Program:

Module 1:

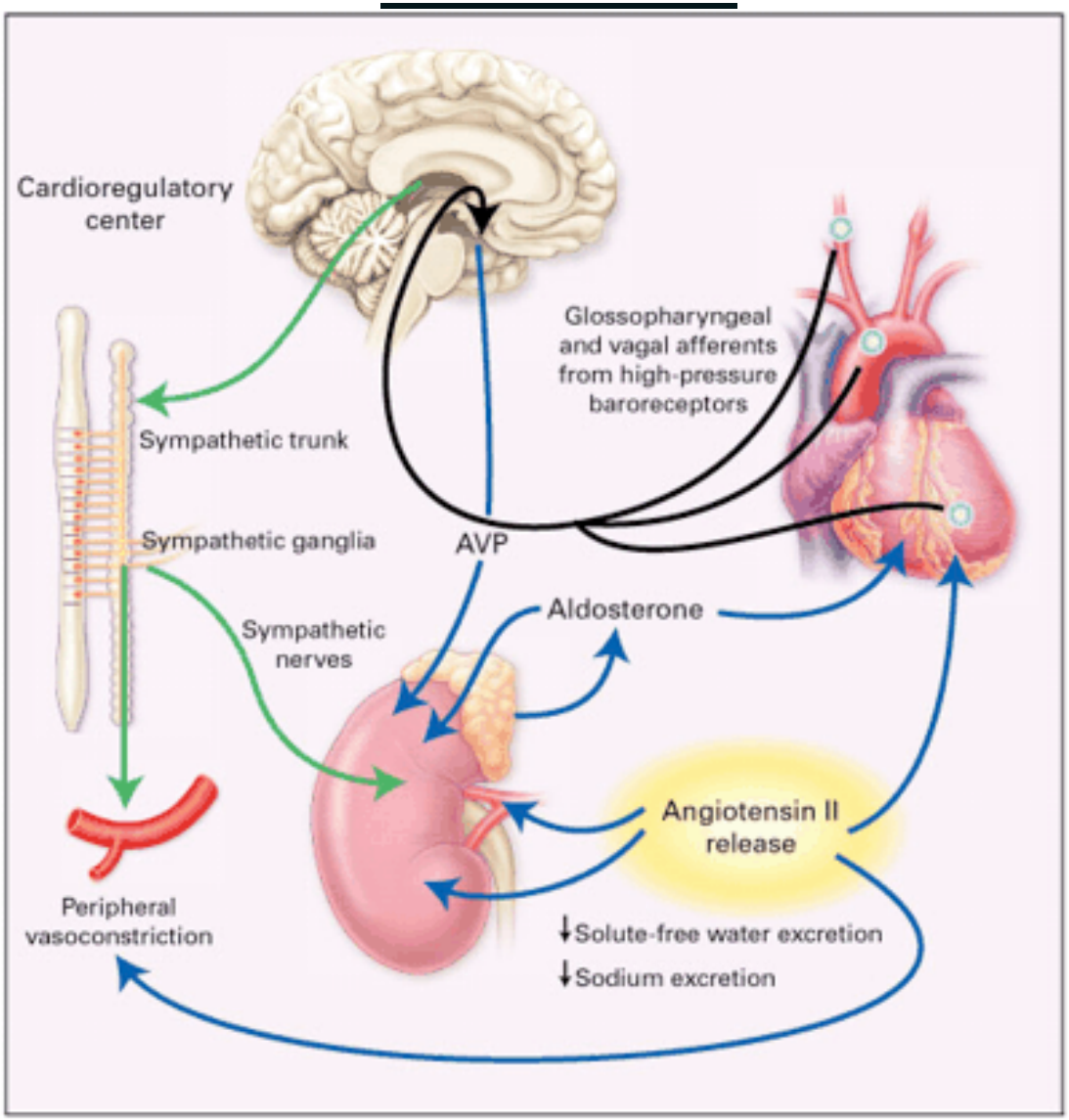
- Definition
- Epidemiology of Heart Failure
- Pathophysiology of Heart Failure
- Specific Diseases causing Heart Failure and practical case studies

Module 2:

- Diagnosis and Investigation of HF and Practical Case Studies
- Treatment of Heart Failure and Practical Case Studies

Heart Failure Society of South Africa [HeFSSA]

The Pathophysiology of Heart Failure.



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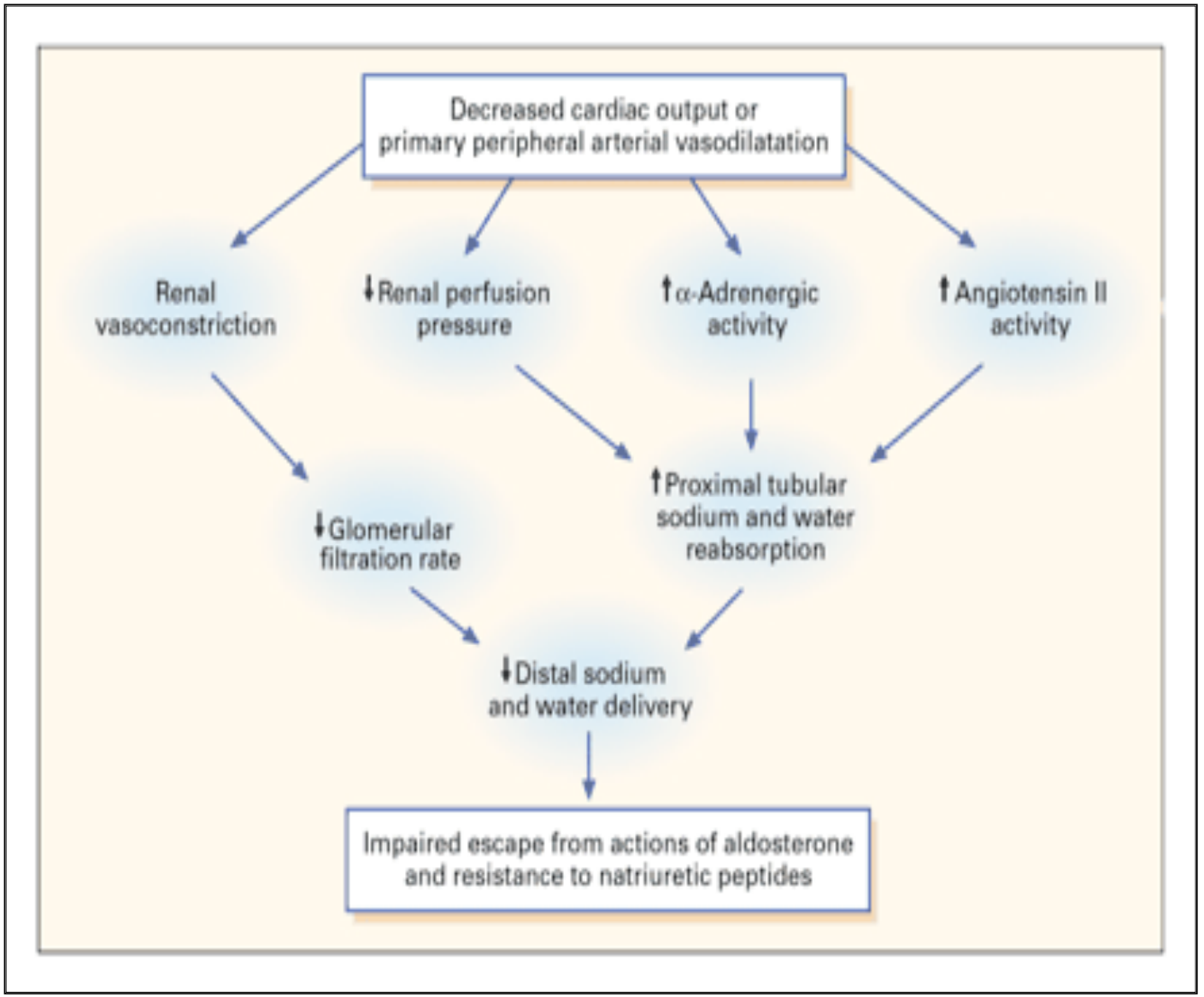
Mechanisms by Which Arterial Underfilling Leads to Diminished Distal Tubular Sodium and Water Delivery, Impaired Aldosterone Escape, and Resistance to Natriuretic Peptide Hormone.



