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EXPERT CONSENSUS DOCUMENT

AHA/ACC/ESC/WHF Expert Consensus Document: Second Universal Definition of Heart Failure (2026)

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ABSTRACT

Heart failure (HF) remains a pressing health concern, with rising prevalence globally. Subjectivity and ambiguity in the definition of HF and its antecedent stages have limited research, global surveillance, and prevention programs. To address this, several cardiac societies and foundations convened to standardize the definition of HF in 2021 and designated stage B or pre-HF to identify individuals at risk of developing HF. In subsequent years, substantial progress and changes have been made in aspects of preventing HF, improving HF diagnosis and management, and recognizing the importance of the affected individual's voice. Global differences and disparities in HF are better understood, as are causes and comorbidities leading to differences in care, which are also influenced by access to care. This consensus document presents the Second Universal Definition of Heart Failure, aiming to standardize terminology and facilitate a uniform approach for clinicians, researchers, health systems, and policymakers. In this definition, the classification of HF phenotypes moves away from rigid left ventricular ejection fraction cutoffs, instead grouping HF into reduced, preserved, and improved ejection fraction categories to better reflect clinical realities. A universal classification of HF causes is also proposed. The document also addresses the dynamic trajectories of HF—improvement, remission, and recovery—and highlights the impact of social determinants and geographic variation on HF risk and outcomes. By providing a comprehensive, standardized framework for HF definition and classification, this document seeks to improve prevention, early detection, and management of HF worldwide, ultimately enhancing patient care and advancing global cardiovascular health.

Universal definitions of clinical syndromes have been instrumental in guiding diagnosis by describing a combination of symptoms, biomarkers, imaging studies, and pathology that clearly characterize the syndrome. The first Universal Definition and Classification of Heart Failure[1] clarified definitions of heart failure (HF) that had previously been ambiguous, poorly understood, and lacking in standardization.

Since then, the incidence and prevalence of HF, particularly among individuals with preserved ejection fraction (HFpEF), have continued to increase, as has the risk of HF hospitalization and mortality.[2,3] The growing prevalence of HF is driven by the increasing numbers of individuals with HFpEF, the aging of the population, and secular trends in the prevalence of comorbidities that drive HF risk. In addition, advances in technology and the development of more advanced imaging methods (magnetic resonance imaging, positron emission tomography), allowing earlier and more accurate disease detection, along with improvements in survival resulting from the availability of expanded treatment options, have increased prevalence. Improved strategies for early diagnosis of HF leveraging artificial intelligence may enhance HF detection even further over traditional biomarker- and imaging-based approaches, making estimates of the total population-level HF burden more accurate.

Definitions of HF, including participant inclusion criteria, vary widely in HF clinical trials. Specifically, requirements for inclusion of only narrow ranges of left ventricular ejection fraction (LVEF) often obscure possible benefits of therapies for HF phenotypes that range outside that criterion.[4]

The identification of more diverse phenotypes of HF is rapidly expanding as a result of improvements in and greater availability of advanced imaging technologies. Routine genetic screening—as ease of testing has increased and cost has decreased—has brought new understanding of genetic and familial cardiomyopathies to the clinic and to many more patients and families. In addition, the impact of comorbid diseases and their therapies on HF phenotypes has made the accurate identification of such phenotypes crucial to aid in appropriate and effective therapies. The development and greater availability of HF treatments that are specific to HF phenotype drive the necessity for accurate identification and diagnosis.

Disparities in HF care are present worldwide, with access to diagnosis and quality treatment being influenced by race, ethnicity, sex, income, geography, access to and coverage of health care, and socioeconomic status.[5] These disparities in care are particularly concerning for high-risk, underserved groups, who have the highest risk for HF hospitalization and death.[6,7]

These considerations underscore the need for the current update. As with other universal definitions such as that of myocardial infarction,[8] the impetus for this updated document is to address changes in disease manifestations, diagnostic strategies, and understanding of pathophysiology. The current document serves as an update of the first definition[1] with collaboration by the American College of Cardiology, the American Heart Association, the European Society of Cardiology, and the World Heart Federation. This document is not a clinical practice guideline, nor is it a clinical decision support document. Treatment recommendations will remain

reserved for the current and future professional society HF guidelines documents.[9,10]

