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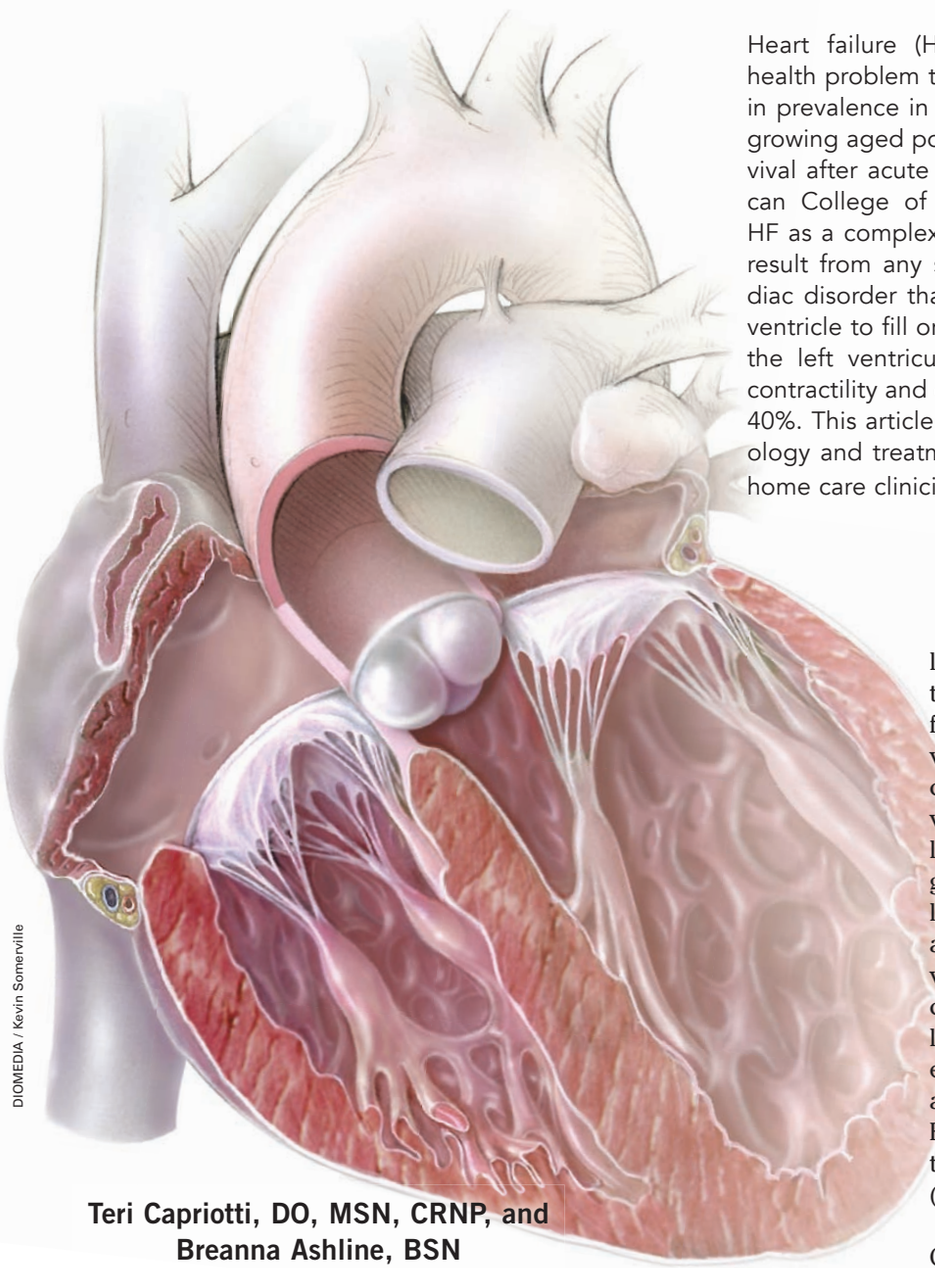
SYSTOLIC HEART FAILURE

An Update for Home Healthcare Clinicians



DIOMEDIA / Kevin Somerville

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Heart failure (HF) is a significant public health problem that is projected to increase in prevalence in the next 20 years due to a growing aged population and improved survival after acute cardiac events. The American College of Cardiology (2019) defines HF as a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill or eject blood. In systolic HF, the left ventricular muscle has diminished contractility and ejection fraction is less than 40%. This article will review the pathophysiology and treatment of HF. Implications for home care clinicians are discussed.

