



## HEART FAILURE: RISK FACTOR, CLASSIFICATION, SYMPTOMS, PREVENTING DISEASE

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### ABSTRACT :

Since heart failure is a potentially fatal condition, treating it ought to be a top priority for world health. Heart failure affects over 26 million people globally at the moment. In the modern world, heart failure is thought to be an epidemic condition that affects 1% to 2% of adults. It is a complex, systemic disease in which cellular, molecular, neurohumoral, and structural systems are triggered upon myocardial infarction and work together to sustain physiological functioning. Different, simultaneous emerging clinical indications and symptoms are the outcome of these coordinated, complicated processes that cause excessive volume overload, increased sympathetic activity, and redistribution of the circulation. Because the combination of these symptoms and indicators creates an ambiguous clinical picture, both invasive and noninvasive diagnostic techniques are employed to identify the underlying cause and make an accurate diagnosis. Heart failure's continuous progression is the most significant aspect that determines how the condition turns out.

In an effort to maintain these compensatory mechanisms within a physiological range, pharmatherapeutic regimens, novel targets, and careful management of these processes are constantly optimized. In addition to medication therapy, new opportunities for the management of heart failure are provided via interventional and surgical therapy alternatives. In order to optimize and establish these and other novel therapeutic approaches, a thorough understanding of the underlying mechanisms is fundamentally required. In addition to diagnosis and treatment, efforts should be made to improve heart failure prevention through managing risk factors or recognizing and adhering to risk groups. In an effort to contribute to a more thorough understanding of the condition, this synopsis of the pathophysiology of heart failure attempts to provide a succinct synopsis of both the unique unfolding, progressive theory of heart failure and its fundamental mechanics.

### INTRODUCTION :

Prioritizing global health should include preventing heart failure-related illnesses and deaths. The general public, lawmakers, and even some healthcare experts are not well-informed about heart failure, despite the fact that the condition is killing and affecting a growing number of people. Heart failure cannot be cured, but it can be prevented in many situations, and most patients can receive appropriate treatment to increase their chances of survival and quality of life. It is the duty of policymakers to guarantee that the greatest number of individuals can take advantage of the strategies for heart failure prevention, diagnosis, treatment, and long-term care. In addition, funding for research ought to be provided in fields with pressing unmet needs. To determine the best strategies to treat heart failure in various regions of the world and to implement the required procedures into routine practice, a global strategy is required. The Global Heart Failure Awareness Programme was initiated by the Heart Failure Association of the European Society of Cardiology with this goal in mind. The program will engage heart failure groups worldwide to ensure that information, insights, and recommendations are shared across borders and continents<sup>[1]</sup>

The Framingham criteria, which include the patient's clinical indicators and manifested symptoms, are typically used to diagnose heart failure<sup>[2]</sup>. At least two main criteria or one major criterion plus two minor criteria are needed for the diagnosis. Only when two or more minor criteria, such as liver cirrhosis, nephrotic syndrome, or chronic lung illness (chronic obstructive pulmonary disease, or COPD), cannot be attributed to distinct organ failure are they recognized as a diagnosis.

Major criteria	Minor criteria
Paroxysmal nocturnal dyspnea	Bilateral ankle edema
Basal crepitations	Dyspnea on ordinary exertion
Cardiomegaly	Nocturnal cough
Jugular vein distension	Pleural effusion

Fig. Framingham Criteria for Congestive Cardiac Failure<sup>[3]</sup>

## ETIOLOGY AND RISK FACTOR

Ischemic heart disease, which is mostly brought on by acute or chronic myocardial ischemia, is the most prevalent cause of heart failure. It is characterized by poor myocardial perfusion. Cardiomyopathies (idiopathic or toxin-induced, such as alcohol and doxorubicin) and valvular heart disorders are other, but nonetheless prevalent, causes.<sup>[4][5]</sup> The etiological categorization of heart failure lacks an established standard; however, it can be classified into numerous subcategories based on factors such as the affected functional phase, circulation system, volume status, etc.<sup>[6]</sup>