PROPOSED CHANGES IN THE DEFINITION: CLASSIFICATION, PHENOTYPES, AND TRAJECTORIES

Definition of HF Syndrome

As outlined in the first Universal Definition of Heart Failure,[1] HF is a clinical syndrome with diverse causes characterized by the presence of typical symptoms, physical examination signs, and laboratory or imaging abnormalities suggestive of pulmonary or systemic congestion or changes in cardiac output that can be attributed to an underlying structural or functional cardiac abnormality. Clinical symptoms and signs of HF include dyspnea on exertion, orthopnea, jugular venous distention, rales, S3 gallop, and abdominal and leg swelling. Diagnostic certainty is amplified by confirmatory elevation in natriuretic peptide (BNP [B-type natriuretic peptide] or NT-BNP [N-terminal B-type natriuretic peptide]) levels or imaging evidence (lung ultrasound, chest radiography, echocardiography, computed tomography) of pulmonary congestion and elevated intracardiac filling pressures. It is important to note that HF is defined by the accumulation of suggestive features in a comprehensive assessment rather than by a single diagnostic test. As an example, a substantial proportion of individuals with HFpEF have normal natriuretic peptide levels despite unequivocal invasive hemodynamic evidence of HF, for example, elevation of ventricular filling pressure at rest or exercise.[11,12] When HF is suspected, imaging is important to evaluate for structural or functional cardiac abnormalities that may inform the cause of HF and help guide the approach to treatment.

Permanence of the Diagnosis/Condition

As outlined in the first Universal Definition of Heart Failure,[1] HF progresses in stages from those at risk (stage A) to those with structural heart disease (pre-HF or stage B), those with symptomatic HF (stage C), and those with advanced disease (stage D). Once a diagnosis of symptomatic HF is established, individuals with HF are generally considered to have the diagnosis permanently, even if the clinical condition improves with treatment.

For the purpose of selecting medical treatment, individuals with HF are commonly segregated by LVEF into those with preserved or mildly reduced ejection fraction (EF) and those with reduced EF. There are limitations to phenotype categorization by EF alone (or by specific cutoff values for EF), and there is evolving evidence for the efficacy and safety of therapies across different EF ranges. Acknowledging that EF may change with effective HF treatment, current guidelines recognize a separate subgroup of individuals whose EF increases with time and medical intervention as a separate category of HF with "improved" EF, emphasizing that these individuals may still be at risk for HF events. Although contention remains about the precise numeric thresholds of EF that should be used for HF phenotyping,[13] it is essential to recognize the clinical relevance of incorporating the individual's trajectory in determining the optimal approach to guideline-directed medical therapy and prognosis.

TABLE 1 Stages in the Development and Progression of HF

Stage	Definition
At risk for HF (stage A)	Individuals at risk for HF but without current or prior symptoms or signs of HF and without structural cardiac changes or elevated biomarkers of heart disease At risk: individuals with hypertension, atherosclerotic cardiovascular disease, congenital heart disease, diabetes, obesity, exposure to cardiotoxins, a family history of cardiomyopathy, or genetic carriers at risk for cardiomyopathy Not all will develop HF, but risk factor intervention may be warranted
Pre-HF (stage B)	Individuals without current or prior symptoms or signs of HF with evidence of one of the following: • Structural abnormalities: left ventricular hypertrophy, cardiac chamber enlargement, ventricular wall motion abnormality, myocardial tissue abnormality (eg, evidence of myocardial edema, scar/fibrosis abnormality by T2-weighted cardiac magnetic resonance imaging or late gadolinium enhancement imaging), valvular heart disease • Abnormal cardiac function: reduced left or right ventricular systolic function, evidence of increased filling pressures (by invasive or noninvasive measures), abnormal diastolic function • Elevated natriuretic peptide levels or elevated cardiac troponin levels, especially in the setting of exposure to cardiotoxins
HF (stage C)	Individuals with current or prior symptoms or signs of HF or both caused by a structural or functional cardiac abnormality or both
Advanced HF (stage D)	Severe symptoms or signs of HF or both at rest or with minimal exertion, recurrent hospitalizations despite GDMT, refractory or intolerant to GDMT, requiring advanced therapies such as inotropic support and consideration for cardiac transplantation, mechanical circulatory support, or palliative care

GDMT indicates guideline directed medical therapy; and HF, heart failure.

Definition and Criteria for Stages of HF

The first Universal Definition and Classification of Heart Failure[1] reviewed the American Heart Association/American College of Cardiology staging system[10] and renamed stage B as pre-HF, emphasizing early detection, close monitoring, and proactive management to prevent symptomatic HF.[10,14] This consensus reaffirms the first Universal Definition and Classification of Heart Failure staging system, as shown in Table 1.

Although most of the current guidelines are devoted to the treatment of symptomatic HF (stages C/D), most of the population is without risk factors (stage 0), at risk

(stage A), or in the pre-HF stage (stage B).[15,16] The progression from stage A to D was initially proposed to be unidirectional; that is, after evolving to clinical HF, there is no return to preclinical stages, and the emphasis should be placed on predicting and preventing HF.[14,15,17] However, the first Universal Definition and Classification of Heart Failure staging system also includes HF "remission" with guideline-directed medical therapy and risk factor modification.

