An Overview of Chronic Heart

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Abstract- Chronic heart failure (CHF) remains the only disease with an increasing hospitalization burden and an ongoing drain on health care expenditures. The prevalence of CHF increases with advancing life span, with diastolic heart failure predominating in the elderly population. Primary prevention of coronary artery disease and risk factor management via aggressive blood pressure control are central in preventing new occurrences of left ventricular dysfunction. Optimal therapy for CHF involves identification and correction of potentially reversible precipitants, target-dose titration of medical therapy, and management of hospitalizations for decompensation. The etiological phenotype, absolute decrease in left ventricular ejection fraction and a widening of QRS duration on electrocardiography, is commonly used to identify patients at increased risk of progression of heart failure and sudden death who may benefit from prophylactic implantable cardioverter-defibrillator placement with or without cardiac resynchronization therapy.

Chronic heart failure (CHF) is a progressive syndrome that results in a poor quality of life for the patient and places an economic burden on the health care system. Despite advances in the control of cardiovascular diseases such as myocardial infarction (MI), the incidence and prevalence of CHF continue to increase. An accurate estimate of disease burden is difficult to gather because of the vast number of patients with asymptomatic left ventricular (LV) dysfunction. As the population ages, there is an epidemiological shift toward a greater prevalence of clinical heart failure with preserved LV function, the so- called stiff-heart syndrome. In fact, heart failure with preserved systolic function may account for up to two-thirds of cases in patients older than 70 years.2 Regardless of age, the lifetime risk of developing heart failure is approximately 20% for all patients older than 40 years.

Keyword: ACE inhibitors, Aldosterone antagonists, angiotensin receptor antagonist, beta blockers, chronic Heart Failure, digoxin ,neprilysin inhibitors, sartans.

INTRODUCTION

Chronic heart failure is a syndrome, not a specific disease, and occurs as a final common pathway in multiple disease states. The pathophysiology of chronic heart failure (HF) exists when either the left ventricle, the right ventricle, or both, require elevated filling pressures to maintain cardiac output. The neurohormonal responses to impaired cardiac function constitute a negative feedback cycle that is an integral part of the HF syndrome and, to date, blockade of that cycle is the most effective pharmacologic approach to management.

DIAGNOSIS

No single test can be used to establish the clinical diagnosis of heart failure. Instead, history and physical examination findings showing signs and symptoms of congestion and/or end- organ hypoperfusion are used to make the diagnosis. Imaging studies documenting systolic or diastolic dysfunction and biomarkers are helpful adjuncts. Physical examination is not helpful in discriminating between systolic and diastolic heart failure because similar findings, including cardiomegaly and an S3 gallop, can be seen in both conditions. Pulmonary rales, often considered a sign of pulmonary venous congestion, are often absent in CHF despite elevated left-sided filling pressures. This absence is due to chronic lymphatic hypertrophy, which prevents alveolar edema despite elevated interstitial pressures. Framingham criteria, widely used in clinical research, comprise a series of major and minor criteria that aid in the diagnosis of heart failure and emphasize the importance of jugular venous pressure elevation, an S3 gallop, and a positive hepatojugular reflex in establishing a diagnosis, while minimizing the importance of lower extremity edema The use of brain-type natriuretic peptides, in their

active or inactive circulating forms, has evolved during the past decade, but the most well-established use remains in discriminating between causes of dyspnea when the diagnosis is in doubt. Comorbid conditions must be taken into account because renal insufficiency increases these levels and obesity lowers them.8.9.

The etiology of systolic heart failure dramatically affects prognosis and treatment. Coronary artery disease (CAD) accounts for the vast majority of cases of systolic heart failure in the United States, followed by hypertensive and dilated cardiomyopathies. 10 In the acute setting of newly diagnosed cardiomyopathy, the exclusion of underlying CAD and potential "at-risk" myocardium that might benefit from revascularization is critical. Patients with CAD and concomitant heart failure have a worse prognosis than those with nonischemic cardiomyopathy, but myocardial function may substantially improve after revascularization in selected cases, highlighting the importance of making the appropriate diagnosis early and accurately