Many medical conditions can be risk factors in and of itself, and people with heart failure typically have a combination of these conditions that can lead to cardiac dysfunction.<sup>[7]</sup>

Causes of systolic heart diseases	Causes of diastolic diseases
Coronary artery disease	Coronary artery disease
Arrhythmia	Hypertrophic cardiomyopathy
Inflammatory diseases	Restrictive cardiomyopathy
Peripartum cardiomyopathy	Constrictive pericarditis

Fig . Causes of systolic and diastolic heart diseases<sup>[8]</sup>

## CLASSIFICATION

A number of pathophysiological or functional viewpoints, including the affected circulatory system (right-left), cardiac function (systolic/diastolic), or the underlying pathophysiological cause (pressure-induced/volume-induced), can be used to categorize heart failure.

### 1] Systolic versus diastolic heart failure

A precise characterization and comprehension of the fundamental mechanisms behind both diastolic (HF-PEF) and systolic (HF-REF) heart failure are crucial for comprehending the fundamental concepts and mechanisms of the pathophysiology of heart failure. Heart failure, both diastolic and systolic, are separate disorders with well-established epidemiology, symptomatology, and pathogenesis.

The macro- and micromorphology of the heart, including the cardiomyocytes or the extracellular matrix structure, differs from the development of decreased cardiac function. Reduced EF is the hallmark of systolic heart failure, which is caused by a compromised left ventricle's ability to contract. Heart failure with reduced left ventricular ejection fraction (HF-REF) is another name for this syndrome. Heart valve disorders, cardiomyopathies, and ischemic heart disease are the most prevalent underlying causes of systolic heart failure.<sup>[9]</sup>

### 2] Pressure-overload versus volume-overload heart failure

Reduced myocardial contractile performance, unfavorable left ventricle chamber remodeling, or a combination of these factors can lead to left ventricular failure via pressure overload. It generally manifests in patients with arterial hypertension and/or aortic valve stenosis. When the heart chambers are overflowing with blood that they are attempting to pump into the systemic circulation, it is referred to as volume overload. Volume overload can result from a number of pathologies, including valvular heart illnesses (e.g., aortic regurgitation and mitral regurgitation), congenital heart diseases (persistent ductus arteriosus and ventricular septal defect), and arteriovenous malformations and fistulas. In addition, congenital atrial septal defects and valvular heart disorders (pulmonary and tricuspid regurgitation) impact the pulmonary circulation.

### 3] Low-output versus high-output heart failure

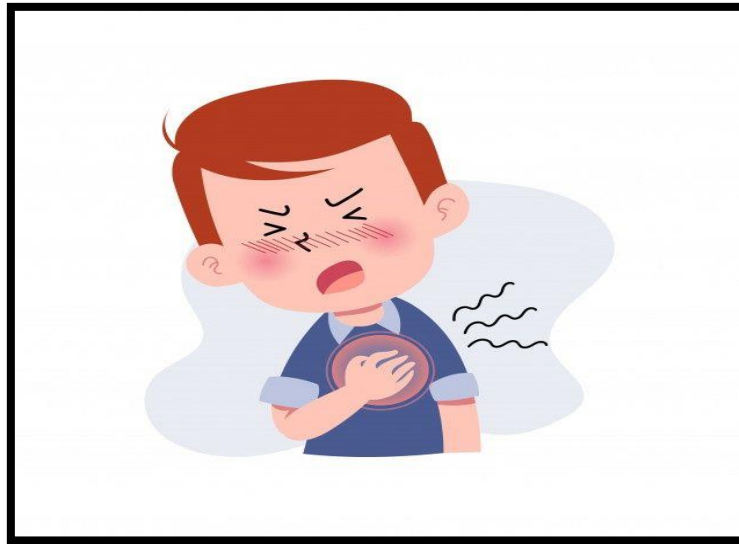
When there is low output, the heart's cardiac output (CO) cannot increase with effort and cannot meet the peripheral tissues' needs for blood and oxygen. Three categories can be used to classify the causes of low output heart failure: excessive preload, excessive afterload, or pump failure.