Social determinants of health should also be emphasized because they significantly affect HF risk and outcomes, particularly among marginalized communities. Poverty, lack of education, neighborhood deprivation, and social disparities are associated with incident HF, and multiple social vulnerabilities have a cumulative effect on HF risk and prognosis.[18,19] Among the traditional stage A risk factors, hypertension and ischemic heart disease have been among the strongest risk factors for developing clinical HF. Other stage A risk factors include congenital heart disease, chemotherapy exposure, and genetic abnormalities. Special attention should be given to cardiometabolic risk factors, including overweight, obesity, chronic kidney disease, and diabetes, because their prevalence is increasing in several global regions,[20,21] and there are promising new opportunities for the prevention of HF in these conditions.[14,22] Last, the American Heart Association's Life's Simple 7[23] and Life's Essential 8[24] may provide a simple way of recognizing higher-risk individuals, focusing on reducing modifiable risk factors and optimizing health behaviors.[25]

Strategies to reduce the risk of developing HF should be individualized. In individuals at stage A with risk factors for HF such as diabetes or obesity, specific treatment options such as antihypertensive medications and treatments for diabetes[26,27] and obesity,[28] including sodium-glucose-transporter-2 inhibitors, glucagon-like peptide-1-receptor agonists, and the nonsteroidal mineralocorticoid receptor antagonist finerenone (for those with chronic kidney disease and diabetes), have been shown to decrease the risk of developing new-onset HF. From stage A to B, there is a gradient of risk (individuals at stage B are at higher risk of short-term progression to overt HF); thus, more intensive proactive strategies are warranted in individuals at stage B such as treatment with some renin-angiotensin-aldosterone system inhibitors and β-blockers in individuals with asymptomatic left ventricular dysfunction.[29] Although optimal strategies for screening for HF risk have not been well established, data suggest that combining natriuretic peptide screening with other risk factors may identify individuals who can benefit from specific HF therapies even in a presymptomatic stage (stage B HF).[30,31] Other biomarkers such as circulating troponin and urine albumin-creatinine ratio may further enhance population screening in higher-risk groups.[32,33] A promising new screening approach uses artificial intelligence–enabled ECG, which accurately classifies and predicts left ventricular systolic dysfunction[34,35] and structural cardiac disease[36] confirmed by echocardiography. For those without left ventricular systolic dysfunction, a positive artificial intelligence–enabled ECG test is strongly associated with incident HF in the future.[37] However, before these tools become clinically useful, further studies are required to demonstrate their impact in different population groups and on individual outcomes and the feasibility of implementation in clinical practice, especially in resource-limited areas.

Phenotypes of HF

The phenotypic classification of HF has traditionally been based on LVEF and cause.

Classification of HF According to LVEF

LVEF is the key criterion on which HF trial eligibility has been anchored for decades. Despite limitations, classification of HF by LVEF is fundamental for extrapolating efficacy from clinical trials to effectiveness in clinical practice, determining appropriate HF medications/devices, understanding the expected response to therapy, and monitoring individuals with HF. However, the LVEF cutoffs used in the first Universal Definition of Heart Failure were arbitrary, with the known variability of echocardiographic measurements of LVEF, difficulties in interpretating LVEF measured with different imaging modalities, and remaining debate over whether LVEF cutoffs should account for differences by sex, age, and ethnicity.[1,38,39] There is growing evidence that individuals with slightly depressed LVEF may benefit from standard therapies for HF with reduced EF, calling for consideration of whether HF with lower LVEFs should be grouped under HF with reduced EF. Furthermore, HF with higher LVEFs should likely be best called HFpEF.[39,40]

The lower limit of normal LVEF is ≈53% for women and 52% for men, with slightly higher thresholds among individuals of Asian origin. An LVEF <50% is thus unlikely to reflect normal function, regardless of sex, age, or self-reported race and ethnicity.[41] This Second Universal Definition of Heart Failure therefore, moves away from using specific LVEF cutoffs and simplifies the classification to clinically actionable groups:

- HF with reduced EF;
- HFpEF; and
- HF with improved EF, HF with previously reduced LVEF that has now increased/normalized.

