

CHRONIC HEART FAILURE: MODERN DIAGNOSTIC AND THERAPEUTIC  
APPROACHES

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**Abstract:** Chronic heart failure (CHF) is a complex clinical syndrome characterized by the heart's persistent inability to pump blood adequately to meet the body's demands or to receive blood effectively. This article provides a comprehensive scientific overview of CHF's pathophysiology, modern diagnostic methods, and treatment strategies. The pathogenesis of CHF involves myocardial systolic or diastolic dysfunction, neurohormonal activation, inflammatory processes, and oxidative stress. Diagnostic approaches emphasize the role of echocardiography, natriuretic peptides (BNP, NT-proBNP), and cardiac magnetic resonance imaging (MRI) in accurate assessment. Treatment strategies include pharmacological interventions such as angiotensin receptor-neprilysin inhibitors (ARNI), sodium-glucose cotransporter-2 inhibitors (SGLT2i), beta-blockers, and mineralocorticoid receptor antagonists, alongside device-based therapies like implantable cardioverter-defibrillators (ICD) and cardiac resynchronization therapy (CRT). Lifestyle modifications and cardiac rehabilitation are integral to improving patients' functional status. Emerging research on gene and cell therapies, as well as artificial intelligence-based models, offers promising future directions. The article underscores the importance of early diagnosis and individualized treatment to enhance patient outcomes and quality of life.

**Keywords:** Chronic heart failure, pathophysiology, echocardiography, natriuretic peptides, ARNI, SGLT2 inhibitors, beta-blockers, implantable cardioverter-defibrillator, cardiac resynchronization therapy, gene therapy, cell therapy, cardiac rehabilitation.

Chronic heart failure (CHF) remains a significant global health challenge, affecting millions and placing a substantial burden on healthcare systems. It arises from the heart's impaired ability to pump or fill with blood, often secondary to conditions such as arterial hypertension, coronary artery disease, myocardial infarction, cardiomyopathies, or valvular defects. The pathophysiology of CHF is multifaceted, involving myocardial dysfunction, neurohormonal activation, inflammation, and oxidative stress. Primary cardiac insults, such as myocardial infarction, lead to necrosis of cardiac tissue, reducing left ventricular contractility. Chronic conditions like hypertension or aortic stenosis impose sustained pressure overload, causing myocardial hypertrophy and fibrosis. Activation of the renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system exacerbates ventricular remodeling, while inflammatory mediators (e.g., IL-6, TNF- $\alpha$ ) and oxidative stress contribute to myocyte apoptosis and fibrosis. Recent studies highlight mitochondrial dysfunction and impaired energy metabolism as critical contributors, opening new therapeutic avenues.

Diagnosis of CHF relies on a combination of clinical evaluation, laboratory tests, and instrumental methods. Common symptoms include dyspnea, fatigue, peripheral edema, orthopnea, and paroxysmal nocturnal dyspnea. The New York Heart Association (NYHA) classification (I–IV) is widely used to assess functional status. Laboratory tests focus on natriuretic peptides, with NT-proBNP levels above 125 pg/mL indicating a high likelihood of CHF, serving as a key biomarker for diagnosis and

prognosis. Additional tests, such as electrolytes, renal, and liver function, are essential due to their frequent involvement in CHF. Transthoracic echocardiography (EchoCG) remains the gold standard, evaluating left ventricular ejection fraction (LVEF), wall motion, valvular function, and diastolic performance. CHF is categorized into three phenotypes: heart failure with reduced ejection fraction (HFrEF, LVEF <40%), mid-range ejection fraction (HFmrEF, LVEF 40–49%), and preserved ejection fraction (HFpEF, LVEF ≥50%). HFpEF, prevalent in older populations, is diagnosed based on diastolic dysfunction markers. Cardiac MRI is increasingly used to assess myocardial fibrosis and differentiate cardiomyopathies. Cardiopulmonary stress testing evaluates functional capacity, while invasive methods like cardiac catheterization aid in assessing coronary artery status. Artificial intelligence (AI)-based algorithms are emerging as tools to enhance early diagnosis and predict outcomes by analyzing imaging and biomarker data.

Treatment aims to alleviate symptoms, slow disease progression, reduce cardiovascular events, and improve quality of life. Pharmacological therapy forms the cornerstone of CHF management. Angiotensin-converting enzyme inhibitors (ACEi, e.g., enalapril) and angiotensin receptor blockers (ARB, e.g., losartan) inhibit RAAS, reducing blood pressure and myocardial remodeling. Angiotensin receptor-neprilysin inhibitors (ARNI, e.g., sacubitril/valsartan) have surpassed ACEi/ARB in efficacy, with the PARADIGM-HF trial demonstrating a 20–25% reduction in cardiovascular death and hospitalization in HFrEF patients. Beta-blockers (e.g., bisoprolol, metoprolol, carvedilol) mitigate sympathetic overactivity, controlling heart rate and reducing arrhythmia risk. Mineralocorticoid receptor antagonists (MRA, e.g., spironolactone, eplerenone) counteract aldosterone's effects, slowing fibrosis. Sodium-glucose cotransporter-2 inhibitors (SGLT2i, e.g., dapagliflozin, empagliflozin) have revolutionized CHF treatment, with trials like DAPA-HF and EMPEROR-Reduced showing significant reductions in cardiovascular events in both HFrEF and HFpEF. These agents exert diuretic effects, reduce edema, and improve insulin sensitivity, benefiting patients with metabolic comorbidities.

Device-based therapies are critical for severe CHF. Implantable cardioverter-defibrillators (ICD) prevent sudden cardiac death in high-risk patients, while cardiac resynchronization therapy (CRT) improves ventricular synchrony in HFrEF, enhancing functional capacity. Transcatheter aortic valve implantation (TAVI) is a minimally invasive option for aortic stenosis-related CHF. Left ventricular assist devices (LVADs) serve as bridge-to-transplant or destination therapy in advanced cases. Lifestyle modifications are vital, including sodium restriction (<2 g/day), increased physical activity (150 minutes/week of moderate aerobic exercise), and cessation of smoking and alcohol. Cardiac rehabilitation programs improve physical endurance and psychological well-being, addressing the impact of stress and depression on disease progression.

Emerging research explores innovative approaches. Gene therapy, such as targeting SERCA2a to enhance myocardial calcium handling, shows promise in restoring contractility. Cell-based therapies, including stem cells and induced pluripotent stem cells (iPSCs), aim to regenerate myocardium but remain experimental. AI-driven models are being integrated into clinical practice to optimize diagnosis and personalize treatment. These advancements hold potential to transform CHF management in the future.

In conclusion, chronic heart failure remains a complex condition, but advances in diagnostics and therapeutics have significantly improved outcomes. Echocardiography, natriuretic peptides, and novel pharmacological agents like ARNI and SGLT2i, combined with device-based therapies and lifestyle interventions, form the backbone of effective management. Future innovations in gene, cell,

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and AI-based therapies may further revolutionize treatment. Early diagnosis and individualized care are critical to preventing disease progression and enhancing patient quality of life.

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