WHAT IS HEART FAILURE?
PATHOPHYSIOLOGY AND SYMPTOMS

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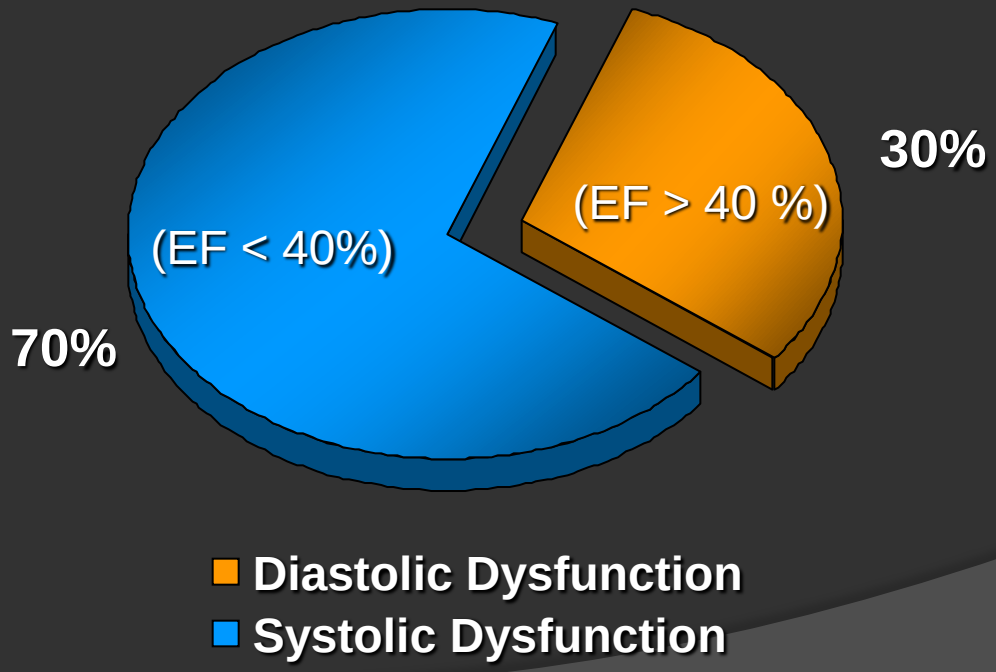
- Heart Failure (HF) is a complex clinical syndrome that can result from any functional or structural cardiac disorder that impairs the ventricle's ability to fill with or eject blood

- 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 dysfunction1
- 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.)