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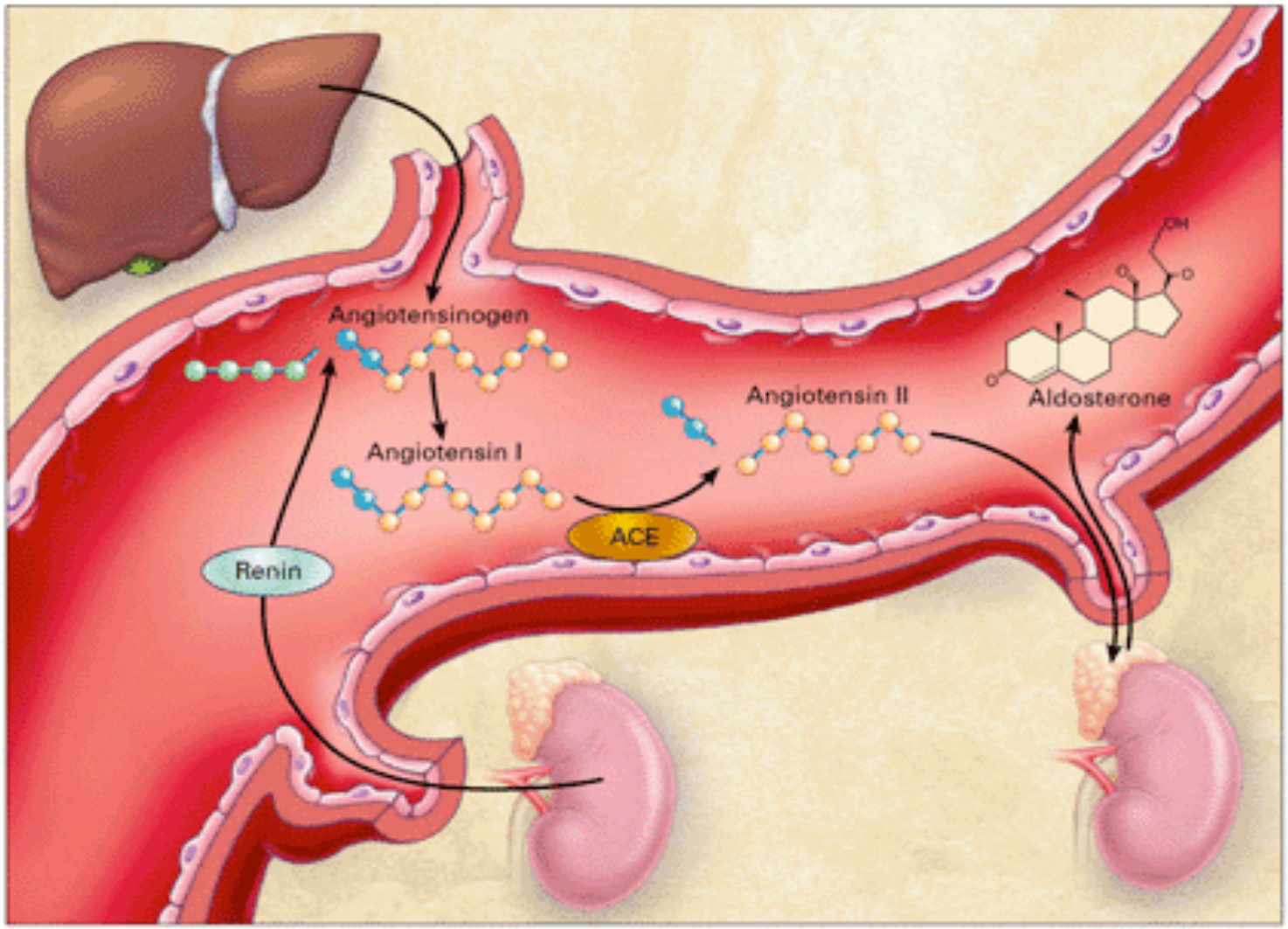
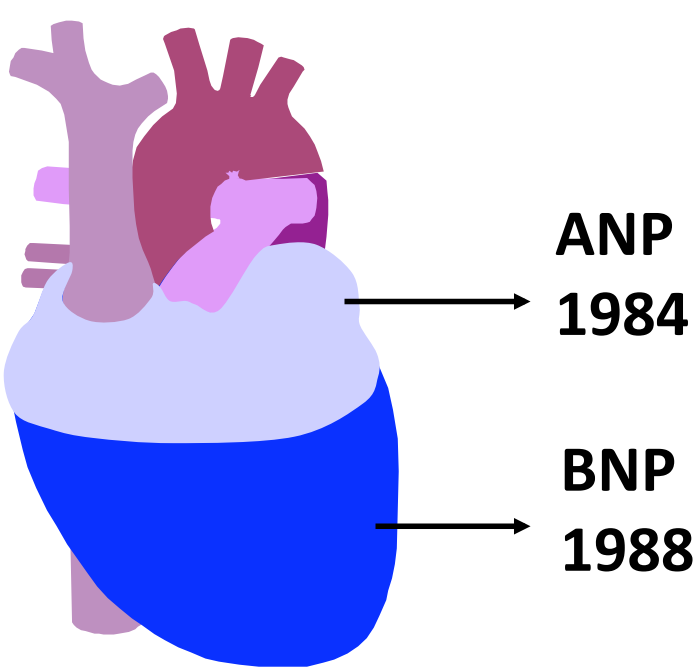
The Renin–Angiotensin –Aldosterone System