Heart failure (HF) is a major public health problem, with a prevalence of over 5.7 million in the United States (Centers for Disease Control and Prevention, 2019). As it most commonly occurs in individuals over age 65, prevalence is increasing due to a growing older adult population. Also, many patients are surviving acute cardiovascular events with surgical procedures, endovascular interventions, and more effective medications. As a result, the prevalence of HF is projected to increase to 8 million people by 2030 (Heidenreich et al., 2013).

The American College of Cardiology (2019) defines

HF as a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill or eject blood. The ability of the left ventricle to eject blood, also called the left ventricular ejection fraction, is key in the definition of HF. Heart failure is differentiated into two distinct types according to the left ventricular ejection fraction: HF with reduced ejection fraction (systolic HF) and HF with preserved ejection fraction (diastolic HF). This article focuses on systolic HF.

Heart failure occurs because cumulative insults to the myocardium cause weakening of the left ventricle. Major risk factors for HF include coronary artery disease, hypertension, hypercholesterolemia, diabetes, smoking, obesity, heart valve disease, familial history of heart disease, cardiomyopathies, and exposure to cardiotoxic agents such as alcohol, cocaine, amphetamines, cancer treatment, and radiation (Ponikowski et al., 2016; Yancy et al., 2013).

Among Medicare patients hospitalized for heart failure between 2008 and 2010, 67% experienced readmission at some time during their disorder.

Ejection fraction refers to the volume of blood pumped forward from the left ventricle with each contraction (also known as stroke volume). Systolic HF occurs when the left ventricular muscle has reduced contractility and cannot pump adequate stroke volume that causes decreased cardiac output (Barrett et al., 2015). Normal left ventricular ejection fraction is greater than 50% of its total blood volume. In systolic HF, less than 40% of blood volume is ejected per ventricular contraction. This lack of sufficient blood flow pumped into the aorta triggers compensatory neurohormonal mechanisms, which include the sympathetic nervous system and renin–angiotensin–aldosterone system. Although these processes work to counterbalance the effect of low ventricular ejection, they create a cycle of worsening HF (Mazurek & Jessup, 2017).

Pathophysiology

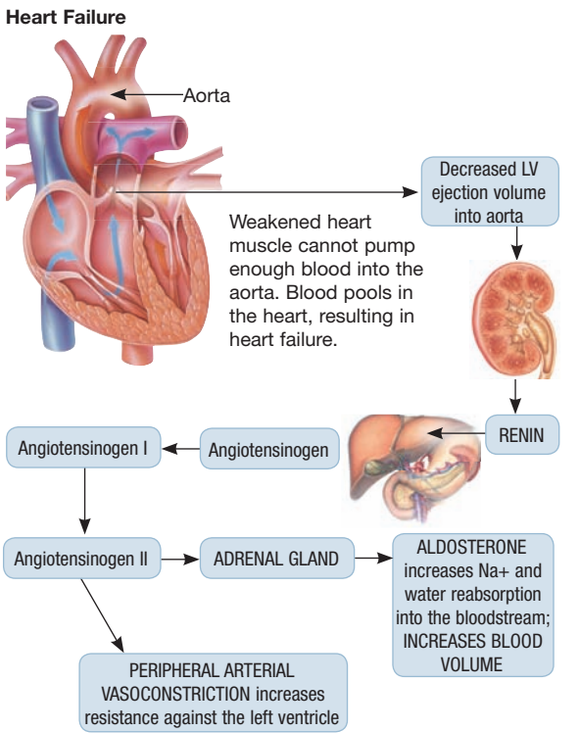
With reduced ejection fraction, decreased blood volume is pumped from the left ventricle into the aorta. Aortic pressure initially drops along with systemic arterial pressure. At the kidney, this decreased arterial pressure stimulates secretion of renin that triggers the renin–angiotensin–aldosterone system (RAAS). Renin circulates within the bloodstream and stimulates the liver to release angiotensinogen. Angiotensinogen is cleaved into angiotensin I and other protein components. In the lungs, angiotensin I is converted into angiotensin II by angiotensin-converting enzyme (ACE). Angiotensin II is a potent arterial vasoconstrictor and stimulates the adrenal gland. At the adrenal gland, aldosterone is secreted and causes increased sodium and water reabsorption into the bloodstream. The blood volume rises and blood pressure increases. Due to the RAAS, arterial vasoconstriction increases the resistance against the left ventricle that also has to pump out the increased blood volume (Figure 1). These changes increase strain on the weakened left ventricle, which can worsen HF (Katz, 2018).

