HEART FAILURE

American Heart Association (2013) defines Heart failure (HF) as complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood. Contributing factors to the development of HF are coronary heart disease, diabetes, hypertension, obesity and other heart related issues.
HEART FAILURE

HFpEF includes ejection fraction 50% or greater, clinical signs and symptoms of HF and evidence of abnormal left ventricular diastolic dysfunction.

- As a group, patients with HF with preserved EF are older, are more likely to be female, and have greater hypertension, obesity, and anemia than those with HF with reduced Ejection Fraction

HFrEF Heart failure with reduce ejection fraction incorporates clinical diagnosis of heart failure and reduced ejection fraction 40%.
HEART FAILURE

National Healthcare Issue

▪ Prevalence
  - 6.5 million
  - > 8 million in 2030

▪ Incidence
  - 950,000

▪ Mortality
  - 30 day: 10.4%
  - 1 year: 22%
  - 5 year: 61%
HEART FAILURE

National Healthcare Issue

- **Hospitalizations**
  - > 1 million/annually

- **Readmission Rate**
  - 20% annually

- **Cost**
  - Direct: $21 Billion to $53 Billion in 2030
  - Total: $30 billion to $70 Billion in 2030
  - Medicare: 14% FFS Medicare Recipients
  - 34% annual expenditure
Stages of heart failure

D
Refractory

C
Prior, current symptoms

B
Structural heart disease
LVH, MI, low LVEF, dilatation, valvular disease

A
High-risk patients
Hypertension, diabetes, coronary disease, family history, cardiotoxic drugs
HEART FAILURE

Hemodynamics

▪ Right Atrium
  o 2-6mmHg (CVP)

▪ Right Ventricle
  o Systolic 15-25mmHg
  o Diastolic 0-8mmHg

▪ Pulmonary artery
  o Systolic 15-25mmHg
  o Diastolic 8-15mmHg
HEART FAILURE

Hemodynamics

• Left Atrium
  o 12-15 mmHg

• Left Ventricle
  o Systolic 110-130 mmHg
  o Diastolic 0-8 mmHg

• Aorta
  o Systolic 110-130 mmHg
  o Diastolic 8-12 mmHg
Cardiac Output (CO) is the amount of blood ejected by the heart measured in liters per minute.

- Cardiac Output 4 – 8 l/min
- Cardiac Index 2.5 – 4 L/min

Stroke Volume (SV) is the amount of blood ejected from the ventricles with one contraction.

- \[ CO = SV \times HR \]
  - Preload
  - Afterload
  - Contractility
HEART FAILURE

Cardiac Function

- Decrease in Cardiac Output
  - Decrease in stroke volume
    - Systolic Dysfunction
      - Loss of inotropy (contractility)
    - Diastolic Dysfunction
      - Less Compliant (stiffer)

- higher ventricular end diastolic pressure
HEART FAILURE

HF Cardiac and Vascular Changes

Cardiac
- Decreased stroke volume & cardiac output
- Increased end-diastolic pressure
- Ventricular dilation or hypertrophy
- Impaired filling (diastolic dysfunction)
- Reduced ejection fraction (systolic dysfunction)

Vascular
- Increased systemic vascular resistance
- Decreased arterial pressure
- Impaired arterial pressure
- Impaired organ perfusion
- Decreased venous compliance
- Increased venous pressure
- Increased blood volume
HEART FAILURE

Compensatory Mechanisms

Cardiac
- Frank-Starling mechanism
- Chronic ventricular dilation or hypertrophy
- Tachycardia

Autonomic Nerves
- Increased sympathetic adrenergic activity

Hormones
- Renin-angiotensin-aldosterone system
- Vasopressin (antidiuretic hormone)
- Circulating catecholamines
- Natriuretic peptides
End organ hypoperfusion:
- Fatigue, cyanosis, cool extremities, renal involvement, confusion

Abnormality in pericardium, myocardium, endocardium or valves

Systolic Abnormality: Decreased Contractility (Increased ESV)
Diastolic Abnormality: Decreased filling/compliance (Decreased EDV)