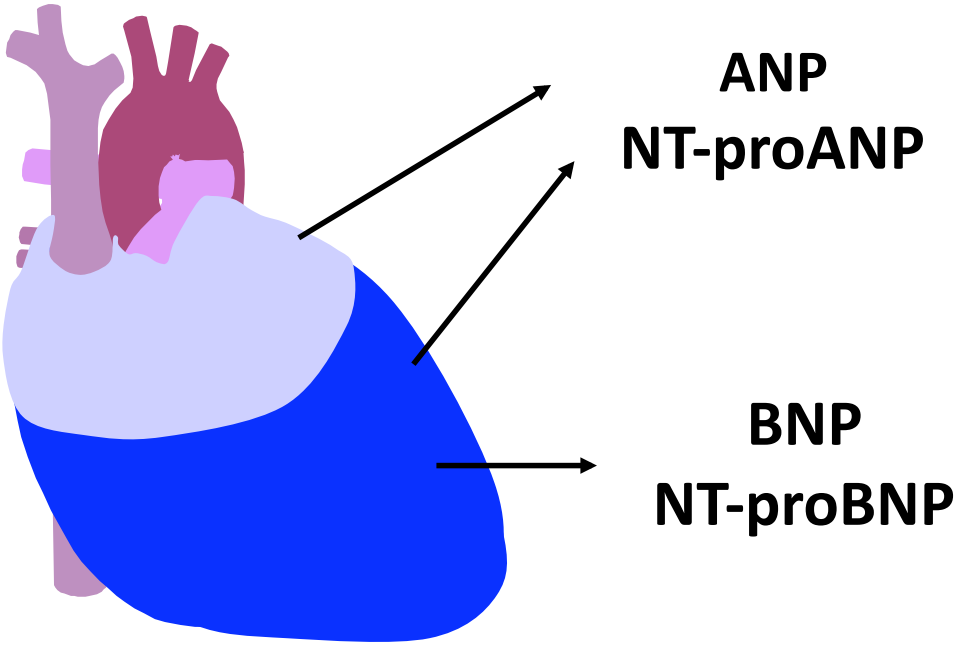
The natriuretic peptides

The heart - an endocrine organ

Normal heart



Heart failure



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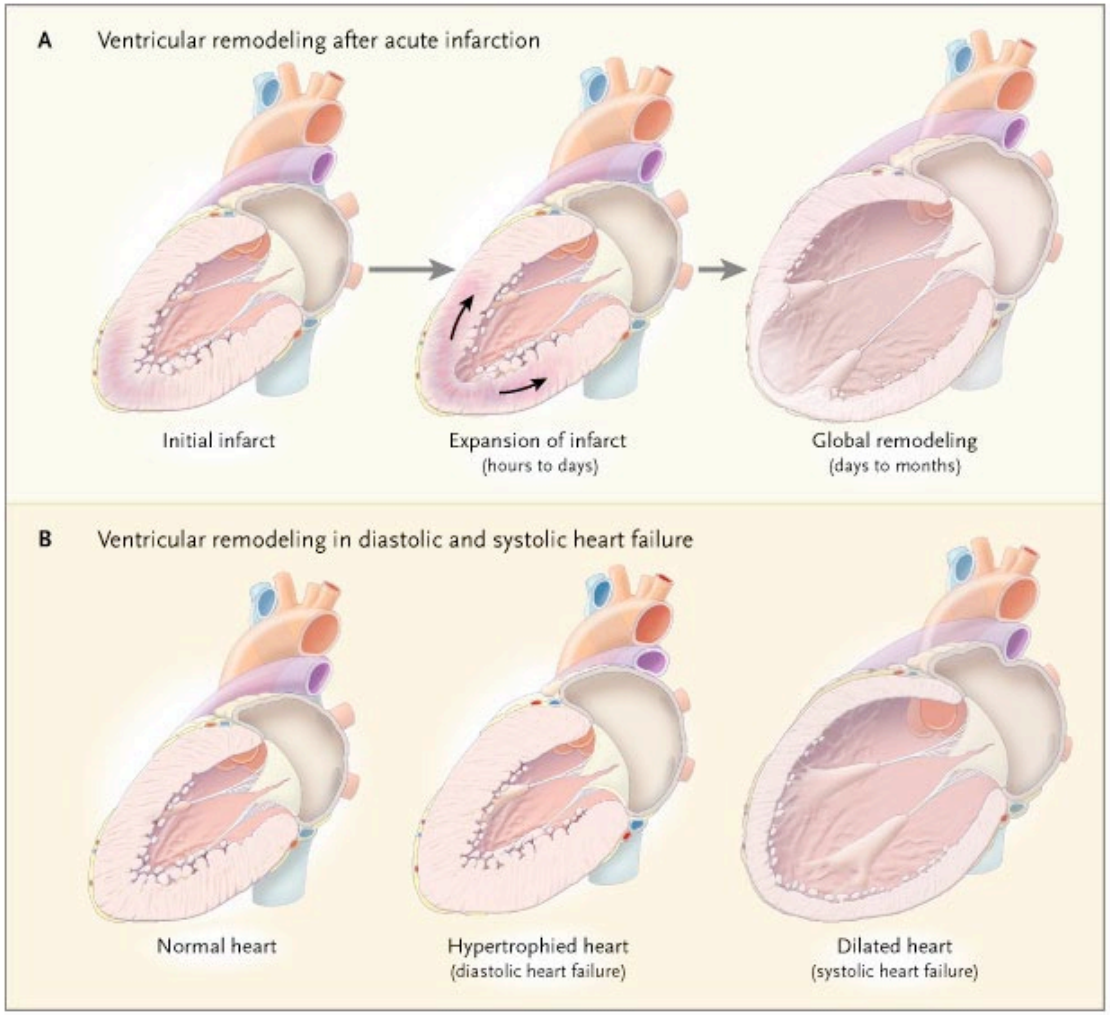
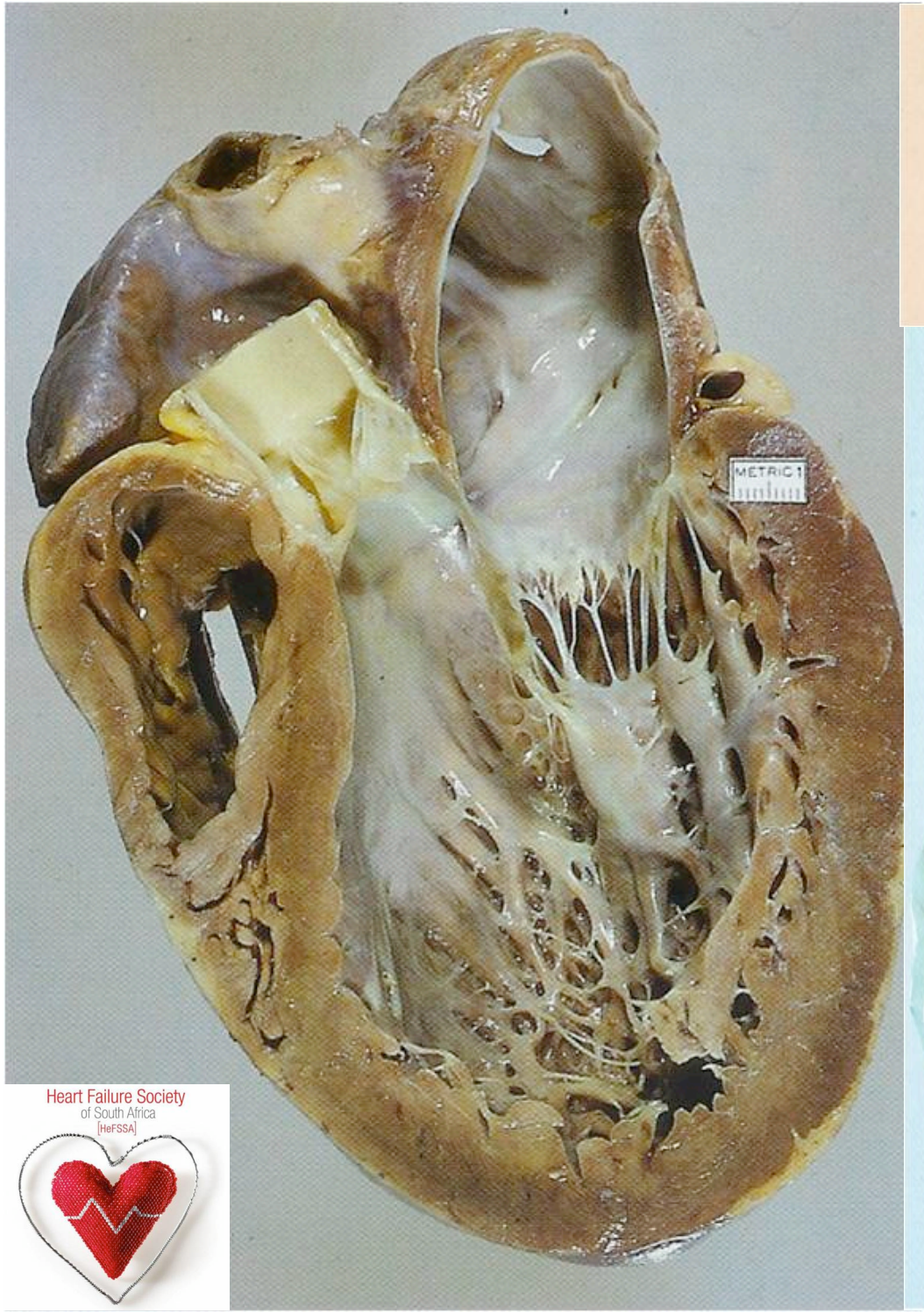