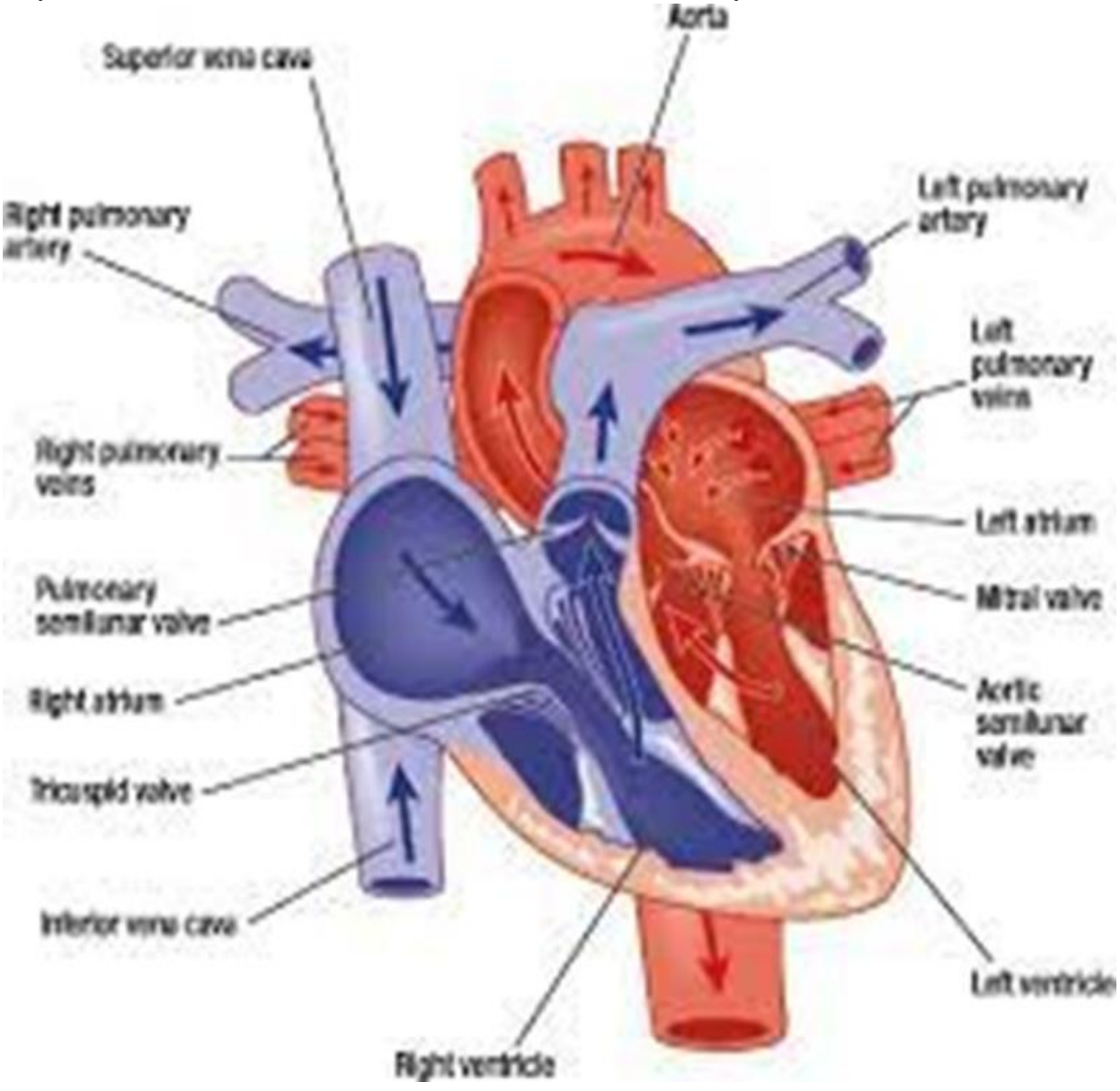


Figure 1: Normal human heart

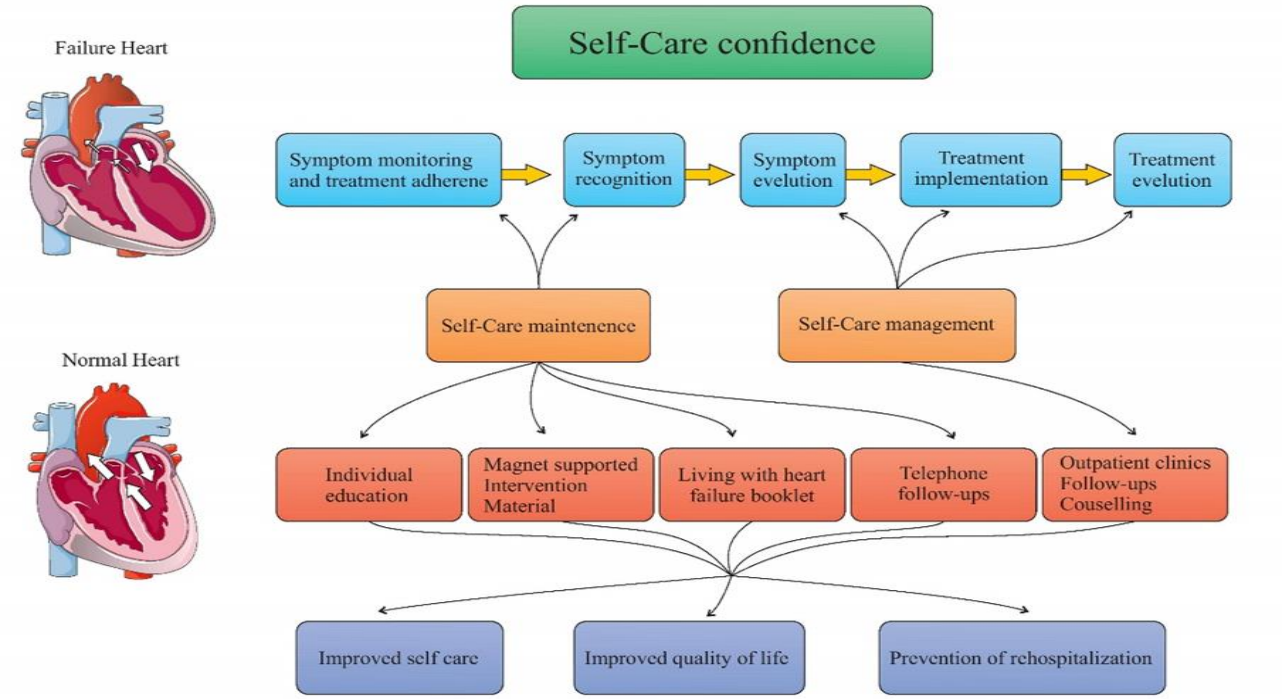


Figure 2: self care confidence

RISK MARKERS, PREVENTION, AND SCREENING

Risk Markers

Multiple cardiovascular conditions, ranging from arrhythmias to valvular heart disease, may ultimately lead to heart failure. Strict adherence to guideline-based management of these conditions is paramount in preventing heart failure. Advanced age is the most potent, albeit nonmodifiable, risk factor. Hypertension, which is easily diagnosed and treated, increases the risk of heart failure 2- to 3-fold. Although the relative risk of developing heart failure is modest, the sheer prevalence renders it a cause in approximately one-third of cases, giving it a high population-attributable risk. In this regard, this risk marker serves as a most viable target for preventive therapy. Analysis of the Framingham heart study revealed the median blood pressure for patients who ultimately developed heart failure was 150/90 mm Hg, emphasizing that risk is increased in suboptimally treated hypertension even at modest levels of severity. Multiple studies across a broad range of agents have unequivocally shown that treatment of blood pressure leads to a marked reduction in heart failure."

Prevention

