# Comprehensive Heart Failure History

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Leah A. Carr, Lisa D. Rathman, and Roy S. Small

## 3.1 Introduction

In 2021, the Heart Failure Society of America, Heart Failure Association of the European Society of Cardiology, and the Japanese Heart Failure Society proposed a new universal definition of heart failure: "Heart failure is a clinical syndrome with symptoms and/or signs caused by a structural and/or functional cardiac abnormality and corroborated by elevated natriuretic peptide levels and/or objective evidence of pulmonary or systemic congestion" [1]. The authors defined a new classification of heart failure based on left ventricular ejection fraction (LVEF), describing normal or less than normal ejection fraction (EF) [1]. Patient reported symptoms and clinical manifestations of heart failure are similar regardless of their ejection fraction [1]. The underlying pathophysiology of the subtypes of heart failure is vastly different and dictates the evaluation and ultimately the treatment [1, 2].

Patients with risk factors should be screened periodically by their primary care providers for clinical symptoms associated with heart failure. Mild myocardial dysfunction and structural changes can exist for years without being clinically detected [3, 4]. A comprehensive clinical history and symptom assessment is essential because early recognition and intervention can prevent adverse outcomes [4, 5]. The American College of Cardiology and American Heart Association developed a staging system for heart failure through which most patients will progress during the course of the disease process [5]. Disease progression through the heart failure

All authors are from The Heart Group of Lancaster General Health/PENN Medicine.

L. A. Carr · L. D. Rathman (✉) · R. S. Small
The Heart Group of Lancaster General Health/PENN Medicine, Lancaster, PA, USA
e-mail: Leah.Seitz1@pennmedicine.upenn.edu; lisa.rathman@pennmedicine.upenn.edu; roy.small@pennmedicine.upenn.edu



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stages can be delayed and perhaps prevented, but generally, not reversed [5]. Once structural disease has been established, there is rarely a mechanism for complete correction [4]. Progression from one stage to the next is clinically relevant as it is associated with a reduction in overall survival (Table 3.1) [5].

Cardiomyopathies are defined as changes in the myocardium secondary to metabolic, mechanical, or electrical dysfunction within the heart [4]. Cardiomyopathies can be classified into three main subgroups with the different etiologies falling into one of these categories (Table 3.2) [2, 4, 6].

**Table 3.1** ACC/AHA stages of heart failure [5]

| Stage | Definition | Examples |
|---|---|---|
| A | At risk for heart failure: no structural changes/functional heart disease or abnormal biomarkers and no past or present signs and symptoms of heart failure | • Hypertension<br>• Diabetes/metabolic syndrome<br>• Obesity<br>• Atherosclerotic vascular disease<br>• Substance abusers (alcohol, illicit drugs)<br>• Family history of cardiomyopathy<br>• Exposure to cardiotoxic agents |
| B | Pre-heart failure: structural heart changes or evidence of increased filling pressures but no signs or symptoms of heart failure | • Previous MI<br>• Left ventricular hypertrophy/remodeling<br>• Valvular disease |
| C | Symptomatic heart failure: patients with current or previous symptoms/signs of HF | • Heart failure signs and symptoms<br>• Symptoms of heart failure at rest/activity despite guideline directed medical therapy |
| D | Advanced heart failure: refractory, end stage heart failure | • Marked heart failure symptoms at all times<br>• Recurrent hospitalizations and decompensations |

**Table 3.2** Three major cardiomyopathy categories and most common etiologies [2, 4, 6]

| Cardiomyopathy | Common etiologies |
|---|---|
| Dilated cardiomyopathy | • Idiopathic<br>• Peripartum<br>• Ischemic<br>• Infectious (viral, bacterial, parasitic)<br>• Alcohol, illicit substances, toxins<br>• Chronic persistent tachycardia-(metabolic)<br>• Developmental (such as non-compaction) or familial (for example, arrhythmogenic right ventricular dysplasia)<br>• Autoimmune<br>• Valvular (mitral or aortic regurgitation) |
| Hypertrophic cardiomyopathy | • Longstanding, persistent hypertension<br>• Hypertrophic obstructive cardiomyopathy<br>• Small vessel disease from diabetes mellitus |
| Restrictive or infiltrative cardiomyopathy | • Amyloidosis<br>• Sarcoidosis<br>• Hemochromatosis<br>• Scleroderma |

There may be an overlap within these subtypes. For example, both amyloidosis and longstanding uncontrolled hypertension can present as dilated cardiomyopathies. Valvular disease can present as dilated cardiomyopathy or restrictive depending on the specific lesion.