Ventricular Remodeling after Infarction (Panel A) and in Diastolic and Systolic Heart Failure (Panel B)



Jessup, M. et al. N Engl J Med 2003;348:2007-2018

The NEW ENGLAND JOURNAL of MEDICINE



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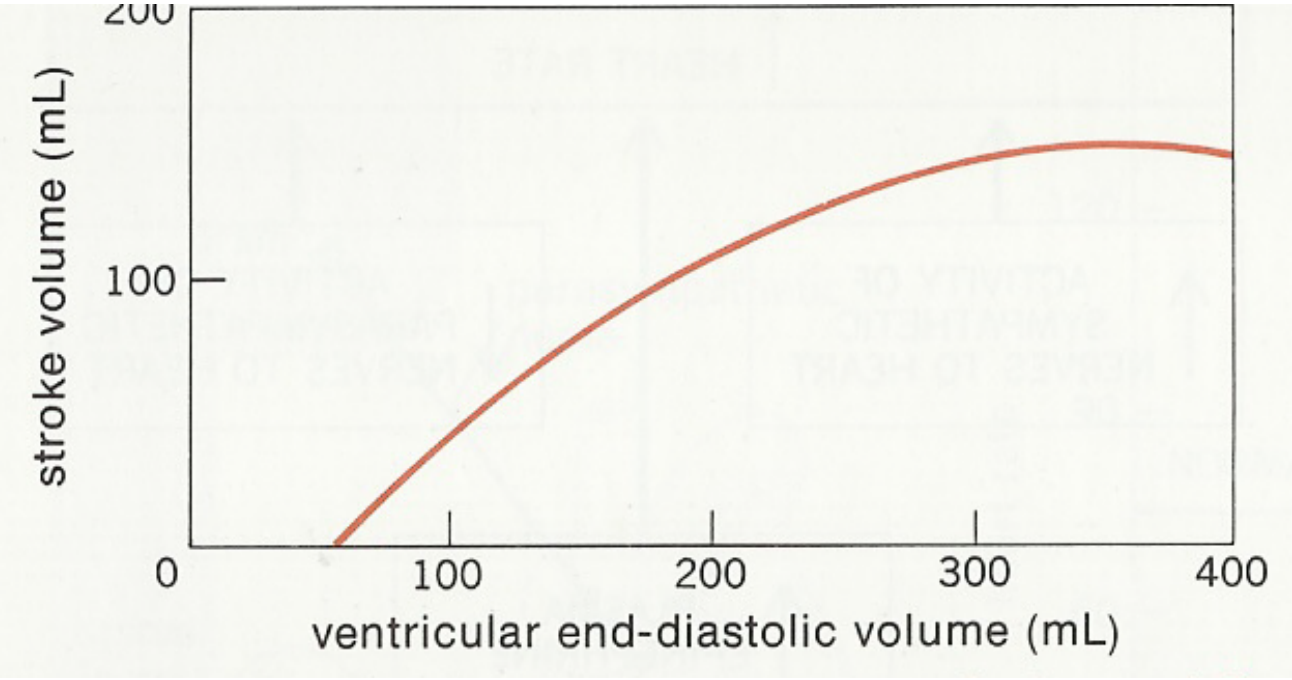
Starling's Law of the Heart