The revised simplified grouping facilitates clinical application and allows the clinician to account for

TABLE 2 Selected Recently Proposed Classifications of HF by Cause

Classification (reference)	Pathogenic Classes
European Society of Cardiology HF guidelines 2021[9]	Coronary artery disease; hypertension; valve disease; arrhythmias; cardiomyopathies; congenital heart disease, infective, drug induced, infiltrative; storage disorders; endomyocardial disease; pericardial disease, metabolic; neuromuscular disease
American Heart Association/American College of Cardiology/Heart Failure Society of America HF guidelines 2022[10]	Ischemic heart disease and myocardial infarction; hypertension; valvular heart disease; familial or genetic cardiomyopathies; amyloidosis; cardiotoxicity with cancer or other treatments or substance abuse such as alcohol, cocaine, or methamphetamine; tachycardia, right ventricular pacing, or stress-induced cardiomyopathies; peripartum cardiomyopathy; myocarditis; autoimmune causes, sarcoidosis; iron overload, including hemochromatosis; thyroid disease and other endocrine metabolic; nutritional
Global Burden of Disease[42]	Ischemic cardiomyopathy; pressure overload of the left side of the heart; pulmonary heart disease; valvular and congenital cardiomyopathy; primary myocardial disease; toxic cardiomyopathy; infectious disease; stress, tachycardias, and high-output mediated cardiomyopathy; volume-overload syndromes; other causes

HF indicates heart failure.

potential differences in LVEF by sex, age, and ethnicity in the classification. For instance, the exact LVEF cutoff classifying an individual as having a reduced EF (with potential benefit from HF with reduced EF treatments) may not be identical in every case.

Classification of HF by Cause

The importance of making every effort to diagnose and define the specific cause of HF was emphasized in the first Universal Definition of Heart Failure. In clinical practice, the cause of HF has often been placed into just 2 categories: ischemic and nonischemic cardiomyopathy. This simplistic categorization omits the many causes of dilated and hypertrophic cardiomyopathies and does not acknowledge the impact of identifying specific causes on treatment. Identifying specific treatable causes will guide targeted treatment selection beyond standard HF treatment approaches. A prime example is an individual with cardiac amyloidosis as the underlying cause of HF, who will benefit from targeted treatment of cardiac amyloidosis beyond conventional HF therapies.

Several classifications of HF causes have been proposed (Table 2).[9,10,42] A systematic review of trials and observational evidence of primary HF prevention across 92 putative HF pathogenic risk factors from US and European clinical practice guidelines showed that prognosis in HF depended on the specific causes and number of underlying risk factors and that almost 85% of individuals with HF had at least 1 identifiable pathogenic risk factor amenable to preventive treatment in the 5 years preceding diagnosis.[43] Note that there is substantial variability in case definitions of HF causes used across real-world registries and clinical trials, potentially leading to inconsistencies in primary data collection and challenges with generalization of trial results.[42] To address this, a universal definition of causes in HF is proposed (Table 3).

Classifying cardiomyopathies remains complex because some diseases fit into multiple categories and their characteristics can evolve over time. The detailed subclassification of primary cardiomyopathies is beyond the scope of this document. Cardiomyopathies were first classified in 1980 as diseases affecting the heart muscle, categorized into dilated, hypertrophic, restrictive, arrhythmogenic right ventricular, and unclassified types. In 1996, the World Health Organization expanded this classification by recognizing inflammatory and viral cardiomyopathies as distinct conditions.[44] As understanding of pathophysiological mechanisms progressed, other classification systems emerged such as the American Heart Association's approach, which categorized cardiomyopathies into primary (affecting only the heart) and secondary (involving systemic diseases such as amyloidosis or diabetes).[45] The European Society of Cardiology organized cardiomyopathies based on morphological and functional characteristics and further divided them into familial and nonfamilial types.[46] More recently, the MOGE(S) system was introduced, incorporating morphofunctional phenotype (M), organ involvement (O), genetic inheritance pattern (G), etiologic annotation (E) including genetic defect or underlying disease/substrate, and the functional status (S) of the disease.[47]

It is important to note that the proposed universal classification of HF by causes aims to facilitate standardized reporting of HF causes in both clinical and research settings. The consideration of key HF causes encourages targeted application of appropriate diagnostic and treatment strategies that prevent the development and progression of HF in individuals with specific identifiable causes. Of note, the pathogenic classification is independent of LVEF. With advancements in deep phenotyping, specific examples may be reclassified with better understanding of the underlying pathogenic processes; for instance, some forms of idiopathic