## 3.2 History: Etiology and Precipitating Factors of Heart Failure

### 3.2.1 Risk Factors

It is important to assess all common risk factors associated with the development of heart failure. This history section will give a closer look into the topics that are imperative to consider when gathering a history for someone who is suspected of having heart failure.

Early identification and modification of risk factors can prevent the development and progression of heart failure [5]. Some of the most common risk factors that lead to the development of left ventricular (LV) dysfunction include advancing age, obesity, hypertension, dyslipidemia, diabetes, obstructive sleep apnea, and alcohol or illicit substance use [4]. This section will review factors that are important to consider when taking a history of a patient suspected of having heart failure.

### 3.2.2 Coronary Artery Disease (CAD)

Myocardial ischemia should be considered in all patients presenting with new onset of heart failure [2, 4, 6]. Approximately 50% of patients diagnosed with heart failure have an underlying ischemic cardiomyopathy [7]. Patients with a new diagnosis of heart failure should be assessed for signs and symptoms of CAD [2, 4, 5, 7]. Risk factors should be addressed and signs/symptoms evaluated [3].

### 3.2.3 Valvular Heart Disease

Mitral regurgitation (MR) is a frequent cause of heart failure which may be the result of a structurally abnormal valve (primary) or due to annular dilatation with incomplete coaptation of the valve leaflets (secondary or functional MR) [8]. A history of rheumatic fever should prompt consideration of valvular heart disease. Aortic stenosis may be due to a congenital defect (bicuspid aortic valve) or degenerative (calcific) disease [2, 3, 9]. Endocarditis may cause severe valve dysfunction and is a particular concern in patients with a history of intravenous drug abuse or an indwelling catheter [3].

### 3.2.4 Hypertension

Patients with a longstanding history of persistent or untreated hypertension are at an increased risk for developing a hypertensive cardiomyopathy [2]. These hearts remodel due to the longstanding increased afterload by increasing LV wall thickness and mass [2]. They develop restrictive filling and may manifest as heart failure with preserved EF (HFpEF) as the disease progresses [3].

### 3.2.5 Endocrine

Numerous endocrine conditions are associated with LV dysfunction and heart failure. Diabetes mellitus (DM) (Types 1 and 2), hypo or hyperthyroidism, growth hormone excess, pheochromocytoma, hyperaldosteronism, and Cushing's syndrome are all potential causes of LV dysfunction and heart failure [2, 4, 10]. Patients who have diabetes mellitus are prone to developing coronary artery disease and resultant myocardial ischemia [5, 11]. DM is also a risk factor for the development of HFpEF [11, 12]. Uncontrolled diabetics tend to have more frequent heart failure decompensations due to hyperosmolar stress and increased infection risk [3, 11, 12].

### 3.2.6 Pregnancy

Heart failure and left ventricular dysfunction can occur in both the peripartum and postpartum phases of pregnancy [2, 13, 14]. If it occurs within the first year after the delivery of a child, it is termed postpartum cardiomyopathy. These women tend to have no history of prior heart disease or peripartum preeclampsia [3, 13, 14]. Women with peripartum cardiomyopathies frequently recover within the first 6 months. Those who do not recover are advised against additional pregnancies [3, 13, 14].

### 3.2.7 Family History/Genetics

Approximately 10–15% of heart failure patients have a genetic mutation likely to be related to their cardiomyopathy [2–4]. Heritable cardiomyopathies include hypertrophic obstructive cardiomyopathy, Fabry's disease, or muscular dystrophies including the laminopathies [15–18]. It is very important for those who have a family history of sudden cardiac death to have a cardiac evaluation as well as genetic testing if indicated [2, 3]. Hereditary TTR amyloid is due to a genetic mutation, which regulates the metabolism and structure of transthyretin [18]. It is important for family members of affected individuals to undergo genetic testing [2, 16].

