WHAT IS HEART FAILURE?
PATHOPHYSIOLOGY AND SYMPTOMS

Svetlana Gorbunova, RNP, MSN
Advanced Heart Failure and Cardiac Transplant Program PAVAHCS
Heart Failure (HF) is a complex clinical syndrome that can result from any functional or structural cardiac disorder that impairs the ventricle’s ability to fill with or eject blood.

Different types of HF
- Systolic vs. diastolic
- Left-sided vs. right-sided
- Acute vs. chronic
Systolic vs. Diastolic

- **Systolic**: Inability of the heart to contract effectively
  - Approximately two-thirds of heart failure patients have systolic dysfunction\(^1\)

- **Diastolic**: Impaired filling/relaxation

\(^1\) Lilly, L. *Pathophysiology of Heart Disease*. Second Edition p 200
Causes of Systolic Dysfunction

- CAD/Ischemic heart disease
- Dilated cardiomyopathies
- Hypertension
- Valvular heart disease
Diastolic Dysfunction

- Hypertension
- Ischemic heart disease
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy
Right Sided Heart Failure

- Results due to failure of the right ventricle
- Often a consequence of LV systolic dysfunction
- Can be secondary to pulmonary disease (i.e. pulmonary hypertension in which the right ventricle will hypertrophy, resulting in cor pulmonale)
Right Sided Heart Failure (cont.)

- Other causes:
  - RV infarction
  - chronic severe tricuspid regurgitation
  - arrhythmogenic RV dysplasia

- Both sides of the heart may fail simultaneously, as with cardiomyopathies of congenital, viral, or alcoholic origin.
Acute vs. Chronic Heart Failure

- **Acute HF:**
  - Develops rapidly (hours/days)
  - Can be immediately life threatening
  - Dramatic drop in cardiac output
  - May be new (e.g. acute MI, sepsis) or an exacerbation of chronic disease
Acute vs. Chronic Heart Failure (cont.)

- Chronic HF
  - Long-term condition (months/years)
  - More insidious
  - Associated with the heart undergoing adaptive responses (e.g. dilation, hypetrophy) to a precipitating cause
Compensatory Mechanisms

- Neurohormonal Activation
- Frank-Starling Mechanism
- Ventricular Remodeling
Neurohormonal Activation

- Cardiac dysfunction causes drop in cardiac output (CO).
- The fall in CO leads to activation of several neurohormonal pathways.
- Although these neurohormonal pathways initially compensatory and beneficial, eventually they are deleterious.
Neurohormonal Response

- Major “players”:
  - Sympathetic Nervous System (SNS)
  - Renin – Angiotensin – Aldosterone System (RAAS)
  - Antidiuretic hormone (Vasopressin)
Sympathetic Nervous System

- One of the first responses to a decreased cardiac output
- Increase in circulating catecholamines (e.g. Norepinephrine)
- Augmentation of ventricular contractility and heart rate
- Systemic and pulmonary vasoconstriction
- Stimulates secretion of renin from juxtaglomerular apparatus of the kidney
Sympathetic Nervous System (cont.)

- Catecholamines aggravate ischemia, potentiate arrhythmias, promote cardiac remodeling and are directly toxic to myocytes.
Renin – Angiotensin - Aldosterone System (RAAS)

- **Renin**
  - enzyme released by the kidneys
  - stimulates formation of Angiotensin

- **Angiotensin**
  - potent vasoconstrictor
  - stimulates sodium reabsorption
  - stimulates release of norepinephrine
  - promotes synthesis and secretion of aldosterone
RAAS (cont.)

- **Aldosterone:**
  - potent mineralocorticoid
  - secreted by the adrenal gland
  - enhances fluid retention by increasing sodium reuptake
  - potentiates vasoconstriction
Angiotensinogen → Angiotensin I → Angiotensin II

Angiotensin II

- AT1 receptor
- AT2 receptor

Aldosterone

RENIN

ACE
RAAS: Final Points

- RAAS is activated as a result of increased sympathetic stimulation and decreased renal perfusion
- Results in:
  - further arteriolar vasoconstriction
  - sodium and water retention
  - release of aldosterone
RAAS: Final Points

- Increased **aldosterone** level leads to:
  - sodium and water retention
  - endothelial dysfunction
  - organ fibrosis
Antidiuretic Hormone (ADH)

- Also known as Vasopressin
- Released from hypothalamus
- Triggered by low cardiac output
- Additionally stimulated by Angiotensin II
ADH in Action

- Increased thirst
- Enhances water reabsorption by the kidneys
- Promotes water retention
- Leads to a fall in plasma sodium concentration
# Neurohormonal Responses to Impaired Cardiac Performance

Initially Adaptive, Deleterious if Sustained

<table>
<thead>
<tr>
<th>Response</th>
<th>Short-Term Effects</th>
<th>Long-Term Effects</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 BP for perfusion of vital organs</td>
<td>Exacerbates pump dysfunction (excessive afterload), increases cardiac energy expenditure</td>
</tr>
<tr>
<td>Sympathetic Stimulation</td>
<td>Increases HR and ejection</td>
<td>Increases energy expenditure</td>
</tr>
</tbody>
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Jaski, B, MD: Basics of Heart Failure: A Problem Solving Approach
Compensatory Mechanisms

Frank-Starling Mechanism

- The ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return.

- In heart failure, there is a compensatory increase in venous return which is augmented by neurohormonal mechanisms.

- Due to increase in venous return, there is a temporary increase in stroke volume.
Compensatory Mechanisms

Ventricular Remodeling

- Alterations in the heart’s size, shape, structure and function

What about the patient?

- Ventricular dysfunction limits a patient's ability to perform the routine activities of daily living...
Clinical Manifestations

- Weight gain
- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea
- Peripheral edema
- JVD
- Tachycardia

- Hepatosplenomegaly
- Ascites
- Fatigue
- Weakness
- Nausea
- Poor appetite
- Cachexia
- Renal hypoperfusion
## NYHA Classification System

<table>
<thead>
<tr>
<th>Class</th>
<th>Official Definition</th>
<th>In Everyday Terms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No limitation of functional activity or only at levels of exertion that would limit normal individuals</td>
<td>Can run up stairs</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation of activity. Dyspnea and fatigue with moderate exercise.</td>
<td>Can run up stairs but is out of breath when he/she gets there</td>
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<tr>
<td>III</td>
<td>Marked limitation of activity. Dyspnea with minimal activity.</td>
<td>Can walk up stairs if he/she can rest a couple of times on the way</td>
</tr>
<tr>
<td>IV</td>
<td>Severe limitation of activity. Symptoms even at rest.</td>
<td>Cannot climb stairs at all</td>
</tr>
</tbody>
</table>
Physical Exam

- Tachycardia
- Tachypnea
- Abnormal lung exam (e.g. inspiratory rales)
- S3 gallop
- Jugular venous distention
- Peripheral Edema
- Hepatojugular Reflux
- Hepatomegaly
Summary

- Heart failure is a complex clinical syndrome, regardless of the precipitating event.
- There are several compensatory mechanisms involved.
- Only when the network of adaptations becomes overwhelmed does heart failure ensue.
- Neurohormonal responses are initially adaptive, deleterious if sustained.
The signs and symptoms of HF are due in part to compensatory mechanisms utilized by the body in attempt to adjust for a primary deficit in cardiac output.

Neurohormonal modulation is the basis for modern treatment of HF.