HEART FAILURE

Backup of blood:
- Pulmonary edema (Elevated PCWP)
- Dyspnea, orthopnea

Decreased Cardiac Output

COMPENSATORY MECHANISM

Left ventricular dilatation:
- Increased preload and thus increased stroke volume

Activation of the sympathetic nervous system:
- Increased heart rate
- Systemic vasoconstriction and hence increase in blood pressure
- Venous vasoconstriction

Activation of the renin angiotensin aldosterone system:
- Sodium and water retention that increases preload and stroke volume
- Systemic vasoconstriction due to angiotensin II

EXACERBATION OF SYMPTOMS AND INCREASED CARDIAC REMODELING

Further decrease in contractility
Mitral regurgitation
Left atrial enlargement and atrial fibrillation

Increased myocardial demands and worsening of preexisting ischemia

Volume retention leading to pulmonary and systemic edema

EXACERBATION OF HEART FAILURE
Changes in cardiac output (CO) and right atrial pressure ($P_{RA}$) in response to cardiac failure and compensatory increases in blood volume (Vol) and systemic vascular resistance (SVR), and decreased venous compliance ($C_V$). A, normal operating point; B, decreased cardiac performance; C, compensatory increase in Vol and decrease in $C_V$; and D, increased SVR coupled with increased Vol and reduced $C_V$. 
Pulmonary capillary wedge pressure (PCWP)
- provides an indirect estimate of left atrial pressure (LAP)

Normal 6 to 12
- Outflow pressure for pulmonary circulation
- Snapshot pulmonary capillaries

Pressures above 20mmHg
- Pulmonary Edema
- PCWP guided therapy
## HEART FAILURE

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Onset</th>
<th>Signs and Symptoms</th>
<th>Hemodynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute decompensated heart failure</td>
<td>Days weeks</td>
<td>Weakness</td>
<td>↑ PCWP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dyspnea</td>
<td>↓ CI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rales</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Third heart sound (S3) gallop</td>
<td>Normal or ↑ RAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Edema</td>
<td>↑ SVR</td>
</tr>
<tr>
<td>Acute pulmonary edema, normal BP</td>
<td>Abrupt-days</td>
<td>Severe dyspnea</td>
<td>↑↑ PCWP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diffuse rales</td>
<td>Normal or ↓ CI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cyanosis</td>
<td>Normal RA</td>
</tr>
<tr>
<td></td>
<td></td>
<td>S3 gallop</td>
<td>Normal or ↑ SVR</td>
</tr>
<tr>
<td>Acute pulmonary edema with hypertension</td>
<td>Acute</td>
<td>Severe dyspnea</td>
<td>↑↑ PCWP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Diffuse rales</td>
<td>Normal CI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Normal RAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↑↑ SVR</td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>Acute</td>
<td>Hypotension</td>
<td>↑↑ PCWP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cyanosis</td>
<td>↑ RAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lethargy</td>
<td>↓↓ CI</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cool, clammy</td>
<td>↑ SVR</td>
</tr>
<tr>
<td>High-output heart failure</td>
<td>Days-weeks</td>
<td>Dyspnea</td>
<td>↑ PCWP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Rales</td>
<td>↑ CI</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↓ SVR</td>
</tr>
<tr>
<td>RV failure</td>
<td>Days-weeks</td>
<td>Hypotension</td>
<td>Normal or ↑ PCWP</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Edema</td>
<td>↑↑ RAP</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>↓↓ CI</td>
</tr>
</tbody>
</table>
Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial

- Compared comprehensive history and physical to the participants, RAP, PCWP and CI.
- In advanced heart failure, the presence of orthopnea and increased jugular venous pressure is useful to detect increased pulmonary capillary wedge pressure.

Other Studies

Circulation: Heart Failure. 2008;1:170-177
HEART FAILURE

ACCF/AHA Heart Failure Guidelines

- The physical examination provides information about the severity of illness and allows assessment of volume status and adequacy of perfusion. In advanced HFrEF, orthopnea and jugular venous pressure are useful findings to detect elevated LV filling pressures.