### 3.2.8 Illicit Substances and Toxic Agents (Chemotherapy, Drugs, Alcohol)

A crucial part of history taking for newly identified cardiomyopathies is to identify past and present alcohol consumption and/or illicit drug use [19]. Alcohol is directly cardiotoxic and chronic consumption of excessive alcohol can cause an alcohol-induced cardiomyopathy [5, 19]. Similarly, drug-induced cardiomyopathies are seen with long-term methamphetamine, cocaine, and other stimulant use, which can directly cause myocardial remodeling and dysfunction, as well as induce LV dysfunction through coronary artery disease [2, 3, 19]. There is about a 35% chance that a cardiomyopathy due to excessive alcohol consumption will resolve if the patient can abstain from drinking [6]. Chapter 16 of this book delves further into a review of alcohol and drug inducted cardiomyopathies.

Chemotherapy agents pose a significant risk for both acute and chronic myocardial damage. Some commonly used drugs that contribute to myocardial dysfunction are anthracyclines (such as doxorubicin or Adriamycin) or cyclophosphamide (Cytoxan) [2, 4, 19]. Comorbidities such as advanced age, preexisting heart disease, or prior radiation increase the risks associated with chemotherapy [3, 19]. Cardiotoxicity may be a direct effect of the drug (for example, anthracyclines, tyrosine kinase inhibitors, or monoclonal antibodies) or a secondary effect from vascular damage and cardiac ischemia (fluorouracil) [19]. Some chemotherapy agents can cause cardiac arrhythmias, myocarditis, or pericarditis [19]. Immune checkpoint inhibitors are monoclonal antibodies, which target host immune regulation receptors and can precipitate acute myocarditis [20, 21].

### 3.2.9 Myocarditis

Acute myocarditis may be a result of a viral infection (SARS COVID-19 or more traditional viruses) or an inflammatory process (giant cell myocarditis or sarcoidosis) [2, 20–23]. Most viral myocarditis cases are sequelae from upper respiratory or gastrointestinal illnesses [20, 23]. It is important to establish the connection with a prior viral illness as it may allow for more direct serologic testing and specific diagnosis [20]. Evidence of myocardial inflammation has been found in 2–3% of college athletes recovering from COVID infection [24]. There have been rare case reports of mostly younger adults with myocarditis or pericarditis associated with the mRNA vaccines with reports of four to five cases per one million vaccinations [25]. Approximately 50% of patient with an acute viral myocarditis will recover their cardiac function within 6–12 months of their index diagnosis [6]. HIV, parasites, Chagas, bacterial, and fungal infections can also cause acute myocarditis [3, 20].

### 3.2.10 Connective Tissue and Systemic Disorders

Autoimmune diseases which can lead to cardiomyopathies include systemic lupus erythematosus, scleroderma, and polymyositis [2, 4]. These patients will often present with heart failure in the setting of preserved left ventricular function [3].

### 3.2.11 Anemia

Anemia is a highly correctable cause of heart failure [26]. Anemia secondary to iron deficiency is a common condition that can cause heart failure exacerbations [26]. It is important to evaluate and treat the underlying etiology [26, 27]. Untreated severe anemia causes increased myocardial oxygen demand as well as increases peripheral tissue oxygen demand to meet metabolic oxygen requirements [3, 26].

### 3.2.12 Nutritional Deficiencies

Nutritional deficits such as thiamine deficiency can lead to the development of a dilated cardiomyopathy and heart failure [28, 29]. Thiamine insufficiency can occur among individuals who are on fad diets, as well as those who have prolonged hospitalizations with inadequate nutritional support [28, 29]. There are two types of thiamine deficiency: dry beriberi and wet beriberi. Dry beriberi manifests as primarily neurological complications, whereas wet beriberi involves cardiac deficits [28, 29]. The cardiovascular complications with wet beriberi include low cardiac output failure, systemic vasodilation, peripheral edema, and fluid retention [28, 29]. The focus of management for thiamine deficient patients with heart failure needs to be normalization of this nutritional abnormality with adequate supplementation of thiamine, which is available in both intravenous and oral formulations [28].

