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The Pathophysiology of Heart Failure: A Review Article

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Abstract. Heart failure (HF) continues to be a major issue in the US and around the world, leading to high rates of morbidity and death at a significant financial burden on healthcare. A basic comprehension of the processes that underlie the heart failure (HF) adaptive response has resulted in the identification of multiple channels of compensation, which have been the focus of medical and pharmacological treatments intended to lessen the debilitating condition. To increase HF patients' chances of survival, further research is required to find novel targets.

Highlights:

1. Heart failure causes high morbidity, mortality, and financial burden.
2. Understanding HF mechanisms aids medical and pharmacological interventions.
3. Further research needed for novel treatment targets and improved survival.

Keywords: Pathophysiology, Heart Failure, Review

Definition and epidemiology

When the heart cannot maintain cardiac output (CO) high enough to support metabolic demands and permit venous return constitutional complaints, a complex clinical disease called heart failure (HF) develops. Lung congestion, ascites due to decreased venous return, and peripheral edema. Fatigue, lack of appetite, and nausea are other typical constitutional complaints (1, 2). This last common clinical pathway, which accounts for more than one-third of all cardiovascular-related fatalities in the US and has a 50% 5-year mortality rate, involves several reasons (3, 4).

The leading cause of death worldwide is still cardiovascular disease, which is also becoming more prevalent. (5, 6). The prevalence of HF is around 5 million in the US and 22 million worldwide, with over two million new cases detected abroad and 500,000 new cases diagnosed in the US annually. Heart failure (HF), which has a one in five chance of developing by the time they are forty, affects ten out of every thousand people over 65 in the United States. HF is more common in men up to age 65 (7, 8).

Black Americans had the highest incidence of heart failure (HF) with 4.6 cases per 1000 person-years, followed by Asian, Caucasian, and Hispanic Americans (3.5, 2.4, and 1.0 instances per 1000 person-years, respectively) (9, 10). Hospitalizations for HF

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patients, who usually take six medications related to HF, are projected to cost between $10 and 38 billion annually, and 78% of them need at least two hospital stays each year (11, 12).

Etimology

After the heart is injured for a variety of reasons, a crucial amount of functioning myocardial cells is lost, which results in heart failure. Diabetes, hypertension, and ischemic heart disease are the most frequent causes. Seventy-five percent of all HF patients already have hypertension, which raises their chance of developing HF in comparison to those with normotension (13, 14).

Disease progression and functional classification

The New York Heart Association (NYHA) classification technique is frequently used to classify patients with HF. It assigns them to one of four groups according to the physical disability that HF causes (15, 16). The NYHA classification is currently employed as an admission requirement for clinical studies of drugs and devices in addition to recording functional heart states. People who have NYHA Class I HF do not have heart disease that prevents them from performing their usual activities. Patients in NYHA Class II have limited physical activity; although they feel OK when at rest, frequent exercise results in angina, palpitations, fatigue, or dyspnea. NYHA Class III patients are still calm while they are at rest, but they have very little physical activity, and symptoms show up when they are not as active as they usually are (17, 18).

More recently, the American Heart Association (AHA) and the American College of Cardiology (ACC) created a new approach that places more emphasis on the beginning and course of the illness process (19, 20). This classification system recognizes the structural parameters for the development of HF as well as the known risk factors for the condition. Individuals in Stage A are at a high risk of heart failure, even if they do not have any structural heart issues. Although they lack HF symptoms, patients in stage B have a structural defect. Individuals in Stage C often have underlying structural heart disease-related HF symptoms, either current or former. Patients in stage D require specialized treatment methods since they have end-stage heart failure (HF).

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Pathophysiology

The blood the heart pumps in a certain amount of time is known as heart output. It is typically between 4 and 8 L/min and is computed by multiplying heart rate (HR) by stroke volume (SV). Other factors include valvular competence, synergistic ventricular contraction, and ventricular wall integrity also affect CO. The ventricle's SV, or the volume of blood it expels with each beating, is normally 1 cc/kg, or about 60 to 100 cc. Three key factors influence SV: contractility (21, 22).

Can be classified as either diastolic (involving impaired relaxation and ventricular filling) or systolic (with impaired contraction and ejection). Although several HF etiologies impair both systolic and diastolic function, systolic dysfunction affects 70% of HF patients, while diastolic dysfunction affects 30%. Additionally, Diastolic dysfunction is present in the majority of patients with systolic dysfunction. The ejection fraction, or the volume of blood pumped from the ventricle in a single pulse. An ejection fraction of b40% results in systolic dysfunction, whereas an EF of N40% results in diastolic dysfunction (23, 24). LV failure is the most frequent cause of right ventricular (RV) failure. The blood volume in the ventricle rises as the RV fails. This stops the body from releasing venous fluid by raising the pressure in the right atrium and the vena caval system (25, 26).

Frank-Starling mechanism

One crucial compensatory mechanism in the early phases of heart failure is the Frank-Starling mechanism. CO and LVEDP, which are directly correlated with LV end-diastolic volume (also known as preload or LVEDV), are depicted in this graph. A healthy patient with a normal heart is represented by point A on the curve. This graph indicates that an increase in preload raises LVEDP, which triggers the Frank-Starling mechanism, a stretching, and reaction of the myocardium that raises CO. There is a limit to this CO increase; as the curve flattens out, the CO increase that results falls as preload levels rise (27, 28).