- **Pump failure (reduced inotropy)**
  - a. Negative inotropic drugs
  - b. systolic cardiac failure
  - c. relevant bradycardia
- **Excessive preload (volume overload)**
  - a. Mitral regurgitation
  - b. Aortic regurgitation
- **Excessive afterload (pressure overload)**
  - a. Aortic stenosis
  - b. Hypertension<sup>[10]</sup>

## SYMPTOMS ON VARIOUS ORGANS

### *Cardiac symptoms*

Cardiomegaly, or the enlargement of the heart, frequently coexists with heart failure. On a chest X-ray, where the cardiothoracic index is noticeably elevated (>50%), it is most visible. Cardiomegaly is characterized by a pronounced perceptible pulsation and a left-lateral displacement of the punctum maximum of the maximal impulse. Generally speaking, the first heart sound is quiet, particularly in tachycardic patients. Patients with heart failure may also exhibit a protodiastolic gallop or a third or even fourth heart sound. It occurs usually in volume overload and is most heard at the pinnacle. Furthermore, auscultation may reveal murmurs of mitral or tricuspid regurgitation.



### *Anemia*

Heart failure raises the incidence and prevalence of anemia, which is often moderate (8); anemia prevalence in NYHA classes II to III is approximately 20%. Particularly in women, older individuals, and patients suffering from renal failure, concurrent anemia is prevalent. Oxygen transport and/or oxygen utilization are reduced in tissues as a result of the current anemia, which drastically lowers physical capacity. Recent research suggests that iron supplementation may benefit anemia (FAIR-HF); yet, a pure anemia correction may not be as effective. A definitive determination, however, will be based on the outcomes of ongoing clinical trials (RED-HF, STAMINA-HeFT)<sup>[11]</sup>



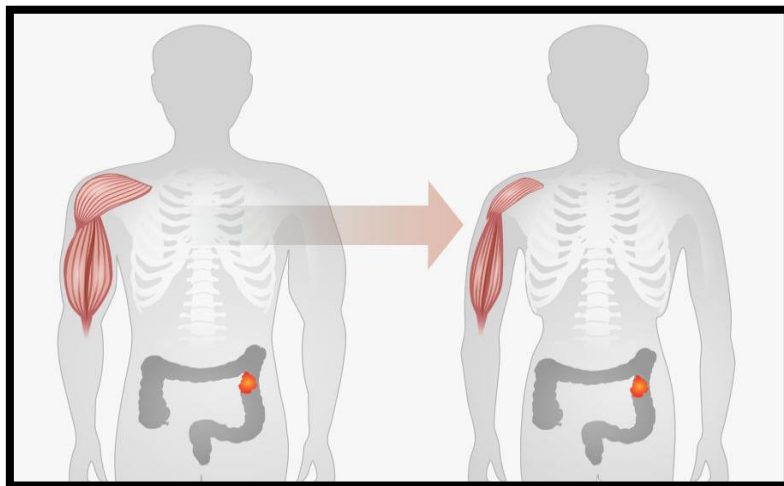
### *Gastrointestinal symptoms*

In addition to digestive issues, patients with severe heart failure may experience digestive issues. A common concomitant condition of systemic congestion is anorexia, nausea, or stomach pain, including the liver or bowels. Hepatomegaly is characterized by an enlarged, inflamed, and pulsing liver that is also frequently tender and uncomfortable <sup>[12]</sup>. Ascites typically develops as a result of the increased hepatic vein pressure. Jaundice is a late symptom that can arise from congestion, hypoxia from poor perfusion, and altered liver function.