In light of the mortility, functional limitation, and health care costs that accompany a diagnosis of heart failure, recognition of the importance of prevention is ever- increasing. To highlight the role of prevention in the overall management strategy of heart failure, the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines have identified 4 stages of heart failure. (Table 1). Important in this schema is delineation of a preclinical class (stage A) consisting of patients with conditions that are associated with an increased likelihood for developing heart failure and who should be targeted for aggressive risk factor reduction. Patients with asymptomatic structural LV disease constitute stage B. SOLVD (Study of Left Ventricular Dysfunction), a landmark study, examined angiotensin-converting enzyme inhibitor (ACEI) treatment in this population, demonstrating a 33% reduction in clinical heart failure and hospitalizations." Although no randomized controlled trials of B-blockers in patients with asymptomatic LV dysfunction have been completed, the most recent version of the ACC/AHA guidelines recommend using ß-blockers in patients with stage B disease.15

TABLE 1. **American College of Cardiology/American Heart Association Classification of Heart Failure**

Stage	Definition	Example
A	Risk factors for heart failure	Hypertension, CAD, diabetes, family history, cardiotoxic medications
B	Asymptomatic LV dysfunction	LV hypertrophy, LV dilatation, valvular heart disease
C	Symptomatic heart failure	Dyspnea at rest or with exertion, fluid retention
D	Advanced heart failure	Inotrope requirement, consideration for assist device or transplant