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Monitoring with a pulmonary artery catheter should be performed in patients with respiratory distress or impaired systemic perfusion when clinical assessment is inadequate</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Invasive hemodynamic monitoring can be useful for carefully selected patients with acute HF with persistent symptoms and/or when hemodynamics are uncertain</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>When ischemia may be contributing to HF, coronary arteriography is reasonable</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>Endomyocardial biopsy can be useful in patients with HF when a specific diagnosis is suspected that would influence therapy</td>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>Routine use of invasive hemodynamic monitoring is not recommended in normotensive patients with acute HF</td>
<td>III: No Benefit</td>
<td>B505</td>
</tr>
<tr>
<td>Endomyocardial biopsy should not be performed in the routine evaluation of HF</td>
<td>III: Harm</td>
<td>C</td>
</tr>
</tbody>
</table>

COR indicates Class of Recommendation; HF, heart failure; and LOE, Level of Evidence.

HEART FAILURE

2 Minute Assessment Hemodynamic Profile

Evidence for Congestion (Elevated Filling Pressure)
- Orthopnea
- High Jugular Venous Pressure
- Increasing S3
- Loud P2
- Edema
- Ascites
- Rales (Uncommon)
- Abdominojugular Reflex
- Valsalva Square Wave

Congestion at Rest?
- Yes
  - Warm and Wet
  - B
- No
  - Warm and Dry
  - A

Low Perfusion at Rest?
- Yes
  - Cold and Wet
  - C
- No
  - Cold and Dry
  - L

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

Nohria, Lewis, & Stevenson,(2002) JAMA. 207(5)
LEFT SIDED HEART FAILURE

- Paroxysmal Nocturnal Dyspnea
- Elevated Pulmonary Capillary Wedge Pressure
- Pulmonary Congestion
  - Cough
  - Crackles
  - Wheezes
  - Blood-Tinged Sputum
  - Tachypnea
- Restlessness
- Confusion
- Orthopnea
- Tachycardia
- Exertional Dyspnea
- Fatigue
- Cyanosis

©2007 Nursing Education Consultants, Inc.
HEART FAILURE

RIGHT SIDED \textcolor{red}{\heart} \textbf{FAILURE}  
(\textit{Cor Pulmonale})

- Fatigue
- Peripheral Venous Pressure
- Ascites
- Enlarged Liver & Spleen
- May be secondary to chronic pulmonary problems
- Distended Jugular Veins
- Anorexia & Complaints of GI Distress
- Weight Gain
- Dependent Edema
HEART FAILURE

Hemodynamic Profile Treatment

- **Reduction of filling pressures and congestion**
  - Warm and Wet
  - Diuretic Therapy
    - If symptomatic hypotension is absent, intravenous nitroglycerin, nitroprusside, or nesiritide may be considered an adjuvant to diuretic therapy for relief of dyspnea in patients admitted with acutely decompensated HF.
Stage A
At high risk for heart failure but without structural heart disease or symptoms of HF

- Patients with:
  - hypertension
  - coronary artery disease
  - diabetes mellitus

- Using cardiotoxins
- With FHx CM

Stage B
Structural heart disease but without symptoms of HF

- Patients with:
  - previous MI
  - LV systolic dysfunction
  - Asymptomatic valvular disease

Stage C
Structural heart disease with prior or current symptoms of HF

- Patients with:
  - known structural heart disease
  - Shortness of breath and fatigue, reduced exercise tolerance

Stage D
Refractory HF requiring specialized interventions

- Patients who have 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)

**THERAPY**
- Treat hypertension
- Encourage smoking cessation
- Treat lipid disorders
- Encourage regular exercise
- Discourage alcohol intake, illicit drug use
- ACE inhibition in appropriate patients (see text)

**THERAPY**
- All measures under stage A
- ACE inhibitors in appropriate patients (see text)
- Beta-blockers in appropriate patients (see text)

**THERAPY**
- All measures under Stage A
- Drugs for routine use:
  - Diuretics
  - ACE inhibitors
  - Beta-blockers
  - Digitalis
  - Dietary salt restriction

**THERAPY**
- All measures under stages A, B, and C
- Mechanical assist devices
- Heart transplantation
- Continuous (not intermittent) IV inotropic infusions for palliation
- Hospice care