CONTRACTILE FORCE



SARCOMERE LENGTH

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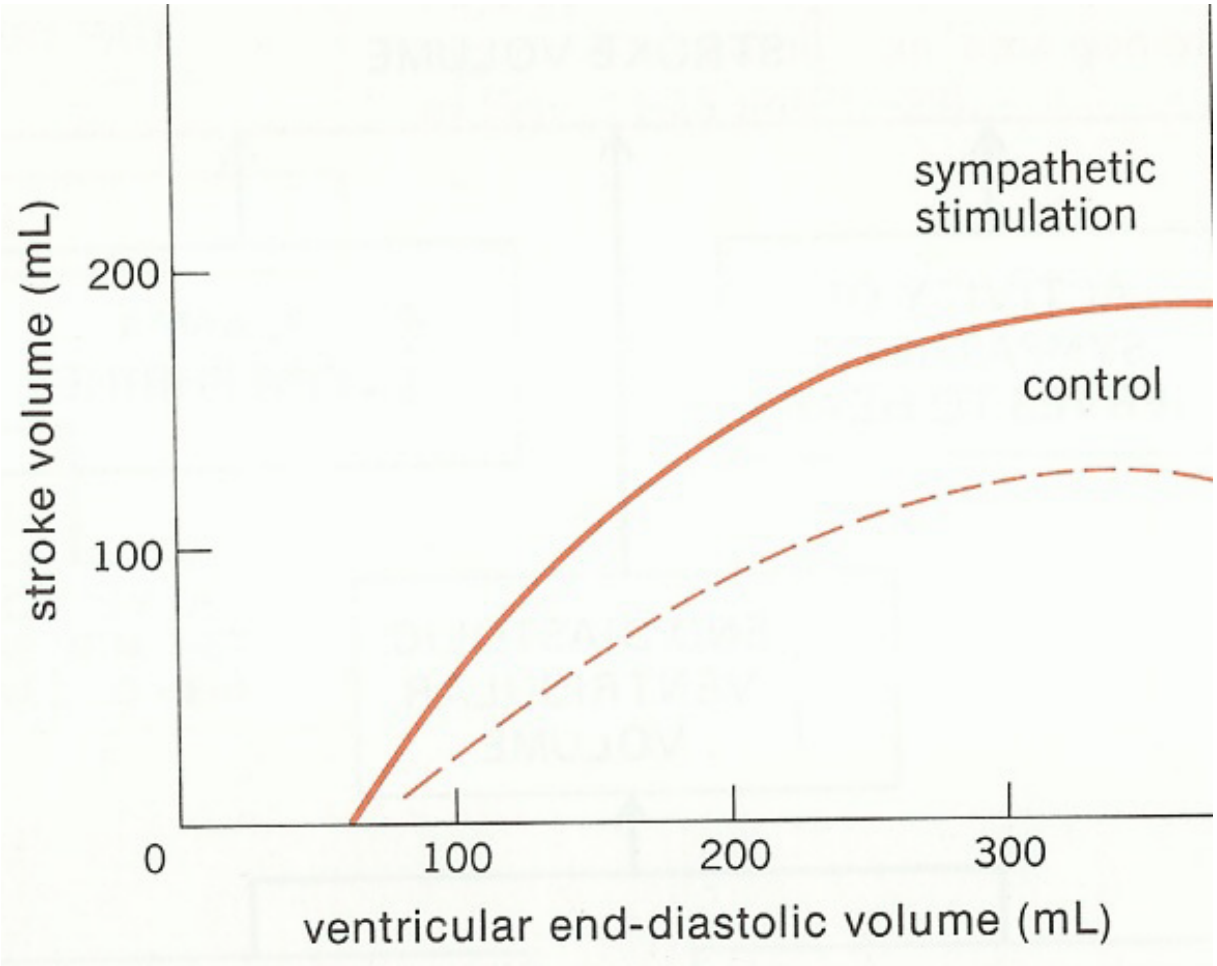
Effects of sympathetic stimulation