Simultaneously, systolic HF causes low blood pressure within the arteries that stimulates baroreceptors. Baroreceptors sense low blood pressure and activate the sympathetic nervous system that causes arterial vasoconstriction and increased heart rate (Figure 2.). The left ventricle has to contend with increased resistance due to the arterial vasoconstriction and also increases its rate of pumping.

Although activation of the RAAS and sympathetic nervous system are compensatory mechanisms, they cause further weakening of the left ventricular pump. The RAAS increases blood volume that increases preload, which then causes congestion of capillary beds. Excessive hydrostatic pressure in the capillary beds leads to pulmonary edema, gastrointestinal, hepatic, and splenic venous congestion as well as peripheral edema in the ankles. Afterload, which is the resistance against the heart, is increased due to widespread arterial vasoconstriction. This causes excess workload and increased wall stress of the heart (Kumar et al., 2018).

Elevated wall stress leads to secretion of B type natriuretic hormone (BNP) and atrial natriuretic peptide (ANP) that cause a compensatory natural diuresis in an attempt to decrease blood volume. BNP is a biomarker that is measured to confirm diagnosis of HF (Mazurek & Jessup, 2017). Neprilysin, another enzyme, secreted by the kidney, is a key component in the process

Figure 1.
The Renin-Angiotensin-Aldosterone System



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of HF. Neprilysin causes breakdown of BNP and ANP. It is currently under intense scrutiny in pharmacologic studies. Inhibition of neprilysin can allow longer action of the natural diuretic peptides BNP and ANP (Jhund & McMurray, 2016). During HF, endothelin and inflammatory cytokines are also released that contribute to left ventricular remodeling. These are also under investigation for pharmacologic strategies in managing HF (Kumar et al., 2018).

Clinical Manifestations

A thorough past medical history and physical examination are essential in the diagnosis of HF. According to McMurray (2012), the diagnosis of systolic HF requires three conditions: symptoms typical of HF, signs typical of HF, and reduced ejection fraction. Common signs and symptoms are listed in Table 1 and Table 2. Plasma concentrations of BNP or the N-terminal prohormone of BNP are key biomarkers in the diagnosis of HF. Echocardiography is an essential procedure in the diagnosis. Transesophageal echocardiography is commonly done as it can detect structural abnormalities, left ventricular hypertrophy, left atrial enlargement, or signs of diastolic dysfunction. Echocardiography can also assess concomitant valve disease, right ventricular function, and systolic pulmonary artery pressure, all of which affect prognosis.

The New York Heart Association Classification is commonly used to identify the stage and severity of HF. In stage I, there are no symptoms of HF with ordinary activity. In stage II, there are mild symptoms with ordinary activity, and in stage III, there are symptoms with minimal exertion. Stage IV is severe HF marked by symptoms at rest (American Heart Association, 2018).

Treatment

Diuretics

Pharmacologic treatment of systolic HF is mainly based on diuretics to relieve symptoms associated with congestion. Diuretics, both loop and thiazide, reduce blood volume and increase excretion of water in the urine. Pulmonary congestion is significantly reduced with use of diuretics and patients' breathing is eased (Chavey et al., 2017).

ACE Inhibitors and Angiotensin Receptor Blockers

ACE inhibitors are the cornerstone of HF treatment. They block the transformation of angiotensin I to angiotensin II. Angiotensin receptor blockers perform similarly. By blocking angiotensin II receptors, angiotensin II cannot bind to the receptor and in turn cannot stimulate arterial vasoconstriction or trigger adrenal release of aldosterone (Pfeffer et al., 2003).