### 3.2.13 Arrhythmias

Patients with incessant, uncontrolled tachycardias can develop a dilated cardiomyopathy [5]. It is typically the supraventricular tachycardias such as uncontrolled atrial fibrillation or flutter that lead to cardiac remodeling [12, 30, 31]. Ventricular tachycardia can occur in patients with dilated cardiomyopathies and heart failure [5]. Persistent frequent ventricular ectopy is associated with LV dysfunction. Patients with tachycardia-induced cardiomyopathies often have reversibility of their cardiac dysfunction if successfully controlled [3, 5, 12, 30, 31].

### 3.2.14 Idiopathic

After a comprehensive medical work-up is completed, there are still times when a definitive etiology or causative factor cannot be identified [2]. These cases are termed idiopathic cardiomyopathies and account for 10–20% of all heart failure cases [3].

In summary, an all-inclusive health history is essential in the setting of any new heart failure diagnosis. Subsequent history taking at all future clinic visits should be completed to ensure the patient's heart failure is controlled and properly treated in order to prevent future exacerbations.

## 3.3 History: Symptoms of Heart Failure

An all-inclusive history of symptoms is essential to make a prompt diagnosis of heart failure. No single historical element or symptom has been proven to be diagnostic of heart failure [5]. A comprehensive history will aid in determining the acuity, etiology, and progression of heart failure.

Symptoms commonly observed in heart failure patients include those due to congestion from excess fluid accumulation and reduced cardiac output (Table 3.3) [5, 32].

The most common symptoms heart failure patients report are dyspnea and fatigue. Dyspnea is reported in >50% of heart failure patients and is the most common complaint in the hospitalized subset of patients [33, 34]. Dyspnea and fatigue are nonspecific with a broad spectrum of differential diagnoses. In the heart failure patient in particular, dyspnea and fatigue are due to congestion and low cardiac output, respectively.

**Table 3.3** Common symptoms of heart failure [5, 32]

| Congestion (excess fluid volume) | Reduced cardiac output |
|---|---|
| • Dyspnea (rest or exertional) | • Fatigue |
| • Paroxysmal nocturnal dyspnea | • Nausea |
| • Edema | • Weakness |
| • Orthopnea | • Early satiety or anorexia |
| • Early satiety or anorexia | • Decreased exercise tolerance |
| • Cough | • Poor concentration or memory |
| • Abdominal bloating | • Sleepiness |
| • Weight gain | • Unexplained weight loss |
| • Abdominal or epigastric discomfort | • Muscle wasting |
| • Nausea | • Malaise |
| • Chest discomfort | • Sleep disturbance (Cheyne–stokes respiration) |
| • Bendopnea | |

Fatigue and exercise intolerance affects nearly 85% of all heart failure patients [33, 34]. The cause is often multifactorial and difficult to treat [33]. In the heart failure patient, orthopnea is highly suggestive of congestion with a high sensitivity rate [35]. In the ESCAPE trial, orthopnea (≥2 pillow) was an indicator of elevated pulmonary capillary wedge pressure [32]. Paroxysmal nocturnal dyspnea is another reportable symptom that is commonly seen in the volume-overloaded patient [34, 36]. Both orthopnea and paroxysmal nocturnal dyspnea have a high specificity [33]. The absence of either of these symptoms has a high negative predictive value.

Bendopnea is a novel heart failure symptom first defined by Thibodeau et al. in 2014 [37]. It occurs when a sitting patient develops dyspnea within 30 s of bending at the waist to touch his or her feet [37]. Several clinical trials have demonstrated that bendopnea is associated with increased cardiac filling pressures and risk for heart failure hospitalization [37, 38]. Bendopnea is not diagnostic for heart failure alone and can occur in patients with pulmonary disease or morbid obesity [37].

Peripheral edema due to right heart congestion is another common feature of heart failure reported by >50% of patients [34]. It typically develops gradually with >5 L of excess fluid before pitting edema is seen [39]. Generally, low albumin or sitting with legs not extended is associated with more prominent edema. Edema can vary from mild ankle or foot swelling to significant swelling of the legs, scrotum, abdomen, sacrum, and periorbital space. It may help in judging treatment response to grade the degree of pitting [1, 3–5] as well as the extent (for example, ankle vs extending to the knee or thigh). Peripheral edema is not specific to heart failure alone and can occur due to other conditions such as venous insufficiency, liver cirrhosis, or chronic lymphedema [34].

