Abstract: Heart failure (HF) is a chronic syndrome that requires patients to manage signs and symptoms and adhere to a complex medication regimen. This article discusses updates in HF care related to a universal definition and new therapies, focusing on the four pillars of therapy for HF with reduced ejection fraction.

Keywords: GDMT, heart failure, HFrEF, nursing care, NYHA class

Case study
JW is a 72-year-old male with a health history that includes diabetes mellitus (DM) and hypertension (HTN). He began experiencing dyspnea after walking his dog one block, which he could previously do without difficulty. He has been sleeping in his recliner for 2 weeks because it was difficult for him to breathe lying flat in bed. He scheduled an appointment with his cardiologist.

JW’s cardiologist ordered a transthoracic echocardiogram (TTE), ECG, and lab work, including complete blood cell (CBC) count, comprehensive metabolic panel, thyroid panel, and N-terminal prohormone B-type natriuretic peptide (NT-proBNP) level. He had a left ventricular ejection fraction (LVEF) of 20% (low), 10 lb weight gain, and an elevated NT-proBNP. He was diagnosed with heart failure (HF) with a reduced ejection fraction.
Introduction
An aging population and people living longer with HF lead to the probability that nearly all nurses will encounter a patient with HF. Approximately 6 million adults older than 20 in the US have HF. The prevalence of HF is projected to increase by 46% by 2030, affecting over 8 million adults, or 3% of the US population. The projected increase is due to new HF definitions and classifications, improved therapies, and downstream effects of COVID-19 on cardiovascular disease (CVD).

Generally, HF is defined as a complex clinical syndrome resulting from structural or functional impairment of ventricular filling or ejection of blood. For decades, patients with HF have been classified using the NYHA classification system. The NYHA class is based on the patient’s description of their physical functioning. Although the NYHA class is still used extensively in clinical and research settings, emphasis has recently been placed on preventing HF.

A staging system based on symptoms and structural heart disease is used to identify a patient’s risk of developing HF (see Stages of HF and NYHA functional classification). Note that the LVEF plays a key role in defining the patient’s type of HF. An LVEF less than or equal to 40% is labeled HFrEF, while HF with an LVEF of at least 50% is (HFrEF) Stage C (New York Heart Association [NYHA] Class II).

The nurse educated JW on restricting dietary sodium to reduce fluid retention, staying physically active, and enrolling in cardiac rehabilitation, as well as the trajectory of HF, and the possibility of other therapies, such as an implantable cardioverter defibrillator (ICD). He was educated on guideline-directed medical therapy (GDMT) and the importance of self-care.

JW returned 2 weeks later for reassessment of his vital signs, serum electrolytes, kidney function, and signs and symptoms. He had frequent routine appointments afterward. The nurse used those opportunities to reinforce the prior education and assess the need for reeducation. JW’s GDMT was optimized, and a repeat TTE showed his LVEF improved to 55%. His signs and symptoms resolved, and he was continued on GDMT. He continued to follow closely with his multidisciplinary HF team as well as his primary care provider to manage his other comorbidities.
The percentage of blood pumped out of the ventricles with each contraction. HFrEF is the best-understood type of HF. In 2020, the American College of Cardiology (ACC), American Heart Association (AHA), and Heart Failure Society of America (HFSA) updated previous guidelines for the treatment of HF, incorporating new therapies.2

The new joint guidelines highlight several important changes in the care and treatment of patients with HF. COVID-19 had an influence on the incidence and prevalence of HF. The risk of developing HF after a SARS-CoV-2 infection increased by 72% overall, and the risk was higher in those who were hospitalized for COVID-19.4 There was a longer length of stay, increased need for acute care, and greater need for mechanical ventilation for patients with HF and COVID-19. Additionally, the incidence of death is more than doubled in patients with HF who contract SARS-CoV-2.5 The incidence of HF and CVD increased significantly in the year following a SARS-CoV-2 infection.4

This article provides nurses with an update on the approach and treatment of patients with HF, specifically those with HFrEF, where new therapies have emerged.

Pathogenesis

The heart's capacity to pump an adequate volume of blood to meet the oxygen and metabolic demands of the body at rest and during activity depends upon the ventricles' ability to relax and fill. During diastole, the ventricles fill and stretch as the blood volume increases. During systole, the stretched ventricles contract, ejecting approximately 70 mL of blood comprising what is known as stroke volume (SV). The proportion or fraction of blood ejected, relative to the amount of blood present in the LV before contraction, is considered the LVEF (see Ejection fraction and cardiac output).6-8 EF is central to understanding HF. Clinical research and therapies for HF are classified by EF.6-8 Related to EF, cardiac output (CO) is the SV ejected with each heartbeat over 1 minute. Recall that the autonomic nervous system regulates heart rate (HR), with sympathetic activation increasing HR and parasympathetic activation decreasing HR. SV is a

Ejection fraction and cardiac output

Ejection fraction (EF) is the volume of blood pumped out with each heart contraction, expressed as a percentage. Most often, EF refers to the blood pumped out of the LV (LVEF). Normal EF for the LV is between 50% and 70%. For example, an EF of 50% means that half of the total blood in the LV is pumped out with each heartbeat. The most common test for measuring EF is echocardiography.