TABLE 3 Proposed Universal Classification of HF by Cause

Pathogenic Group	Specific Examples
Ischemic cardiomyopathy	Ischemic heart disease, myocardial infarction, coronary artery disease
Hypertensive cardiomyopathy	Hypertensive heart disease
Valvular cardiomyopathy	Structural valve disease: calcific aortic valve disease, degenerative mitral valve disease, other nonrheumatic and congenital valvular diseases, rheumatic heart disease
Arrhythmia-related cardiomyopathy	Atrial fibrillation (uncontrolled), tachycardia, dyssynchrony, or premature ventricular contraction–induced cardiomyopathy, right ventricular pacing-induced cardiomyopathy, desmoplakin
Infiltrative cardiomyopathy	Cardiac amyloidosis, hemochromatosis, Fabry disease, glycogen storage disease, neoplastic/cancer-related infiltration
Infective cardiomyopathy	Viral myocarditis, Chagas disease, HIV, Lyme disease
Inflammatory cardiomyopathy	Autoimmune disease, sarcoidosis, hypersensitivity, desmoplakin
Toxic cardiomyopathy	Medication-induced cardiotoxicity, substance use disorders, for example, alcohol, cocaine, amphetamine
Heritable cardiomyopathy	Hypertrophic cardiomyopathy, dilated cardiomyopathy, restrictive cardiomyopathy, arrhythmogenic cardiomyopathy, nondilated left ventricular cardiomyopathy
Pericardial disease	Constrictive and restrictive pericarditis
Metabolic disease and nutritional deficiency–associated cardiomyopathy	Obesity; diabetes; endocrine disorders, for example, thyroid disease; nutritional disease, for example, thiamine, vitamin B_1, and selenium deficiencies; inborn errors of metabolism
Pregnancy-related cardiomyopathy	Peripartum cardiomyopathy
Stress-induced cardiomyopathy	Takotsubo cardiomyopathy
Pulmonary/right-sided heart disease	Chronic obstructive pulmonary disease, interstitial lung disease, coal workers' pneumoconiosis, silicosis, asbestosis, other pneumoconiosis, pulmonary arterial hypertension
Congenital cardiomyopathy	Systemic right ventricular failure, Fontan circulation, repaired tetralogy of Fallot
High-output mediated cardiomyopathy	Hemoglobinopathies, hemolytic anemias, atrioventricular malformations, endocrine causes (eg, pheochromocytoma)
Other causes	Other cardiovascular and systemic disorders, for example, neuromuscular disease, endomyocardial fibrosis, Loeffler endocarditis
Idiopathic	Idiopathic cardiomyopathy

HF indicates heart failure.

cardiomyopathy may be reclassified as genetic/familial cardiomyopathy as underlying genetic variants are discovered. The list of causes (Table 3) represents common examples and is not exhaustive. Some reversible causes can cause transient/acute rather than chronic HF; this is covered later in the Mode of Presentation section.