### *Cardiac cachexia*

Peripheral muscle wasting, also known as cardiac cachexia, is typically seen in patients with severe heart failure. This condition is often limited to the lower limbs (disuse atrophy) or can affect the entire body and other tissues (10). One of the severe symptoms of advanced heart failure, cardiac cachexia, has an exact mechanism that is unknown, however there are some factors that influence its development. There's little doubt that factors like elevated metabolic rate, nausea, vomiting, anorexia, malabsorption from liver and stomach congestion, and proinflammatory processes that are triggered are implicated. Heart cachexia is linked to a dismal prognosis for the illness.



### *Preventing heart failure in high-risk groups*

Heart failure prevention is really important. Once detected, the heart's decline in health can frequently be treated, albeit it is usually irreversible. Legislators ought to emphasize how important it is for medical experts in all clinical specialties to recognize individuals who have conditions that put them at risk for heart failure and to provide preventative drugs. Regardless of age, gender, or financial status, individuals most at risk of developing heart failure should have equitable access to preventive drugs<sup>[19]</sup>. The eradication of specific infectious diseases in regions of the world where they still cause heart failure should be a top priority for policymakers<sup>[13]</sup>

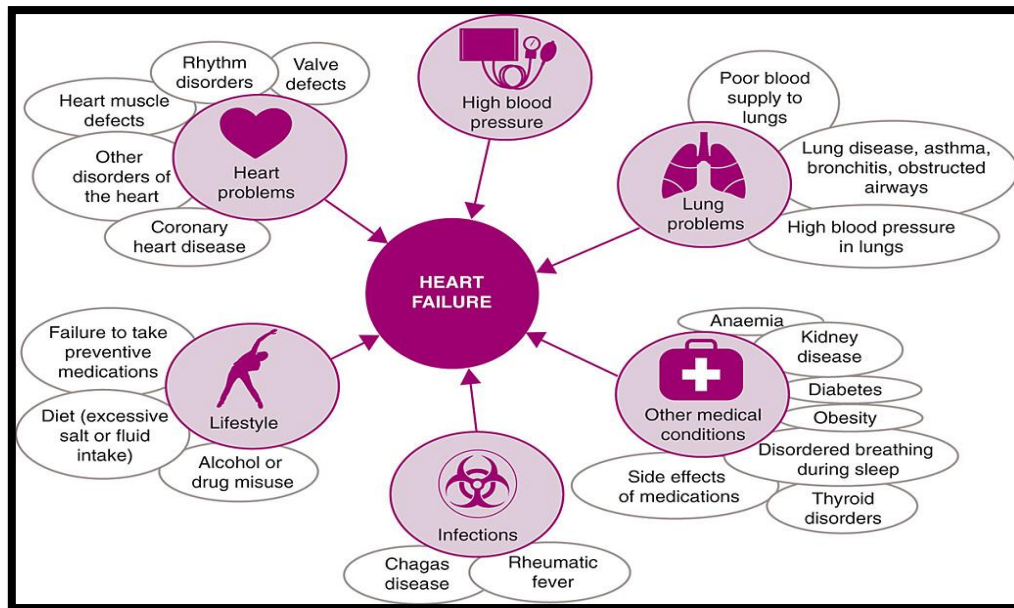


Fig. Causes of heart failure and illness.

### Identifying and treating patients at risk of developing heart failure

By identifying those who have early indicators of aberrant remodeling of the heart muscle, preventive treatment could begin earlier. Sadly, there is no easy diagnostic test for heart failure, making large-scale screening programs unfeasible (see Section on The Need to Apply Best Practice). These programs have allowed earlier treatment for breast, cervical, and bowel cancer.<sup>90</sup> Medical imaging technology can identify early alterations in the structure or function of the heart; nevertheless, it is impractical to carry out these intricate operations on the vast majority of patients suffering from conditions that result in heart failure, and most definitely not on the general public.<sup>[18]</sup>