Gastrointestinal complaints such as nausea, abdominal bloating, early satiety, and anorexia are commonly reported by heart failure patients [33]. These complaints may stem from low cardiac output due to poor gut perfusion or fluid volume overload and vascular congestion in the peri-abdominal space.

During each patient encounter, it is important to re-evaluate patient symptoms to assess for progression or improvement as a result of therapy. A careful interim history may prevent heart failure hospitalizations and disease advancement. Some patients have a tendency to minimize their symptoms, which can sometimes be discerned with careful questioning or confirmation with other household members [40]. Patients will unconsciously alter their daily activity to avoid symptoms or dismiss their limitations as a normal result of aging or reduced fitness. Multiple factors such as including age, mentation, and comorbid conditions may influence a patient's ability to recognize early symptoms of heart failure [40].

## 3.4 History Taking: Assessment of Symptom Severity

A comprehensive assessment of symptoms is important to determine a patient's functional limitations. The New York Heart Association functional class helps clarify the severity of patient symptoms (Table 3.4) [5].

**Table 3.4** New York Heart Association (NYHA) functional class

| | |
|---|---|
| NYHA class 1 | No limitation in physical activity |
| NYHA class 2 | Slight limitation in physical activity |
| NYHA class 3 | Marked limitation in physical activity |
| NYHA class 4 | Symptoms at rest; inability to carry out any physical activity without shortness of breath or discomfort |

Adapted from nomenclature and criteria for the diagnosis of diseases of the heart and great vessels. 9th ed. Little, Brown, and company [41]

The American Heart Association and American College of Cardiology (AHA/ACC) recommends patient management and treatment based on patient's AHA/ACC stage (Table 3.5) and NYHA functional classification [5]. Patients with ACC/AHA stage C and D heart failure should be assigned a NYHA class at baseline and with each subsequent patient encounter, as the patient's functional status will change over time [5]. Worsening NYHA functional class is associated with increased morbidity and mortality [1]. Providers should target management and interventions to improve patient symptoms and quality of life. Guideline directed medical therapy will mitigate disease progression and improve prognosis.

## 3.5 Sample History Taking: Etiology, Risk Factor Assessment, and Symptoms

Mr. HF is a 58 year male with a history of myocardial infarction 5 years ago with a stent to his right coronary artery, hypertension, hyperlipidemia who presents to his primary care office with vague complaints of fatigue and decrease in exercise tolerance. He has a family history of ischemic heart disease in his paternal family line. He has no significant history of autoimmune disease, connective tissue disorders, anemia, alcohol/illicit substance abuse, or recent viral illnesses. Table 3.5 highlights additional history questions and symptoms to address during the encounter with Mr. HF.

**Table 3.5** Heart failure clinical history pertinent questions/review of systems to explore

| | |
|---|---|
| Cardiovascular | • Chest pain or pressure<br>• Angina<br>• Palpitations or irregular heartbeat |
| Pulmonary | • Shortness of breath at rest<br>• What activities cause dyspnea on exertion? How many flights of stairs before dyspnea occurs?<br>• What is the most strenuous activity you are able to do?<br>• Paroxysmal nocturnal dyspnea<br>• Orthopnea (Do you sleep in bed? How many pillows do you use at night or do you need to prop yourself up to sleep?)<br>• Snoring or witnessed apnea by significant other? Have you been diagnosed or tested for sleep apnea?<br>• Cough |
| Gastrointestinal | • Early satiety/anorexia<br>• Abdominal bloating<br>• Abdominal pain<br>• Constipation/diarrhea<br>• Nausea/vomiting |
| Neurologic | • Anxiety/depression<br>• Confusion |
| Renal | • Nocturia |
| General symptoms | • Recent weight loss/gain<br>• Fatigue/weakness<br>• Daytime sleepiness<br>• Edema |
| General history | • Tobacco use<br>• Illicit drug use<br>• Alcohol intake<br>• Current medications/OTC including PRN use of nitroglycerin<br>• Regular exercise<br>• Pertinent family history |

## 3.6 Conclusions

Heart failure is a progressive and chronic illness. Patients with heart failure suffer substantial symptoms such as shortness of breath and edema, which impact patient quality and duration of life. A thorough assessment of patient's history and symptoms is essential not only for a timely diagnosis but ongoing clinical management to improve outcomes.

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