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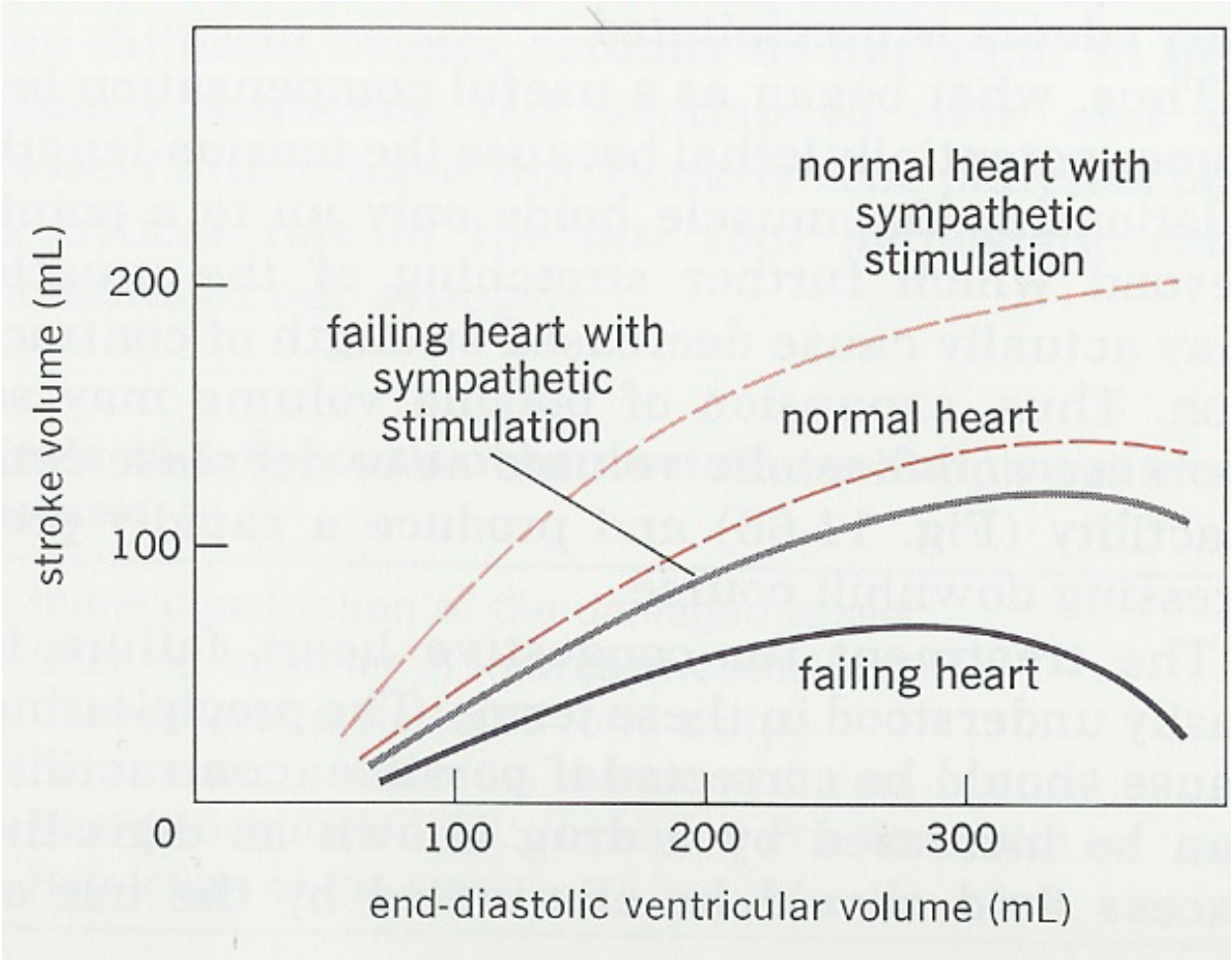
The Failing Heart



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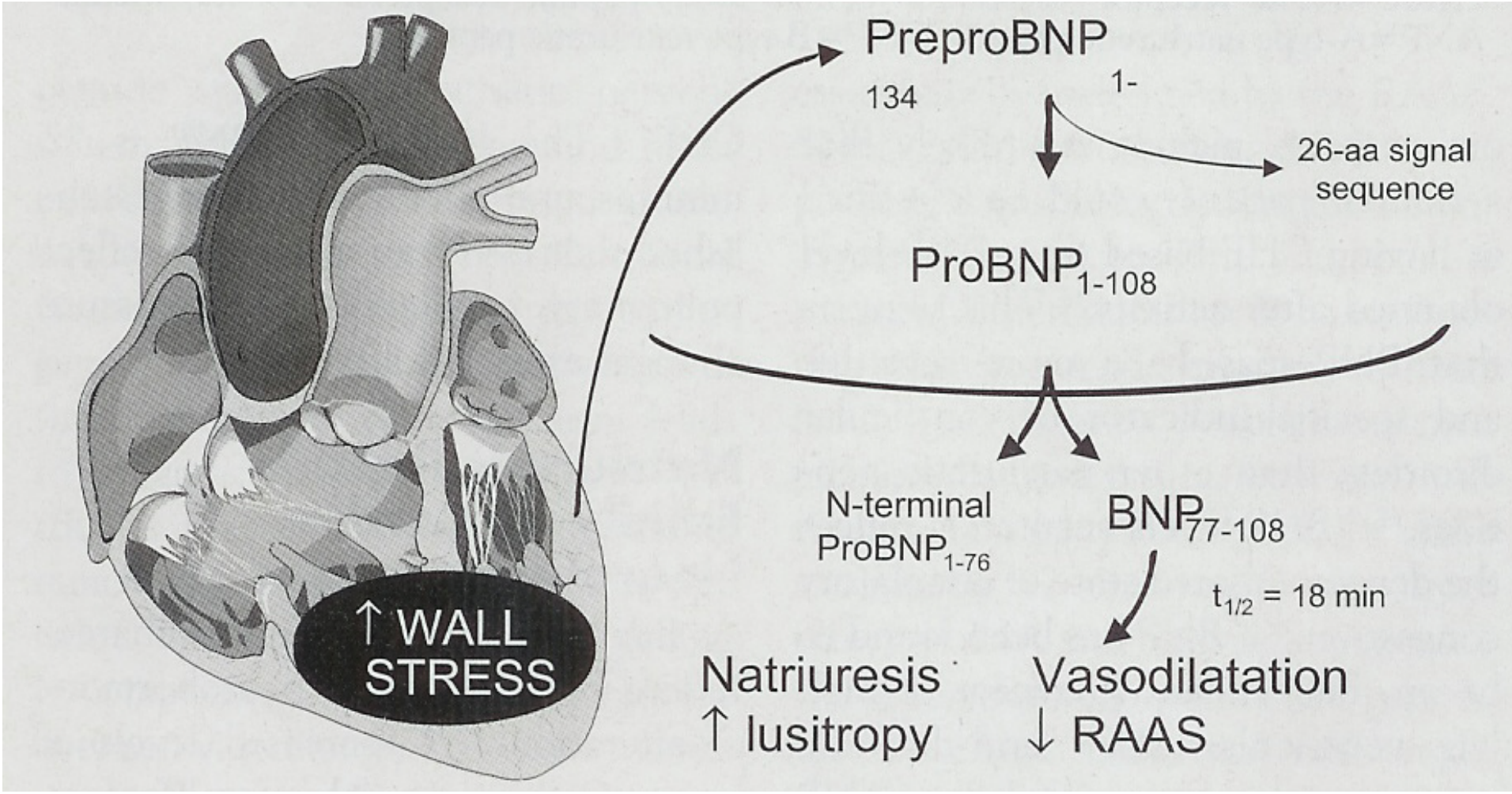
The Failing Heart