More people could benefit from preventative medicine if it is directed towards those who are most at risk of developing heart failure.<sup>[20]</sup> This would also make it more cost-effective. Public and private funding should keep supporting the ongoing research in these fields. Programs to raise awareness should also target all individuals with illnesses that put them at risk of heart failure. Educating people about heart failure symptoms and the advantages of adopting healthier lifestyle choices should be among them. Programs aimed at raising public awareness of heart failure should emphasize the same themes (see Section on Improving Public Awareness of Heart Failure)<sup>[14]</sup>

### Preventing heart failure in the elderly and socioeconomically disadvantaged: unique challenges

As the population ages, preventing heart failure in the elderly is becoming increasingly important from a healthcare perspective.<sup>91</sup> In economically developed regions, heart failure is the most prevalent cause of hospital admission for adults over 65 (see Section on The Global Burden of Heart Failure). Women make up the majority of elderly heart failure patients in hospitals.<sup>94</sup> While several studies on heart failure patients have suggested that women have higher survival rates than males, more recent investigations have revealed that women's long-term prognosis may not be as favorable as previously believed.<sup>95</sup> Reaching out to older individuals, especially older women, should therefore be a part of initiatives aiming at increasing heart failure prevention.<sup>[15]</sup>

Governments in fast developing nations must balance the need to eradicate infectious diseases from resource-constrained or rural areas with the need to address diseases that are spreading to cities due to a shift in lifestyle toward a more Western one.<sup>[17]</sup> The fact that heart failure is more common in the old, lonely, female, and impoverished hasn't done much to increase awareness of the condition. It is time to raise public awareness of heart failure through large-scale initiatives supported by business and government grants.<sup>[16]</sup>

### CONCLUSION :

Heart failure was once thought to be the outcome of renocardial syndrome, or salt and water retention brought on by reduced renal perfusion. Subsequently, the hemodynamic theory came to be, explaining cardiac dysfunction as a result of elevated afterload and decreased CO. Although these two ideas outline the essential characteristics of heart failure, they are unable to explain why the condition progresses inexorably. Consequently, a brand-new progressive model of heart failure was created.

Heart failure develops as a consequence of a main cardiac event. It can happen either persistently, as in cardiomyopathies, or abruptly, as in myocardial infarction.

To keep things stable, a lot of work has gone into reversing the remodeling processes that are progressing. In addition to new and improved medication regimens, surgical and interventional techniques have advanced significantly during the past few decades. Several techniques were successful in achieving reverse remodeling; during cardiac resynchronization therapy, for example, notable progress was evident. Cardiovascular medicine continues to focus on understanding the pathophysiological mechanisms that lead to heart failure due to its theoretical complexity and intricate processes as well as its practical significance for the development of novel therapeutic strategies.

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REFERENCES :