Cardiac output (CO) is the volume of blood pumped out by the heart in a minute, expressed in L/min. Generally, normal CO at rest is 5-6 L/min. CO reflects the body's metabolic and oxygen needs. During increased need, CO increases to maintain adequate tissue perfusion. CO is the product of heart rate (HR), in beats per minute, times stroke volume (SV): CO = HR x SV.

Abbreviations in this article

ACC - American College of Cardiology
ACEI - angiotensin-converting enzyme inhibitor
AF - atrial fibrillation
AHA - American Heart Association
ARB - angiotensin receptor blocker
ARNi - angiotensin receptor/neprilysin inhibitor
BB - beta-blocker
BNP - B-type natriuretic peptide
cGMP - cyclic guanosine monophosphate
CO - cardiac output
CRT - cardiac resynchronization therapy
CVD - cardiovascular disease
DM - diabetes mellitus
EF - ejection fraction
ECG - electrocardiogram
GDMT - guideline-directed medical therapy
HF - heart failure
HFrEF - heart failure with reduced LVEF
HFmrEF - heart failure with mildly reduced or midrange ejection fraction
HFpEF - heart failure with preserved ejection fraction
HR - heart rate
HTN - hypertension
ICD - implantable cardioverter defibrillator
I, - pacemaker current
LVEF - left ventricular ejection fraction
MRA - mineralocorticoid receptor antagonist
NP - natriuretic peptides
NT-pro BNP - N-terminal pro B-type natriuretic peptide
NYHA - New York Heart Association
PUFA - polyunsaturated fatty acid
RAAS - renin-angiotensin-aldosterone system
RASI - renin-angiotensin system inhibition
RVD - right ventricular dysfunction
sGC - soluble guanylate cyclase
SGLT2i - sodium-glucose cotransporter 2 inhibitor
SNS - sympathetic nervous system
SV - stroke volume
TTE - transthoracic echocardiogram

The incidence and prevalence of HF increased significantly with mildly reduced or midrange EF (HFmrEF). Patients with an LVEF between these two parameters are now classified as HFmrEF (HF with mildly reduced or midrange EF).2 A new definition of HF has been proposed, which considers a patient's ejection fraction (EF) or the percentage of blood pumped out of the ventricles with each contraction.3

HFpEF is the best-understood type of HF. In 2020, the American College of Cardiology (ACC), American Heart Association (AHA), and Heart Failure Society of America (HFSA) updated previous guidelines for the treatment of HF, incorporating new therapies.2

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EF is central to understanding HF. Clinical research and therapies for HF are classified by EF.6-8 Related to EF, cardiac output (CO) is the SV ejected with each heartbeat over 1 minute. Recall that the autonomic nervous system regulates heart rate (HR), with sympathetic activation increasing HR and parasympathetic activation decreasing HR. SV is a
function of preload, afterload, and contractility. The interplay among these factors maintains an adequate CO; low CO is characteristic of HF, often resulting from myocardial injury. Thus, HF is associated with derangements in these factors.

Preload, determined by the venous return to the heart, is the volume of blood in the ventricle just before contraction. Afterload reflects the force the heart must generate to eject blood from the filled ventricles. Peripheral vascular resistance and ventricular wall tension influence afterload. When peripheral vascular resistance is high, as with arterial HTN, greater pressure must be generated to eject blood out of the LV through the aortic valve and into the systemic circulation. In response to the increased pressure, the heart enlarges and changes shape.

Contractility or inotropy reflects cardiac performance. Contractility is related to calcium handling within the myocyte. All these factors are important as the CO falls and the heart tries to compensate to preserve adequate blood flow.8

Compensatory mechanisms

CO is maintained through compensatory mechanisms, including the Frank-Starling mechanism; neurohormonal activation through the sympathethic nervous system (SNS) and the renin-angiotensin-aldosterone system (RAAS); natriuretic peptides; and vasactive substances (nitric oxide, endothelin, vasopressin). Initial compensatory changes in response to the decrease in CO may maintain heart function, and the insult may go unnoticed. However, as the heart slowly adapts, the heart remodels, changing shape and enlarging (called hypertrophy) even at the cellular level (see Compensatory mechanisms in heart failure). Although these mechanisms may successfully initially preserve CO, the heart will eventually fail, resulting in HF signs and symptoms.