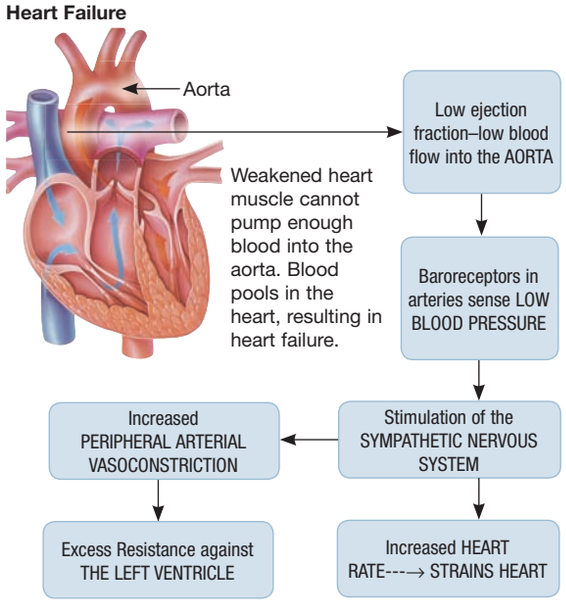
Beta$_1$-Adrenergic Blockers

Beta$_1$-adrenergic blockers are also indicated as these drugs counteract the sympathetic nervous system effects in HF. These agents lower heart rate and decrease peripheral arterial vasoconstriction. Beta-blockers are effective at reducing mortality in patients with symptomatic HF when combined with ACE inhibitors (Chavey et al., 2017).

Aldosterone Antagonists

Aldosterone antagonists, such as Spironolactone and Eplerenone, block the action of aldosterone

Figure 2.
Baroreceptors are stimulated by low circulation to stimulate the Sympathetic Nervous System.



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that reduces sodium and water reabsorption at the kidney. These drugs are recommended in combination with ACE inhibitors and beta$_1$-blockers (Zannad et al., 2011).

Ivabradine

When beta-blockers are not effective in reducing heart rate, Ivabradine is recommended. Ivabradine decreases heart rate by directly inhibiting the sinoatrial node, and is indicated in patients with an ejection fraction of 35% or less and persistent symptoms despite maximally tolerated doses of a beta1-blocker (Chavey et al., 2017).

Digoxin

Digoxin should be considered for those who remain symptomatic despite therapy with other agents. It works as a positive inotrope but can also stimulate dysrhythmias. In a large controlled trial of digoxin in patients with HF, digoxin had no impact on mortality but decreased hospitalization rates (Ambrosy et al., 2014). Further analysis of digoxin has revealed increased morbidity and mortality with digoxin levels greater than 0.8 ng/mL, particularly in women, older adults, and those with compromised renal function (Rathore et al., 2003).

Implications for Home Healthcare Clinicians

Patients with HF are commonly hospitalized due to episodes of acute decompensation. Among Medicare patients hospitalized for HF between 2008 and 2010, 67% experienced readmission at some time during their disorder (Dharmarajan et al., 2015). Preventing hospital readmission for HF patients is a high priority for clinicians and insurers. Predicting acute decompensation of HF is significant as it can save lives and dollars. Home healthcare clinicians play a major role in the prevention of HF decompensation and exacerbation among their patients by closely monitoring signs and symptoms of HF.

A multidisciplinary healthcare team is needed as patients often have comorbidities such as diabetes, arthritis, osteoporosis, or chronic lung disease. Depression and anxiety are very common in patients with HF, which can lead to social isolation, apathy, and diminished cognition. Patients are often homebound and lack social support (van der Wal et al., 2017). Closely monitor the patient's condition, assist in coordinating multidisciplinary care, evaluate the home for safety and the mobility needs of the patient, assist patients with lifestyle changes, and provide emotional support. Astute assessment at every visit can prevent patient relapse and rehospitalization. Close contact with the patient's primary provider is essential. Involvement of the family in the care of the patient is also vital (Rogers & Bush, 2015a).

Provide educational materials, assess the patient's knowledge through teach-back, and reiterate the goals at every visit. Each medication's usage and purpose should be explained and kept in a timed, pillbox. Alternatively, a smartphone can be set to alert the patient to take medications. The necessary laboratory testing schedule should be recorded on a calendar for the patient. Transportation issues should be discussed and arranged. The patient should keep a list of all their medications and bring it to every healthcare appointment. Those who live alone may need an emergency alert system (Waters & Giblin, 2019).

Table 1. Common Symptoms in Heart Failure and Causative Mechanisms

Symptoms of HF	Causative Mechanism
Dyspnea	Pulmonary congestion
Orthopnea	Pulmonary congestion; extravasation of fluid throughout lung fields in supine position
Paroxysmal nocturnal dyspnea	Same as above
Fatigue	Skeletal muscle hypoperfusion
Ankle swelling	Fluid retention
Anorexia; early satiety	Gastrointestinal congestion
Depression, confusion	Cerebral hypoperfusion
Cachexia	Chronic activation of inflammatory pathways and cytokines such as TNF-alpha

Note. HF = heart failure; TNF = tumor necrosis factor. Adapted from Dumitru & Sharma (2018).