1. Lloyd-Jones DM, Larson MG, Leip EP *et al.* Lifetime risk for developing congestive heart failure: the Framingham Heart Study. *Circulation* 2002; **106**: 3068–72.
2. Rodriguez KL, Appelt CJ, Switzer GE *et al.* 'They diagnosed bad heart': a qualitative exploration of patients' knowledge about and experiences with heart failure. *Heart Lung* 2008; **37**: 257–65.
3. Wasywich CA, Gamble GD, Whalley GA *et al.* Understanding changing patterns of survival and hospitalization for heart failure over two decades in New Zealand: utility of 'days alive and out of hospital' from epidemiological data. *Eur J Heart Fail* 2010; **12**: 462–8.
4. Borlaug BA. The pathophysiology of heart failure with preserved ejection fraction. *Nat Rev Cardiol* 11: 507-15, 2014
5. Bortone AS HO, Chiddo A, Gaglione A, Locuratolo N, Caruso G, Rizzon P. Functional and structural abnormalities in patients with dilated cardiomyopathy. *JACC* 14: 613-623, 1989
6. Braun J, Bax JJ, Versteegh MI, Voigt PG, Holman ER, Klautz RJ, Boersma E, Dion RA. Preoperative left ventricular dimensions predict reverse remodeling following restrictive mitral annuloplasty in ischemic mitral regurgitation. *Eur J Cardiothorac Surg* 27: 847-853, 2005.
7. Bristow MR, Ginsburg, R, Minobe W, Cubicciotti RS, Sageman WS, Lurie K, Billingham ME, Harrison DC, Stinson EB. Decreased catecholamine sensitivity and  $\beta$ -adrenergic-receptor density in failing human hearts. *N Engl J Med* 307: 205-211, 1982.
8. Carabello BA. Concentric versus eccentric remodeling. *J Card Fail* 8: S258-263, 2002.
9. Park JH, Negishi K, Grimm RA, Popovic Z, Stanton T, Wilkoff BL, Marwick TH. Echocardiographic predictors of reverse remodeling after cardiac resynchronization therapy and subsequent events. *Circ Cardiovasc Imaging* 6: 864-872, 2013
10. Pepys MB, Hirschfield GM, Tennent GA, Gallimore JR, Kahan MC, Bellotti V, Hawkins PN, Myers RM, Smith MD, Polara A, Cobb AJ, Ley SV, Aquilina JA, Robinson CV, Sharif I, Gray GA, Sabin CA, Jenvey MC, Kolstoe SE, Thompson D, Wood SP. Targeting C-reactive protein for the treatment of cardiovascular disease. *Nature* 440: 1217- 1221, 2006
11. Voelkel NF, Quaife RA, Leinwand LA, Barst RJ, McGoon MD, Meldrum DR, Dupuis J, Long CS, Rubin LJ, Smart FW, Suzuki YJ, Gladwin M, Denholm EM, Gail DB, National Heart, Lung, and Blood Institute Working Group on Cellular Molecular Mechanisms of Right Heart, Failure. Right ventricular function and failure: Report of a National Heart, Lung, and Blood Institute working group on cellular and molecular mechanisms of right heart failure. *Circulation* 114: 1883- 1891, 2006
12. Waldum B, Stubnova V, Westheim AS, Omland T, Grundtvig M, Os I. Prognostic utility of B-type natriuretic peptides in patients with heart failure and renal dysfunction. *Clin Kidney J* 6: 55-62, 2013
13. Anker SD, Chua TP, Ponikowski P, Harrington D, Swan JW, Kox WJ, Poole-Wilson PA, Coats AJ. Hormonal changes and catabolic/anabolic imbalance in chronic heart failure and their importance for cardiac cachexia. *96*: 526-534, 1997.
14. Arakawa HUaK. Angiotensin II-forming systems in cardiovascular dis- eases. *Heart Fail Rev* 3: 119-124, 1998
15. Anand I, McMurray JJ, Whitmore J, Warren M, Pham A, McCamish MA, Burton PB. Anemia and its relationship to clinical outcome in heart failure. *Circulation* 110: 149-154, 2004.
16. Tang YD, Katz SD. Anemia in chronic heart failure: Prevalence, etiology, clinical correlates, and treatment options. *Circulation* 113: 2454-2461, 2006.
17. McMurray JJ, Adamopoulos S, Anker SD *et al.* ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2012: the task force for the diagnosis and treatment of acute and chronic heart failure 2012 of the european society of cardiology. Developed in collaboration with the heart failure association (HFA) of the ESC. *Eur J Heart Fail* 2012; **14**: 803–69.
18. Endoh M. Heart failure: management and prevention of heart failure based on current understanding of pathophysiological mechanisms. In I Wakabayashi, K Groschner, eds. *Interdisciplinary concepts in cardiovascular health*. Springer, 2014: 41–67. Available from: [http://link.springer.com/chapter/10.1007%2F978-3-319-01074-8\\_3](http://link.springer.com/chapter/10.1007%2F978-3-319-01074-8_3) (Accessed 25 February 2014).
19. Baker DW. Prevention of heart failure. *J Card Fail* 2002; **8**: 333–46
20. Andrade JP, Marin-Neto JA, Paola AA *et al.* I Latin American guidelines for the diagnosis and treatment of Chagas cardiomyopathy. *Arq Bras Cardiol* 2011; **97**: 1–48.