Determinants of Ventricular Function

Contractility

Preload

Afterload

Stroke Volume

- Synergistic LV Contraction
- Wall Integrity
- Valvular Competence

Heart Rate

Cardiac Output

Left Ventricular Systolic Dysfunction

Pressure Overload

Loss of Myocardium

Volume Overload

Impaired Contractility

LV Systolic Dysfunction
EF < 40%

↓ Cardiac Output

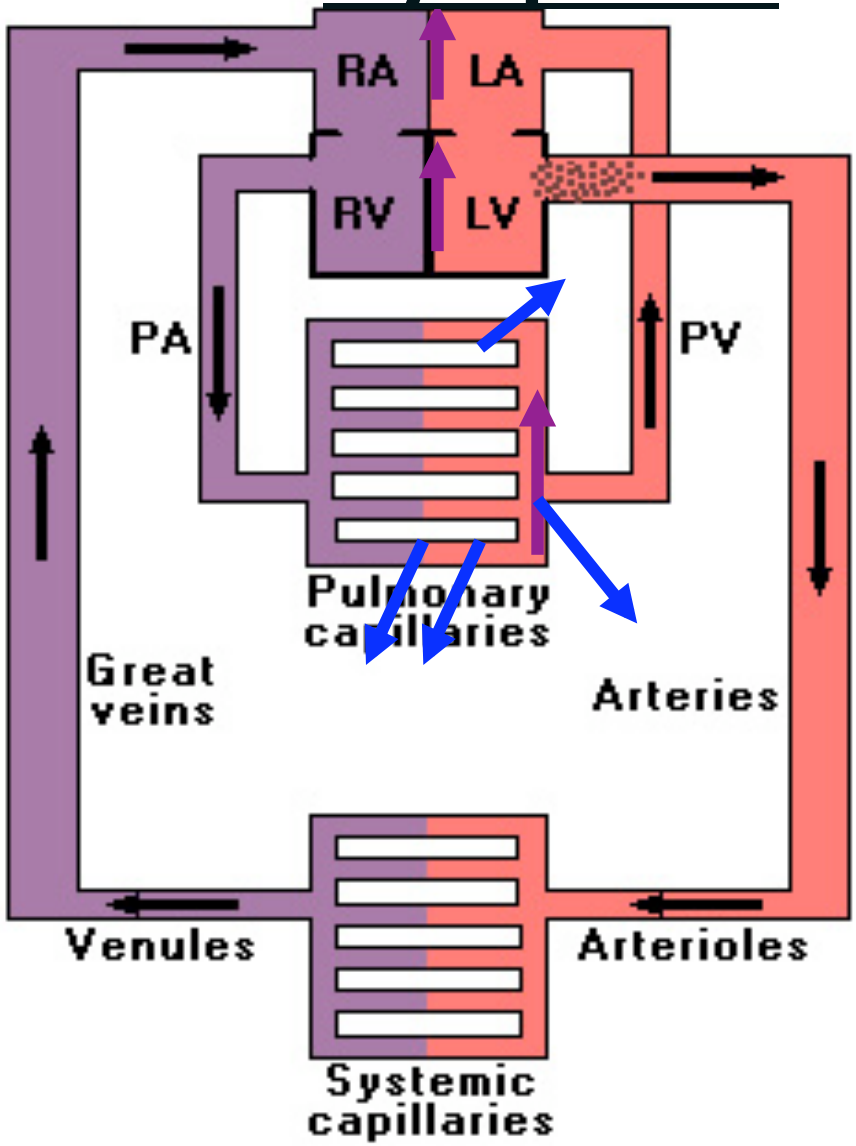
Hypoperfusion

↑ End Systolic Volume

↑ End Diastolic Volume

Pulmonary Congestion

Hemodynamic Basis for Heart Failure Symptoms



Hemodynamic Basis for Heart Failure Symptoms

LVEDP ↑

↓

Left Atrial Pressure ↑

↓

Pulmonary Capillary Pressure ↑

↓

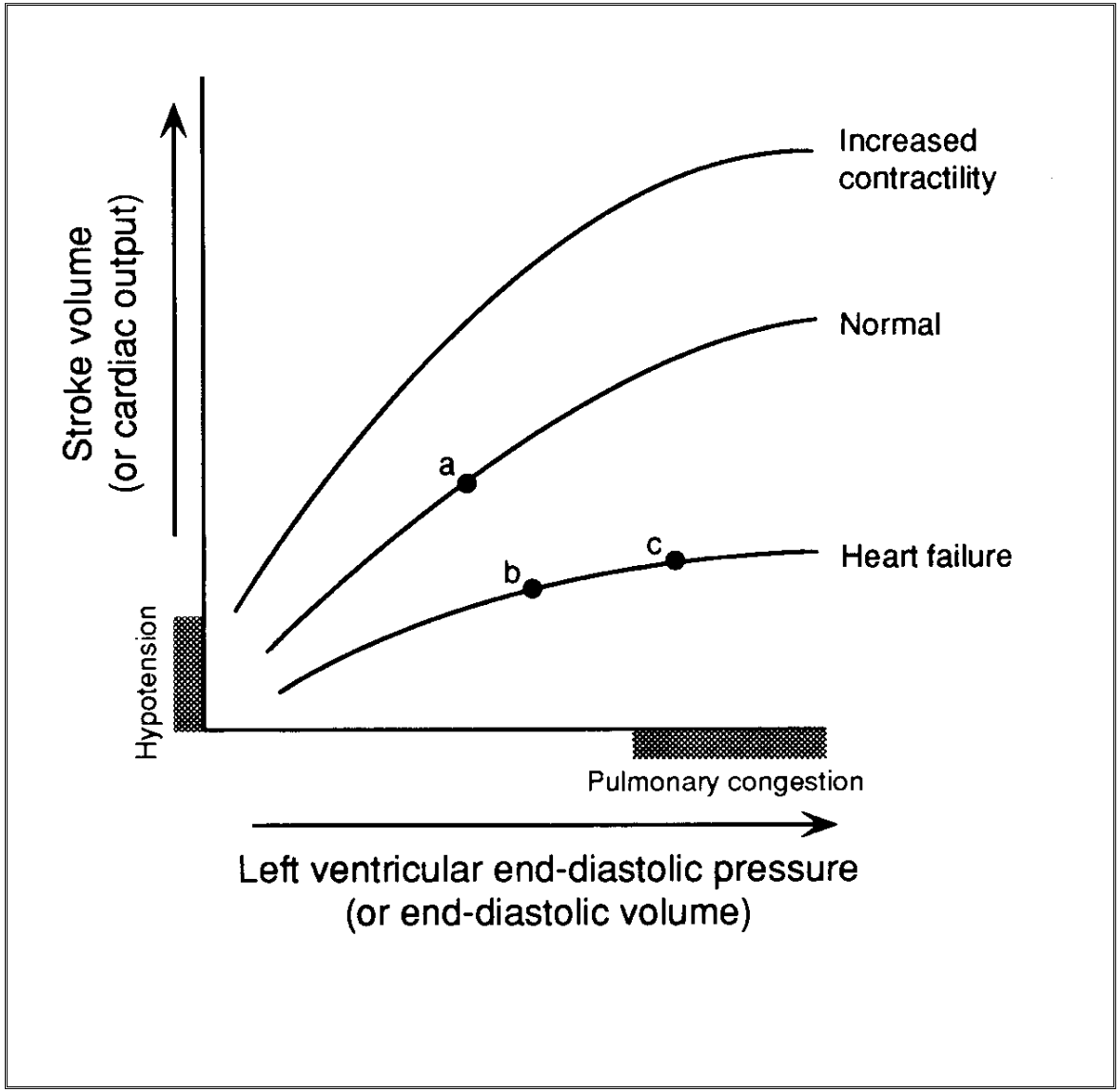
Pulmonary Congestion

Frank-Starling Mechanism

a. At rest, no HF

b. HF due to LV systolic dysfunction

c. Advanced HF



Compensatory Mechanisms

- Frank-Starling Mechanism
- Neurohormonal Activation
- Ventricular Remodeling

Neurohormonal Activation

Many different hormone systems are involved in maintaining normal cardiovascular homeostasis, including:

- Sympathetic nervous system (SNS)
- Renin-angiotensin-aldosterone system (RAAS)
- Vasopressin (a.k.a. antidiuretic hormone, ADH)