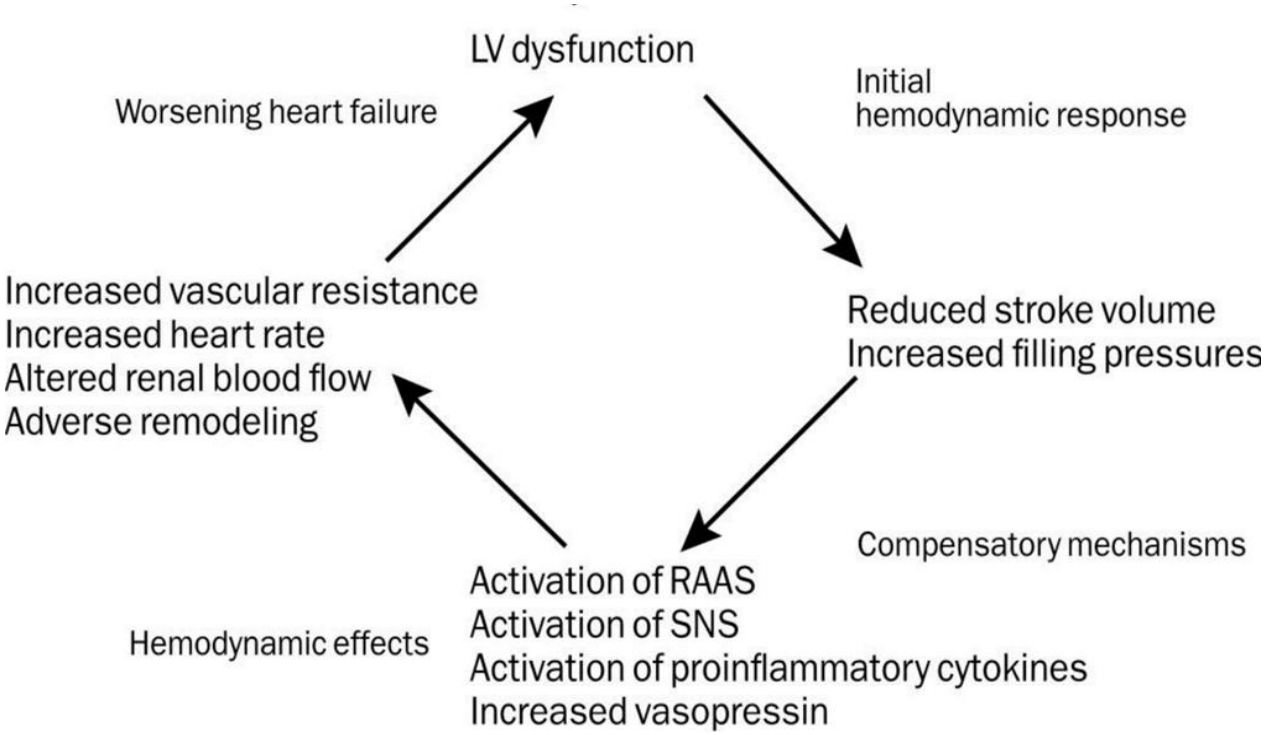
CAD = coronary artery disease; LV = left ventricular.

Screening

Screening asymptomatic patients for heart failure remains controversial and is an area of active investigation. Evidence in support of this practice comes from the Cardiovascular Health Study. Only 9% of patients who ultimately developed systolic heart failure had a reduced LV ejection fraction (LVEF) on study enrollment.25 Biomarkers, such as N-terminal prohormone brain natriuretic peptide and troponin, may potentially function in this role; however, the cost-effectiveness and target populations for these strategies remain unsettled.26.27 Clearly, meaningful strides in heart failure reduction can be attained simply through adherence to existing guidelines and elimination of the financial and psychosocial barriers that deter patients from taking prescribed medical therapy. In a primary care practice, it is incumbent upon the practitioner to develop a focused approach to screening for latent structural heart disease and to develop a clinical screen for manifest CHF. Such screening can be accomplished by asking a simple series of questions related to the occurrence of such symptoms as easy fatigability, functional limitations, and development of lower extremity swelling.