Patients should weigh themselves daily to detect water retention. A gain or loss of 2 lb from 1 day to the next is usually due to water weight and should be reported to the primary care provider. A low-sodium (<1,500 mg) diet needs to be fully explained. Excessive sodium in the diet is a common cause of fluid congestion in HF (Waters & Giblin, 2019). Foods with high sodium content need to be clearly listed. Patients should understand how to read nutrition labels in order to choose low-sodium foods, and they should understand how to choose low-sodium meal options on a restaurant menu. If permitted, look in the refrigerator and cabinets to get an idea of the patient's diet; nonrecommended foods should be pointed out. Use teach-back to verify the patient's understanding of recommended food choices. Weight control is also important as obesity increases the work of the heart.

Why Are Patients Commonly Rehospitalized?

Many patients with HF who are rehospitalized cite escalating shortness of breath, physical activity intolerance, and increasing fatigue as reasons for seeking healthcare (Gheorghiade et al., 2013). Commonly, patients who are readmitted for HF demonstrate signs of fluid overload such as weight gain, pulmonary crackles, jugular venous distension, and increased peripheral edema. Therefore, physical assessment to rule out fluid congestion is extremely important in patients with HF. There are a number of history inquiries and physical assessments that should be made at every home healthcare visit (Table 3).

Telehealth

Some healthcare organizations using remote monitoring systems referred to as telemedicine. Telemedicine uses technology that can assess relevant biologic parameters to evaluate the patient's risk of decompensation in HF. Remote monitoring of the patient's vital signs, weight, cardiogram, and physical activity can be done through body sensors in some telemedicine systems. The goal of remote monitoring systems is to gather data that can be used to optimize treatment and prevent decompensation. At this time, these types of remote support systems are not available in many parts of the country.

Palliative Care

Palliative care is an approach that focuses on alleviation of suffering and improving quality of life for those living with serious illness, regardless of prognosis. During the course of HF, patients can experience debilitating physical and emotional symptoms that severely degrade quality of life. Physical symptoms in advanced HF, such as dyspnea and pain are highly distressing for patients and caregivers (Goebel et al., 2009). Patients and their caregivers often face decisions about high-risk and complex treatments (e.g., cardiac devices, transplanta-

Table 2. Common Signs of Heart Failure and Causative Mechanisms

Signs of HF	Causative Mechanism
Jugular vein distension	Increased venous congestion/right atrial pressure
Ascites	Gastrointestinal venous congestion
Hepatomegaly	Liver venous congestion
Hepatojugular reflux	Increased venous congestion/right atrial pressure
Splenomegaly	Spleen venous congestion
Displaced PMI	Left ventricular enlargement
S3 gallop	Increased left ventricular filling volume

Note. HF = heart failure; PMI = point of maximum impulse. Adapted from Dumitru & Sharma (2018).

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Commonly, patients who are readmitted for heart failure demonstrate signs of fluid overload such as weight gain, pulmonary crackles, jugular venous distension, and increased peripheral edema.

tion) without adequate education about prognosis, decision support, or advance care planning. Currently, a palliative care approach for HF is not widely used; however, multiple clinical trials are currently underway examining various forms of palliative care delivery for HF (Kavalieratos et al., 2017). ☗

Table 3. History Questions and Physical Assessments in Heart Failure Patients

Home healthcare clinicians should assess the following at every visit:
• Patient appetite and diet diary
• Patient adherence to medications
• Mental and emotional status
• Ambulation (is patient steady on feet, can they rise from chair?)
• Vital signs; body temperature, pulse, respirations, blood pressure, oxygen saturation
• Heart rate and rhythm, extra heart sounds
• Jugular vein distension (noticeable above the clavicle)
• Respiratory rate, effort, adventitious lung sounds (crackles/wheezes)
• Dyspnea, orthopnea or paroxysmal dyspnea
• Abdominal distension (ask about bloating, early satiety, clothes tighter than usual)
• Weight change (greater than 2-lb weight gain overnight)
• Physical activity tolerance (how far they can walk without dyspnea; can they climb a flight of stairs?)
• Peripheral edema (palpate ankles, are shoes tight? are rings tight on fingers?)
• Ask patient about urine output
• Ask patient about side effects of medication

Note. Adapted from Bowers (2013), Riley (2015), Rogers & Bush (2015b), and Waters & Giblin (2019).

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The authors declare no conflicts of interest.

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