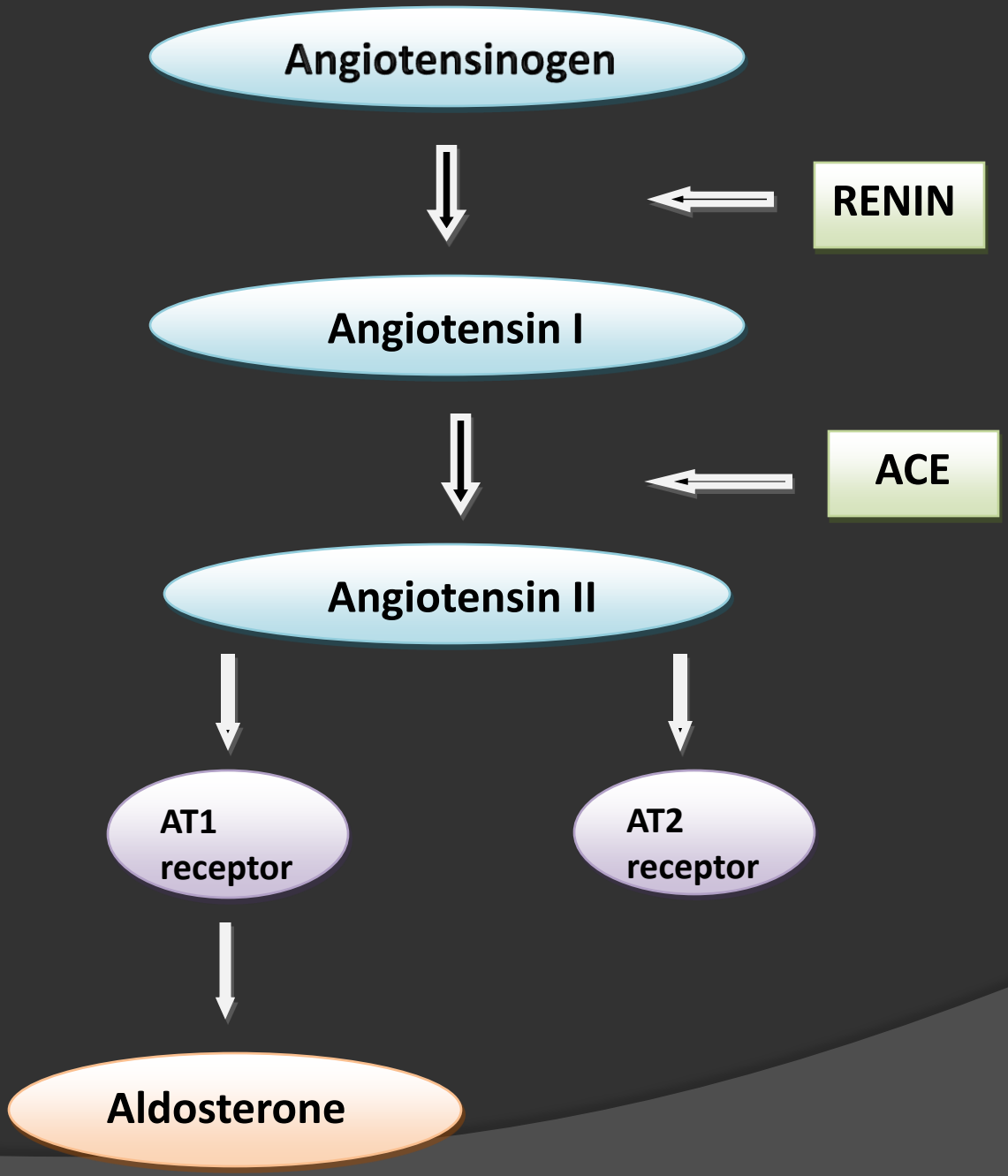
- Catecholamines aggravate ischemia, potentiate arrhythmias, promote cardiac remodeling and are directly toxic to myocytes.

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
RENIN
Angiotensin I
ACE
Angiotensin II
AT1 receptor
AT2 receptor
Aldosterone

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 Agiotensin 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

Response	Short-Term Effects	Long-Term Effects
Salt and Water Retention	Augments Preload	Pulmonary Congestion, Anasarca
Vasoconstriction	Maintains BP for perfusion of vital organs	Exacerbates pump dysfunction (excessive afterload), increases cardiac energy expenditure
Sympathetic Stimulation	Increases HR and ejection	Increases energy expenditure