**Development of symptoms of HF**
HEART FAILURE

Hemodynamic Monitoring

• Current State
  - Invasive hemodynamic monitoring
  - Estimate Volume Status
  - Lab Test
  - Other Comorbid states
  - Telemonitoring
HEART FAILURE

Future of Hemodynamic Monitoring
* Chronicle
  o RV systolic pressure
  o Diastolic pressures
  o HR
  o Pressure derivatives
  o Outcome
* 57% reduction in admission

* Compass-HF
  o Randomize trial
  o Outcome
* non-significant reduction in admission and ED visit
* Retrospective analysis of time to first hospitalization showed reduction
* Observation of high filling pressures prior to symptom
Future of Hemodynamic Monitoring

▪ CHAMPION (CardioMEMS Heart Sensor Allows Monitoring of Pressure to Improve Outcomes on NYHA Class II HF Patients) Trail
  o 550 patients regardless of EF
  o Standard of care plus PAP
  o Specific pressure targets
  o Outcomes
    ▪ Reduction in HF hospitalizations
    ▪ Reduction in PAP
    ▪ Increase number of days alive and not hospitalized
    ▪ Improved QOL
HFrEF or diastolic HF patients represent ~50% of all HF patients.

The effect in HFrEF patients demonstrates an estimated NNT = 2.

**PURPOSE**

Evaluate the effect of PA pressure-guided therapy with the CardioMEMS™ HF System in patients with preserved ejection fraction (EF ≥ 40%), a group with no clinically proven therapies.

---


---

**HF Hospitalization Reduction**

(18 mo follow-up)

n=115, p=0.0004

- 50% Reduction
- p<0.0001 vs. control

---

PA pressure-guided therapy

**SIGNIFICANTLY REDUCED HF HOSPITALIZATIONS**

in HFrEF patients in the treatment group, demonstrating that the CardioMEMS™ HF System is the first effective treatment strategy to manage 50% of patients hospitalized with HF.
Delivers insight into the early onset of worsening HF to more proactively manage HF patients and improve outcomes

CardioMEMS™ HF SYSTEM: PROVIDES CLARITY IN THE MANAGEMENT OF HEART FAILURE

Abraham WT, Lancet, 2011
THE CARDIOMEMS™ HF SYSTEM ENABLES TREND-BASED HEART FAILURE MANAGEMENT

Systolic, mean, and diastolic pressure and heart rate are displayed

- Target specific pulmonary artery pressure ranges, customized for each patient
- Easily track patient data over time and make medication changes when readings are outside of desired ranges

<table>
<thead>
<tr>
<th>PA Systolic</th>
<th>PA Mean</th>
<th>PA Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>80</td>
<td>60</td>
<td>40</td>
</tr>
<tr>
<td>70</td>
<td>50</td>
<td>30</td>
</tr>
<tr>
<td>60</td>
<td>40</td>
<td>20</td>
</tr>
<tr>
<td>50</td>
<td>30</td>
<td>10</td>
</tr>
</tbody>
</table>

[Graph showing PA metrics and events over time]
HEART FAILURE

Future of Hemodynamic Monitoring

- PAP Monitoring Systems in Development
  - Medtronic
    - PAP
    - Cardiac Arrythmias
    - Bluetooth capability Endotronix
Future of Hemodynamic Monitoring

- Left Atrial Pressure (LAP) Monitoring
  - Direct reflection of LAP as primary pressure target may provide more clinical information in the management of HF than PAP.
  - Animal trials have shown strong correlation between increased LAP and pulmonary congestion.
  - Correlation between LAP and LVEDP
  - Advance HF LAP and PAP “mismatch”
  - Pulmonary Hypertension affects liability of PAP
Future of Hemodynamic Monitoring

- LAP Monitoring Systems in Development
  - HeartPod (Medtronic)
  - LAPTOP-HF
  - LAP was measured twice daily, study was stopped early related to perceived excess implant complication
  - Failed to meet end points of decreased hospitalization and complications of heart failure
  - When analyzed using Champion trial endpoints where similar
CENTRAL ILLUSTRATION: The Concept of Pressure-Guided Heart Failure Therapy

HEART FAILURE

QUESTIONS