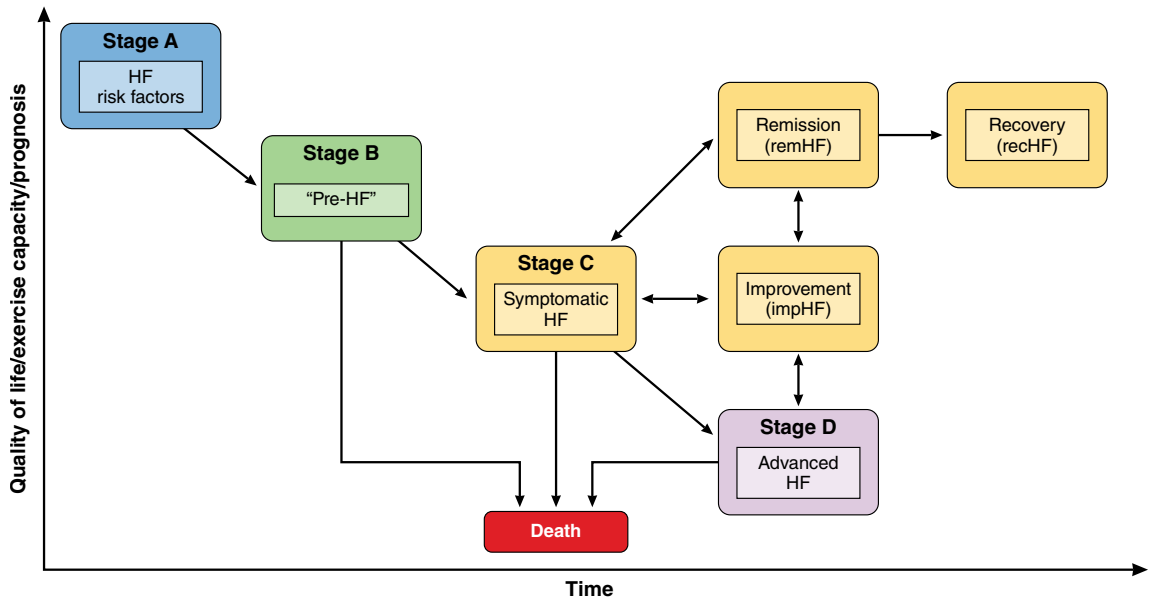
Geographic Variation

With increasing global migration and industrialization, the spectrum of cardiovascular disease (CVD) contributing to HF is changing globally. The spectrum of CVD leading to HF differs significantly between high-income and low- to middle-income countries.[48] As a result of conflict, poverty, and environmental factors, migration to industrialized countries (particularly to those of the Global North) has escalated over the past 2 decades; consequently, the profile of CVD in host countries has changed considerably.[49] It has been found that up to 3% of immigrants arriving in Europe have underlying cardiac disease.[50] Migrant populations could present with CVDs that are unfamiliar to health care workers practicing in Global North countries that include rheumatic heart disease, Chagas disease, endomyocardial fibrosis, tuberculous pericarditis, or peripartum cardiomyopathy. On the other hand, physicians practicing in low-income countries need to adapt to the increase in CVD linked to the so-called Western lifestyle such as coronary artery disease and myocardial infarction, which have previously contributed to <10% of all HF cases in large cohort studies from South Africa[51] and Nigeria[52] published within the past 2 decades.

A systematic review of worldwide risk factors for HF[53] found that ischemic heart disease was the major risk factor for HF in >50% of individuals in Western high-income regions, as well as Eastern and Central European regions. In contrast, it contributed to 30% to 40% of HF cases in East Asia, high-income Asia Pacific regions, Latin America, and the Caribbean. At the lowest end of the scale, in sub-Saharan Africa, ischemic heart disease contributed to <10% of cases. Hypertension was a common contributor in Eastern and Central Europe (35%; range, 32.7%–37.3%) and sub-Saharan Africa (32.6%; range, 29.6%–35.7%). Of the other 2 commonly reported antecedents, rheumatic heart disease was particularly prevalent in East Asia (34%) and sub-Saharan Africa (14%) cases.

Another systematic analysis of the underlying causes of HF in 195 countries and territories from 1990 to 2017 showed significant geographic and sociodemographic variation.[54] Globally, ischemic heart disease accounted for the highest proportion (26.5%) of the age-standardized prevalence rate of HF in 2017, followed by hypertensive heart disease (26.2%) and chronic obstructive pulmonary disease (23.4%). However, chronic obstructive pulmonary disease accounted for the highest proportion of age-standardized prevalence rate of HF in South Asia (38.3%) and East Asia (34.3%). Alcoholic cardiomyopathy was a major cause of HF in Eastern Europe,

FIGURE 1 Trajectories of HF



An individual's heart failure (HF) journey starts left to right, and quality of life or exercise capacity and prognosis become poor as HF stage advances. Every stage carries certain risk of sudden death.

accounting for 16.4% of age-standardized prevalence rate of HF. Chagas disease was a major cause of HF in Andean (7.4%), tropical (6.5%), central (3.5%), and southern (9.4%) Latin America but had almost no impact on HF in other regions.

Geographic variation by LVEF subgroup in the characteristics and contributing factors for individuals has also been described.[55] Individuals from lower- and middle-income regions present with HF at a considerably younger age compared with those from high-income regions, and region-specific phenotypes include lean diabetic HFpEF in Southeast Asia, Chagas disease in Latin America, and hypertensive heart disease and peripartum cardiomyopathy in Africa. Socioeconomic determinants such as country income level and out-of-pocket costs may contribute to geographic differences, but the full extent of the influence of socioeconomic determinants on presentation, management, and outcomes of HF remains poorly understood. There remains a shortage of high-quality data on HF prevalence, causes, and outcomes in many low- and middle-income countries. This gap limits global generalizability of current trial evidence underscored by recent analyses, including the near absence of sub-Saharan African participants in HF trials.[56]

It is important to note that the geographic variation of pathology contributing to HF has implications for the interpretation and generalizability of clinical trial results, design and conduct of future studies, and optimal care for individuals around the world.