Jaski, B, MD: Basics of Heart Failure: A Problem Solving Approach

Compensatory Mechanisms

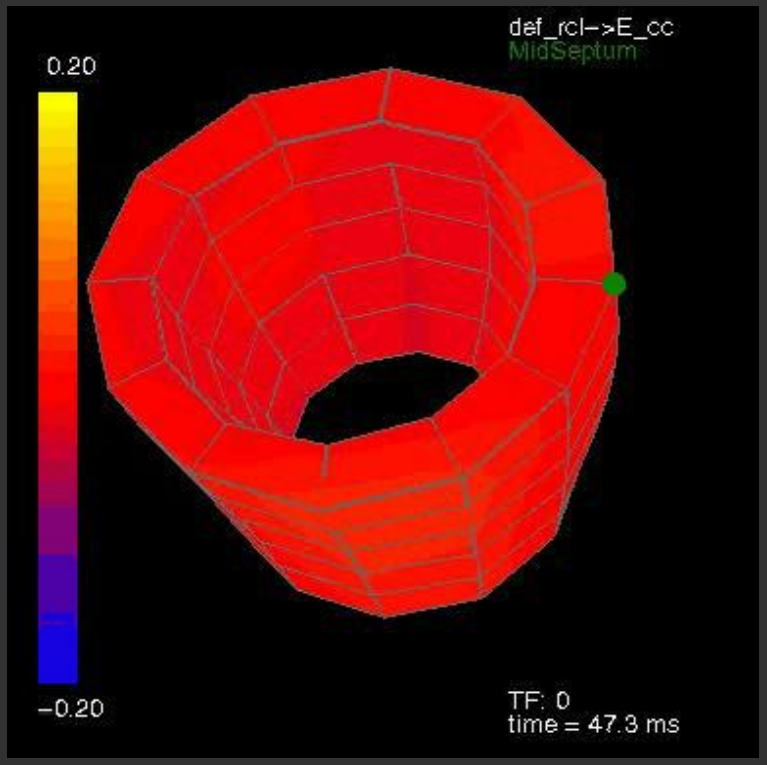
Frank-Starling Mechanism

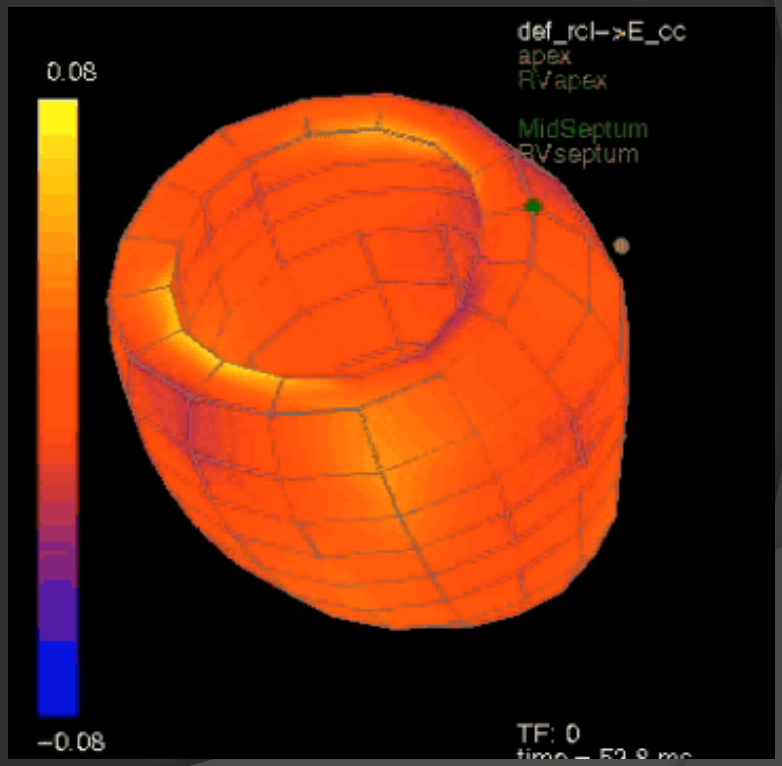
- The ability of the heart to change its force of contraction and therefore stroke volume in response to changes in venous return.
- In heart failure, there is a compensatory increase in venous return which is augmented by neurohormonal mechanisms.
- Due to increase in venous return, there is a temporary increase in stroke volume

Compensatory Mechanisms

Ventricular Remodeling

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





Curry CW, et al. Mechanical dyssynchrony in dilated cardiomyopathy with intraventricular conduction delay as depicted by 3D tagged magnetic resonance imaging. Circulation 2000 Jan 4;101(1):E2.

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

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

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 envolved.
- Only when the network of adaptations becomes overwhelmed does heart failure ensue
- Neurohormonal responses are initially adaptive, deleterious if sustained

Summary

- 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