Neurohormonal activation

In the early phases of HF, neurohormonal activity is crucial for both MAP maintenance and compensation. Remembering that MAP is the result of TPR, neurohormonal stimulation increases TPR, which in turn augments MAP. Furthermore,

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some neurohormones encourage water and sodium retention, which increases CO through the Frank-Starling mechanism and maximizes SV (29, 30).

The sympathetic nervous system (SNS), which releases catecholamines (epinephrine and norepinephrine), is triggered by the drop in MAP observed in HF. The effects of the sympathetic nervous system are mediated by three receptors: β1, β2, and α1. Patients with heart failure are overstimulated by the SNS, which activates both β1 and β2 receptors and damages the heart. This shows up as decreased EF, arrhythmias, and tachycardia. Vasoconstriction, thirst, and sodium retention are caused by the renin-angiotensin-aldosterone system (RAAS), which is activated by β1 and α1 receptors in the peripheral vasculature. Furthermore, this method increases MAP (31,32).

Both this sympathetic stimulation and decreased renal blood flow caused by a decrease in MAP cause the kidneys to release renin which instantly constricts blood vessels and promotes the production of aldosterone. The ultimate effects of activating this specific neurohormonal pathway include increased contractility, salt reabsorption, norepinephrine release facilitation, and vasopressin release stimulation. The hypothalamus produces vasopressin, which the posterior pituitary gland secretes. The production of angiotensin II facilitates its release, which is similarly regulated by a negative feedback loop. The hypothalamus receives fewer inhibitory impulses from central baroreceptors when they detect a drop in MAP in HF, which lifts the negative regulation and promotes the production of vasopressin. Vasoconstriction and enhanced water retention are brought on by elevated vasopressin, and they both contribute to the reduction in MAP in HF (33,34).

Ventricular remodeling

Remodeling is the term used to describe the alterations in the ventricle's size, shape, structure, and function brought on by the heart's ongoing hemodynamic strains. Ventricular mass, composition, and volume all alter during remodeling, and the ventricular's general shape shifts from elliptical to spherical.

Because a failing heart expands to increase ventricular volume, these geometric changes are initially compensatory and elevate CO and SV even while EF is lowered. An initial rise in contractility is also caused by increased myocardial wall thickness and total ventricular mass. In HF, the remodeling process progresses over time and eventually

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becomes harmful. Increased wall tension and fibrosis result from the myocardial hypertrophying and the ventricle's continued enlargement, which ultimately reduces contractility. Myocardial apoptosis also rises as a result of the remodeling process throughout time. Furthermore, the dilated and modified ventricle exhibits severe contractile dyssynchrony, which reduces pumping efficiency (35,36).

Other neurohormones

In HF, additional neurohormonal processes are at play. Some of them are brain natriuretic peptide (BNP), atrial natriuretic peptide (ANP), and c-type natriuretic peptide (CNP). ANP and BNP are released by the atria and ventricles, respectively, during an atrial or ventricular stretch. The central nervous system is where CNP is most frequently observed. These hormones have a direct impact on blood vessels, resulting in vasodilation, water and salt excretion, and suppression of the release of vasopressin, aldosterone, and renin. For instance, elevated BNP is used to track the illness's course and is believed to be one of the initial indicators of HF (37,38).

Additionally, the vascular endothelium generates endothelium-derived vasoactive substances that operate locally to encourage either vasodilation (prostacyclin, bradykinin, and nitric oxide) or vasoconstriction (endothelin I). Increased production of several cytokines, such as interleukin 1α, interleukin 6, interferon α, and tumor necrosis factor α, is linked to heart failure. Because these small compounds are negative inotropes that decrease contractility (39,40).

Treatment of the Heart Failure

The specifics of the HF patient's treatment are outside the purview of this assessment. The ACC/AHA guidelines serve as the foundation for the fundamental recommendations provided here. Both medication interventions and lifestyle changes are common treatments for heart failure. It is important to support patients in quitting smoking and drinking, losing excess weight, and exercising as tolerated to enhance their physical health. Medical treatments include limiting sodium and water intake and treating hypertension, diabetes, dyslipidemia, and arrhythmias. It is important to treat revascularizable coronary artery disease as needed (41,42).

The goal of many medications used in the pharmacologic treatment of heart failure is to reverse the detrimental effects of the compensatory mechanisms previously

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outlined. It functions by reducing RAAS and SNS activity and raising the cardiac muscle's inotropy (43,44). Diuretics, like furosemide, increase tolerance to exercise and reduce fluid retention, which causes peripheral edema and lung congestion. Captopril and enalapril are examples of ACE inhibitors that decrease RAAS activation by preventing angiotensin I from becoming angiotensin II (45,46).

Conlusion

Heart failure (HF), which causes substantial morbidity and mortality at a large cost to society and the healthcare system, is still a major issue in the US and around the world. These channels have been the focus of medical and pharmaceutical interventions intended to ameliorate this debilitating condition. To increase the survival of HF patients, further research is required to find novel targets

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