Trajectories of HF

HF is a dynamic clinical syndrome with changing clinical trajectory over time based on symptoms, signs, and disease progression or remission (**Figure 1**). Recent guidelines emphasize early identification of HF and targeted interventions, including guideline-directed medical therapy, to alter disease progression.[57]

Optimal HF therapy may induce substantial improvement in cardiac function over time. Patients with HF with improved EF, characterized by antecedent HF with reduced EF and a subsequent ≥10-point increase in LVEF to a new LVEF >40%, illustrate the dynamic nature of HF. However, an exclusively LVEF-based definition insufficiently reflects the broader HF phenotype given the intrinsic variability of LVEF measurement and the frequent persistence of structural abnormalities, biomarker elevation, or residual symptoms.[57]

A more holistic, HF-oriented framework encompassing improvement, remission, and recovery may better characterize these trajectories. Improvement denotes LVEF improvement despite ongoing structural or clinical abnormalities. Remission involves normalized LVEF with

minimal symptoms and stable biomarker profiles while acknowledging persistent vulnerability to relapse. Only a minority reach recovery, defined by sustained normalization of cardiac structure, function, biomarkers, and symptoms over extended follow-up.[57]

This continuum reinforces that LVEF improvement alone does not signify disease resolution. Patients with HF with improved EF remain susceptible to recurrent left ventricular dysfunction and therefore require continued guideline-directed medical therapy and longitudinal clinical surveillance. In select asymptomatic or minimally symptomatic individuals (New York Heart Association class I), a state of HF in remission may be observed, indicating stable but not definitive recovery.

This term is distinct from the misnomer stable HF because individuals with HF always have a residual risk for worsening symptoms, hospitalization, or sudden cardiac death. Individuals with HF who have recurrent hospitalizations and continue to decline despite escalation in therapy are noted to have refractory HF (stage D). These individuals are assessed for advanced therapies, including mechanical circulatory support, cardiac transplantation, and palliative care.

WORSENING HF

Worsening HF represents a pivotal event in the progression of HF and is associated with an unfavorable clinical prognosis. It is characterized by a progressive deterioration in both the symptoms and signs of HF and quality of life in individuals with a prior diagnosis of HF.[58,59] By definition, worsening HF necessitates a prior diagnosis of HF, thereby excluding new-onset HF cases. It also excludes symptom worsening attributed primarily to precipitating events unrelated to HF progression such as acute coronary syndromes, infections, or poor adherence to treatment.[58,59]

The clinical definition of worsening HF encompasses a range of manifestations, including exacerbation of dyspnea and peripheral edema. Worsening HF may also involve the deterioration of related clinical parameters such as malignant arrhythmias, diminished exercise capacity, and subclinical changes indicative of HF severity, including elevated pulmonary pressures, impaired ventricular function (right or left) identified through imaging, and increased levels of biomarkers such as BNP, NT-BNP, and cardiac troponins. Such changes are linked to poorer outcomes.[60-62]

Worsening HF often precipitates unplanned medical interventions, including outpatient and emergency department visits or hospital admissions. However, these health care encounters are not prerequisites for the diagnosis of worsening HF.

Decompensated HF

Worsening HF can include progression to decompensated HF (DHF), a condition defined by the need for escalated care or rescue therapy.[63,64] However, DHF can occur at any time in the HF disease course and is not limited to those with worsening HF. Unlike worsening HF, which represents an event in HF progression, DHF is specifically characterized by the requirement for treatment modification, often during a hospitalization.

DHF may present as either a gradual clinical deterioration over days to weeks, commonly observed in chronic HF, or an acute episode such as acute pulmonary edema or cardiogenic shock.[9,10]

The defining feature of DHF is the necessity for treatment intensification, which typically involves an increase in diuretic dose, the initiation of combination diuretic therapy, and, if not already present, implementation of guideline-directed medical therapy. In more severe cases, further advanced interventions, including escalation of medical therapy, resynchronization therapy, valvular interventions mechanical circulatory support, or heart transplantation, may be required.[64] It is important to note that the subset of individuals meeting DHF criteria represents a more severe progression of worsening HF, with clinical changes demanding therapeutic augmentation or rescue strategies.[63]

The threshold for defining therapeutic escalation can vary according to regional clinical practices and health care resource availability, emphasizing the context-dependent nature of DHF management.

MODE OF PRESENTATION

Acute Presentations

Cardiovascular conditions precipitating acute HF commonly include acute myocardial infarction or acute coronary syndrome, hypertensive emergency, arrhythmias, and pulmonary embolism.

Individuals with acute myocardial infarction and acute coronary syndrome complicated by HF may recover with timely treatment strategies without progression to chronic HF, but some may progress to chronic HF. Specific trials have been conducted in this setting.[29,65,66] Hypertensive emergencies encompass a spectrum of clinical presentations that can lead to acute left ventricular dysfunction, pulmonary edema, or both. Arrhythmia-induced cardiomyopathy encompasses tachycardiomyopathy, atrial fibrillation–induced cardiomyopathy, and premature ventricular ectopic beat-induced cardiomyopathy.[67-69] Acute valvular heart diseases can cause HF, and pulmonary embolism can cause acute isolated right HF.