PATHOPHYSIOLOGY

Multiple models have been conceptualized to explain the complex clinical syndrome of heart failure, which stems from a combination of structural pathology, neurohormonal activation, and altered cardiorenal dynamics with end-organ hypofunction. The development of heart failure is characterized by an inciting cardiac injury that triggers a cascade of neurohormonal responses. The previously normal heart may be subject to either an acute (MI) or a chronic (hypertension, valvular heart disease) insult, resulting in altered loading conditions. Subsequent stretching of myocardial fibers or their loss evokes a neurohormonal response characterized by activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system. In the short term, these mechanisms are beneficial and adaptive, sustaining heart rate, blood pressure, and cardiac output, thereby maintaining organ perfusion. Over time, these responses become detrimental, resulting in disruptions of B-adrenergic signaling and impaired mobilization of intracellular calcium. Left untreated, this abnormal neurohormonal milieu leads to myocyte hypertrophy, apoptosis, fibroblast proliferation, and interstitial collagen accumulation, culminating in adverse remodeling and pump dysfunction. The consequences of these pathologic structural changes are a reduction in stroke volume, an increase in systemic vascular resistance, and development of signs and symptoms of congestion and hypoperfusion. These principles have guided the development of therapeutic agents and clinical trial design.



DEVICE THERAPY

Device therapy adds incremental benefit in patients with systolic heart failure or in those who remain ill after receiving medical therapy and can be used to prevent a crisis such as a sudden arrhythmic event. Mechanical dyssynchrony, defined as nonsynchronous contraction between the walls of the left ventricle (intraventricular) or between the ventricular chambers (interventricular), impairs systolic function, adversely affects ventricular filling, increases wall stress, and worsens mitral regurgitation (MR).

Dyssynchrony is most readily defined by the presence of QRS widening on the electrocardiogram and can be visualized on 2-dimensional echocardiography. Placement of a pacing lead via the coronary sinus to the lateral wall of the ventricle enables a more synchronous ventricular contraction. Current indications for cardiac resynchronization therapy (CRT) placement are summarized in. Early studies showed improved exercise capacity, reduction in symptoms, and evidence of reverse remodeling. The CARE-HF (Cardiac Resynchronization in Heart Failure Study) trial was the first study to demonstrate a statistically significant reduction in all-cause mortality with CRT placement. A meta- analysis of 14 randomized trials of CRT confirmed significant reductions in morbidity and mortality. Attempts to further optimize risk stratification and expand indications for CRT using modalities other than electrocardiography have proven disappointing. In particular, echocardiographically derived measures of dyssynchrony vary tremendously, and narrow QRS dyssynchrony has not proven to be a good target for treatment. At this time, CRT should not be used as salvage therapy in patients admitted with acute decompensated heart failure (ADHF). Current indications for CRT implantation are summarized in.

SURGICAL TREATMENT

Patients with ischemic cardiomyopathy often have multivessel CAD. The recognition that hibernating myocardium, defined as myocardial tissue with abnormal function but maintained cellular function, could recover after revascularization greatly affected treatment of patients with ischemic cardiomyopathy. Allman et al99 performed a meta- analysis of 24 studies investigating late survival in 3088 patients with ischemic cardiomyopathy treated with revascularization or medical therapy. In patients with myocardial viability (42% of patients), a marked 79% reduction in annual mortality (16% vs 3%) was observed, with the greatest benefit derived among patients with the poorest LV function and the most

viability. Furthermore, patients without substantial viability showed no incremental benefit with revascularization. Revascularization is most robustly supported in individuals with ongoing angina and LV failure. Revascularizing those with LV failure in the absence of angina remains controversial, but many clinicians opt for revascularization if a substantial aount of hibernating silently ischemic myocardium is discovered.

Multiple surgical techniques to reduce LV volume and thereby alleviate LV wall stress have been used. The recently published STICH (Surgical Treatment for Ischemic Heart Failure) trial randomized patients with ischemic cardiomyopathy undergoing coronary artery bypass grafting (CABG) to CABG alone vs CABG plus surgical ventricular reconstruction.101 Although surgical reconstruction reduced LV volumes and LV wall stress, no difference in mortality or hospitalizations was found. On the basis of these study results, routine surgical LV reconstruction with CABG is discouraged. However, LV volume reduction may still play a role when nonviability of the akinetic segment can be established and when the procedure is likely to provide a volume reduction of a magnitude approaching 30%. summarizes the surgical approach to the patient with heart failure.

CONCLUSION

As the population ages and cardiovascular risk factors become increasingly prevalent, health care professionals in multiple disciplines will encounter patients at risk of heart failure. Successful management of this population depends on risk factor reduction via lifestyle modification and application of currently established guidelines. During the past generation, a combination of behavioral, pharmacological, device-based, and surgical treatment modalities has tremendously enhanced the survival and quality of life of patients with heart failure. In light of the increasing prevalence of heart failure, continued application of these principles and research into novel treatment strategies remain vital

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