></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>Severe</td>
</tr>
<tr>
<td></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>

ACC/AHA Staging System

STAGE A  High risk for developing HF

STAGE B  Asymptomatic LV dysfunction

STAGE C  Past or current symptoms of HF

STAGE D  End-stage HF

**ACC/AHA Staging System**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Patient Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>High risk for developing heart failure</td>
</tr>
<tr>
<td></td>
<td>• Hypertension</td>
</tr>
<tr>
<td></td>
<td>• Coronary artery disease</td>
</tr>
<tr>
<td></td>
<td>• Diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>• Family history of cardiomyopathy</td>
</tr>
<tr>
<td>B</td>
<td>Asymptomatic heart failure</td>
</tr>
<tr>
<td></td>
<td>• Previous myocardial infarction</td>
</tr>
<tr>
<td></td>
<td>• Left ventricular systolic dysfunction</td>
</tr>
<tr>
<td></td>
<td>• Asymptomatic valvular disease</td>
</tr>
<tr>
<td>C</td>
<td>Symptomatic heart failure</td>
</tr>
<tr>
<td></td>
<td>• Known structural heart disease</td>
</tr>
<tr>
<td></td>
<td>• Shortness of breath and fatigue</td>
</tr>
<tr>
<td></td>
<td>• Reduced exercise tolerance</td>
</tr>
<tr>
<td>D</td>
<td>Refractory end-stage heart failure</td>
</tr>
<tr>
<td></td>
<td>• Marked symptoms at rest despite maximal medical therapy (e.g., those who are recurrently hospitalized or cannot be safely discharged from the hospital without specialized interventions)</td>
</tr>
</tbody>
</table>

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**Goals of Treatment**

1. Identification and correction of underlying condition causing heart failure.
2. Elimination of acute precipitating cause of symptoms.
4. Improve long term survival.
Etiologies

- Ischemic cardiomyopathy
- Valvular cardiomyopathy
- Hypertensive cardiomyopathy.
- Inflammatory cardiomyopathy
- Metabolic cardiomyopathy
- General system disease
- Muscular dystrophies.
- Neuromuscular disorders.
- Sensitivity and toxic reactions.
- Peripartal cardiomyopathy

Percipients /Associated Factors

- Inappropriate reduction in the intensity of treatment, including
  - Dietary sodium restriction,
  - Physical activity reduction,
  - Drug regimen reduction, or,
  - most commonly, a combination of these measures.
- Ischemia
- Hypertension
- Anemia
- Volume Overload
- Increased Metabolic Demand
  - Infection
  - Thyroid Disease
- Arrhythmia
- Asthma/COPD
### Targets of Treatment

**Standard Pharmacological Therapy**
- ACE inhibitors
- Angiotensin Receptor Blockers
- Beta Blockers
- Diuretics
- Aldosterone Antagonists
- Statins
- Vasodilators
- Inotropes

### Treatment

<table>
<thead>
<tr>
<th>Stage</th>
<th>Patient Treatment</th>
</tr>
</thead>
</table>
| A     | High risk for developing heart failure | - Optimal pharmacologic therapy (OPT)  
- Aspirin, ACE inhibitors, statins, b-blockers, a-b-blockers (carvedilol) diabetic therapy |
| B     | Asymptomatic heart failure | - OPT  
- ICD if left ventricular (LV) dysfunction (systolic) present |
| C     | Symptomatic heart failure | - OPT  
- ICD if LV dysfunction (systolic) present  
- CRT (if QRS wide, LVEF≤35%) |
| D     | Refractory end-stage heart failure | - OPT  
- Intermittent IV inotropes  
- ICD as a bridge to transplantation  
- CRT  
- Other devices (LVAD, pericardial restraint) |
Treatment of Acute Heart Failure

Diuretics
Reduce fluid volume

Vasodilators
Decrease Preload And Afterload

Inotropes
Augment Contractility

ACC/AHA Staging System

From Risk Factors to Heart Failure

Myocardial infarction
Coronary thrombosis
Myocardial ischemia
CHD
Atherosclerosis
LVH
Risk factors
HTN
Hyperlipidemia
Diabetes
Smoking
Renal disease

Remodeling
Ventricular enlargement
HF
Stage C & D
Death
Stage A
Stage B
Sudden death
Summary

- Complex Clinical Syndrome
- Multiple Etiologies and Classification Systems
- Physiologic Understanding Essential

http://www.columbia.edu/itc/hs/medical/heartsim/