In addition, acute noncardiovascular diseases such as kidney injury, liver failure, and respiratory failure may

lead to acute HF as a result of volume overload and neurohormonal compensatory mechanisms involved in some of these conditions. Symptoms and signs of HF typically resolve once the underlying primary cause is treated.

Chronic Presentation

Subacute or indolent presentations of HF are common. These are characterized by a gradual onset of symptoms that may evolve over weeks to months, often making initial diagnosis more challenging. Patients may experience subtle signs such as increasing fatigue, mild exertional dyspnea, or gradual weight gain caused by fluid retention rather than the dramatic symptoms seen in acute decompensation. Because these changes can be mistaken for deconditioning or normal aging, they may be overlooked by both patients and clinicians. Data from population studies demonstrate that substantial numbers of patients in primary care are treated with chronic diuretic therapy without a formal diagnosis of HF.[70] Randomized trial data support the use of natriuretic peptide screening to improve identification of chronic HF in primary care settings.[30,31]

HF Mimics

HF mimics are conditions that are not consistent with an HF diagnosis and are not pathophysiologically dependent on neurohumoral activation that originates from the myocardium.

Coronary Artery Disease

In approximately half of the HF population (across the EF spectrum), HF is a consequence of coronary artery disease. Myocardial ischemia can serve as an HF mimic, as a condition occurring jointly with HF or a cause of DHF. Although chest discomfort is the leading symptom of chronic coronary syndromes, 10% to 15% of individuals report only exertional dyspnea.[71] In scenarios in which chronic coronary syndromes and HF may coexist, determination of the impact of these 2 conditions on symptoms will depend on the clinical likelihood of obstructive coronary artery disease, in conjunction with 12-lead ECG and echocardiography, followed by further testing for obstructive and nonobstructive coronary artery disease. In parallel, HF diagnostic algorithms should be used to elucidate the contribution of HF to the symptoms.

Chronic Kidney Disease

Individuals with chronic kidney disease may develop dyspnea, exertional limitations, and peripheral edema resulting from inadequate diuresis or worsening disease, which could be accompanied by abnormalities of cardiac structure or function (eg, left atrial enlargement, left ventricular hypertrophy, or diastolic dysfunction secondary to hypertension) and elevated biventricular filling pressures.

Pregnancy

Dyspnea, exertional limitations, and peripheral edema also commonly occur in the third trimester of pregnancy. In this scenario, natriuretic peptides and echocardiography can be useful in excluding a myocardial cause for the presentation. Investigation to exclude peripartum cardiomyopathy or the unmasking of preexisting cardiomyopathy is crucial.

Obesity and Deconditioning

Obesity and deconditioning are often accompanied by dyspnea, exertional intolerance, and peripheral edema. Determining a myocardial cause for dyspnea can be challenging with concurrent obesity as a result of reduced specificity of the physical examination and falsely normal natriuretic peptides even when HFpEF is present. Invasive exercise hemodynamics may be key to determining whether HF is present.

CONCLUSIONS

The diagnosis and treatment of individuals with HF is an evolving field of research, clinical practice, and a deeper understanding of the diverse phenotypes and cause that characterize this complex syndrome. The Second Universal Definition of Heart Failure builds on the foundational work of the first definition by refining the definitions of HF stages, emphasizing the importance of early detection and individualized risk reduction, and introducing a universal classification of HF causes, as well as acknowledging geographic variation of causes.

A major advancement in this update is the move away from LVEF cutoff values, instead advocating for clinically actionable groupings that better reflect the spectrum of HF presentations and allow more personalized care. The recognition of HF trajectories—improvement, remission, and recovery—underscores the dynamic nature of the disease and the need for ongoing surveillance and tailored therapy, even for patients who demonstrate significant improvement.

This document also highlights the profound impact of social determinants and geographic variation on HF risk, presentation, and outcomes. Addressing these disparities is essential for achieving equitable care and improving global health outcomes. The inclusion of a comprehensive table on HF causes provides a valuable tool for clinicians and researchers, facilitating targeted diagnostic and therapeutic strategies.

Last, the recognition of HF mimics and the importance of distinguishing them from true HF syndromes reinforce the need for vigilance and a holistic approach to patient assessment. As our understanding of HF continues to grow, this universal definition serves as a living framework, one that will adapt with future discoveries and innovations, always with the goal of improving patient care and outcomes.

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DISCLOSURES

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Writing Group Member	Employment	Research Grant	Other Research Support	Speakers' Bureau/ Honoraria	Expert Witness	Ownership Interest	Consultant/ Advisory Board	Other
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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives EUR 10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns EUR 10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.
*Modest.
†Significant.

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*Modest.
†Significant.