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 (body oedema)
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

Neurohormonal Compensatory Mechanisms: Summary

Decreased Cardiac Output

↑ Sympathetic nervous system

↑ Renin-angiotensin system

↑ Antidiuretic hormone (vasopressin)

↑ Contractility

↑ Heart rate

Vasoconstriction

↑ Circulating volume

Anteriolar

Venous

Maintain blood pressure

↑ Venous return to heart (↑ preload)

Peripheral edema and pulmonary congestion

Cardiac output

+

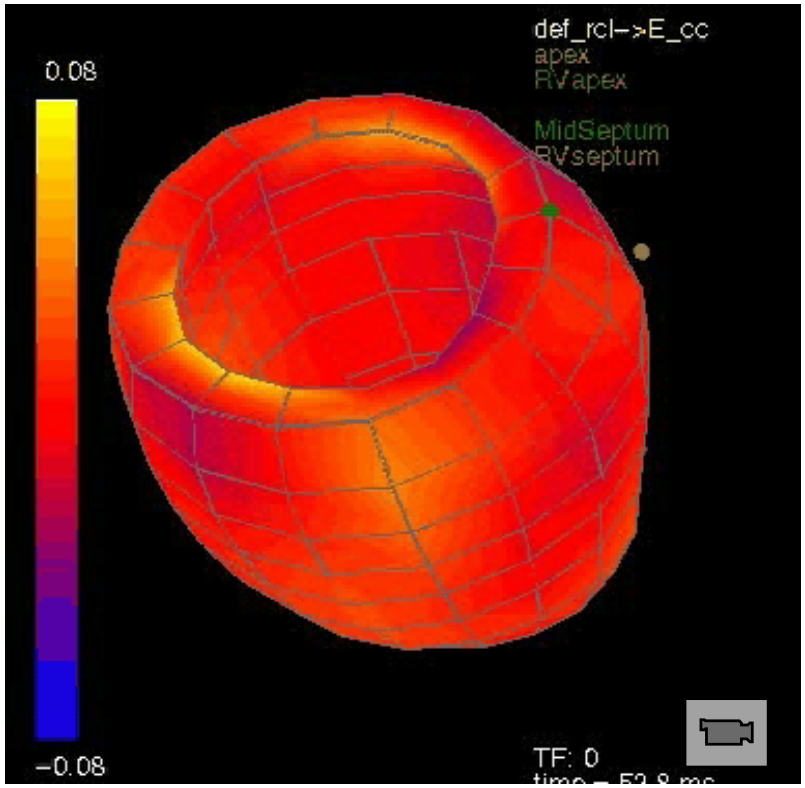
-

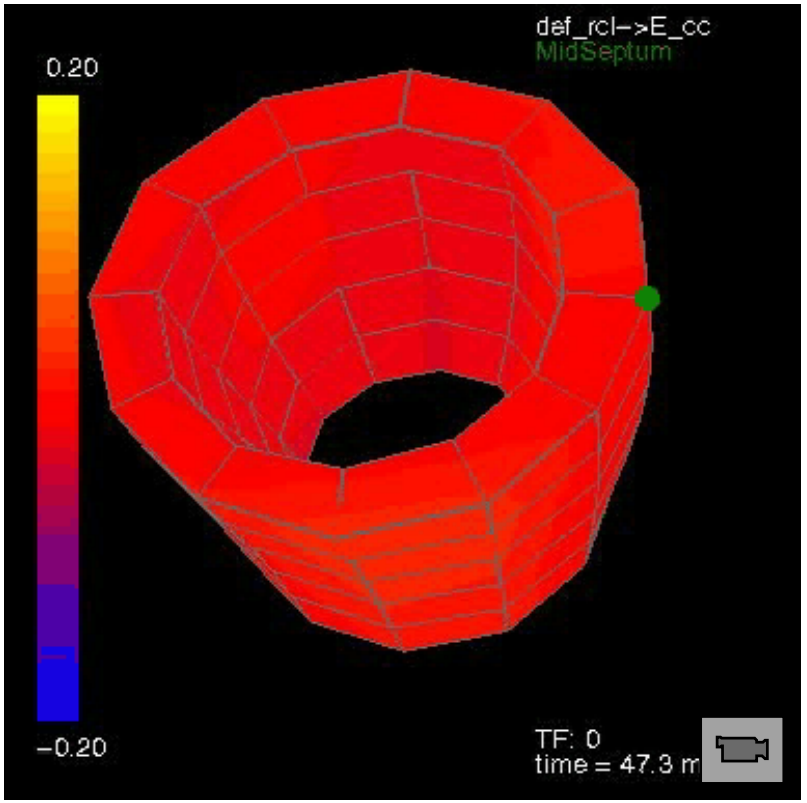
+

↑ Stroke volume

Ventricular Remodeling

Alterations in the heart's size, shape, structure, and function brought about by the chronic hemodynamic stresses experienced by the failing heart.





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.

Vicious Cycle of Heart Failure

LV Dysfunction

Decreased cardiac output
and
Decreased blood pressure

Frank-Starling mechanism
Neurohormonal activation
Ventricular remodeling

Increased cardiac output (via increased contractility and heart rate)
Increased blood pressure (via vasoconstriction and increased blood volume)

Increased cardiac workload
(increased preload and afterload)