# Heart Failure: Clinical Breakdown

*Pathophysiology, Ejection Fraction Classifications, Etiology, Neurohormonal Activation, and Therapeutic Management*

**Heart Failure (HF)** is a complex clinical syndrome resulting from any structural or functional impairment of ventricular filling or ejection of blood. It is characterized by hallmark symptoms such as dyspnea, fatigue, and fluid retention (peripheral edema and pulmonary congestion). Heart failure represents the final common pathway for numerous cardiovascular diseases.

> **Core Clinical Principle:** Heart failure is classified based on left ventricular ejection fraction (LEF): Heart Failure with Reduced Ejection Fraction (HFrEF, LVEF ≤ 40%) versus Heart Failure with Preserved Ejection Fraction (HFpEF, LVEF ≥ 50%), each requiring distinct diagnostic and therapeutic strategies.

## 1. Classification by Left Ventricular Ejection Fraction (LVEF)

Standardized clinical criteria categorize heart failure phenotypes according to systolic and diastolic performance.

| HF Phenotype | Ejection Fraction (LVEF) | Primary Pathophysiological Defect |
|---|---|---|
| **HFrEF** (Reduced EF) | 40% or lower | Systolic dysfunction; impaired myocardial contractility and pump output. |
| **HFmrEF** (Mildly Reduced EF) | 41% – 49% | Intermediate phenotype with mild systolic and diastolic impairment. |
| **HFpEF** (Preserved EF) | 50% or higher | Diastolic dysfunction; impaired ventricular relaxation and stiffened filling. |

## 2. Primary Etiology & Neurohormonal Activation

Understanding the underlying triggers and compensatory mechanisms explains the progressive nature of heart failure.

- **Primary Etiologies:** Coronary artery disease (myocardial infarction), chronic hypertension, valvular heart disease, cardiomyopathies, and diabetes mellitus are leading structural causes.
- **Neurohormonal Compensation:** Decreased cardiac output triggers compensatory activation of the sympathetic nervous system and the Renin-Angiotensin-Aldosterone System (RAAS). While initially maintaining perfusion, chronic neurohormonal overactivation drives adverse myocardial remodeling, vasoconstriction, and fluid retention.

## 3. Clinical Presentation & Management Principles

Recognizing systemic congestion and implementing guideline-directed medical therapy improves survival and quality of life.

- **Symptoms & Signs:** Manifests as exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, peripheral pitting edema, elevated jugular venous pressure (JVP), and fatigue.
- **Therapeutic Management:** Combines lifestyle modifications (sodium restriction, fluid monitoring) with guideline-directed medical therapy (GDMT), including ACE inhibitors/ARBs/ARNI therapies, beta-blockers, mineralocorticoid receptor antagonists (MRAs), and SGLT2 inhibitors.