Frank-Starling mechanism
The Frank-Starling mechanism explains that increased preload results in increased CO. The greater the influx of blood into the ventricle, the more forceful the contraction until muscle fibers are over-stretched or stiffened and the heart fails. Depending upon the clinical scenario, a patient may be given a positive inotrope, such as oral digoxin and I.V. milrinone or dobutamine, to increase the force of contractility. Although these drugs may help with preload and contractility, they are not without risk and require monitoring.

Sympathetic nervous system
The SNS is an integral component of the neuroendocrine compensatory response in HF. SNS activation is triggered by pressure-sensitive baroreceptors when CO falls. Myocardial oxygen demand increases, and sustained SNS activation and renal hypoperfusion actuate other neuroendocrine responses through the RAAS, causing vasoconstriction and tachycardia.

Renin-angiotensin-aldosterone system (RAAS)
The RAAS and the SNS augment SV and HR to maintain CO. Due to chronic neuroendocrine activation of these systems, hypertrophy, fibrosis, and myocardial necrosis occur. Changes within the myocytes (which lead to remodeling), impairment of the heart’s pumping ability, and aberrations in the heart’s electrical activity occur.

Natriuretic peptides
The heart produces and secretes natriuretic peptides (NPs)—peptide hormones with potent diuretic, natriuretic, and neuroendocrine actions. Of the NPs, B-type (BNP) has the most clinical significance in HF. In patients with HF, especially

Several compensatory neuroendocrine mechanisms maintain cardiac output, but these mechanisms contribute to heart failure over time. From Porth Figure 20-2.
**Risk factors and common contributors for developing HFrEF**

- Hypertension
- Diabetes
- Coronary artery disease
- Exposure to cardiotoxic agents or substance abuse (chemotherapy, alcohol)
- Genetic/inherited cardiomyopathies
- Tobacco abuse
- Rheumatologic or autoimmune diseases
- Thyroid disease
- Cardiac dysrhythmias, such as atrial fibrillation
- Infiltrative cardiac diseases
- Myocarditis (viral)
- Pregnancy
- Stress
- Valvular heart disease

HFrEF plasma levels of BNP are elevated. NT-proBNP and BNP assays correlate with ventricular dysfunction and are used clinically to diagnose HF and predict disease severity.2,7,9

**Risk factors**

Many cardiac and noncardiac conditions, as well as aging, increase the risk of developing HF (see Risk factors and common contributors for developing HFrEF). An elevated systolic or diastolic BP is one of the most important risk factors.

**Clinical manifestations of HF**

Ideally, both the left and right ventricles should contract in tandem to produce and maintain CO. The clinical manifestations of HF depend on whether the left or right side of the heart is compromised; most often the left side of the heart fails, followed by the right side. Biventricular failure or dysfunction typically occurs as HF progresses. A hallmark sign of HF is fluid retention.

**Signs**

Fluid retention or congestion can manifest in several ways. When left ventricular dysfunction (LVD) is present, the heart’s ability to pump oxygenated blood to the body is impaired, leading to pulmonary congestion.10 In contrast, the failure of the right side of the heart leads to abdominal and lower extremity edema.10 Worsening congestion and right ventricular dysfunction (RVD) can be visualized by jugular vein distension when sitting or standing.8 Fluid retention, in combination with a decreased CO, diminishes oxygenation to skeletal muscles, causing the patient to experience symptoms such as activity intolerance and fatigue.

**Symptoms**

The cardinal symptoms of LVD are dyspnea and fatigue.1 1 Difficulty breathing or dyspnea on exertion, the inability to lie flat (orthopnea), sudden onset of shortness of breath that causes awakening from sleep (paroxysmal nocturnal dyspnea), and palpitations due to tachycardia are common symptoms due to fluid retention and poor CO.10 Diminished oxygen supply to the brain leads to cognitive impairment, while poor perfusion to muscles results in activity intolerance and fatigue.8 In contrast, RVD affects the ability of deoxygenated blood to travel outward into the lungs and beyond. Ascites, causing a sense of abdominal fullness, and peripheral edema, causing a sense of heaviness in the lower extremities, are related to the continued backward flow of blood due to right-sided dysfunction.12

**Dysrhythmias**

Atrial fibrillation (AF) is common in HF; almost half of the patients with NYHA Class IV HF have AF13 Disruption of the conduction tissue within the left atrium from LVD commonly results in atrial and ventricular tachydysrhythmias, such as uncontrolled AF and ventricular tachycardia.8 Ventricular tachycardia, caused by ventricular dysfunction, is a well-known source of sudden cardiac death in patient with HF.8

**Diagnostic studies**

The initial evaluation of a patient with suspected new, or “de novo,” HF is a TTE to confirm the diagnosis, additional testing for coronary artery disease (if ischemia is suspected), and revascularization if indicated.3 Reperfusion of the coronary arteries may help improve patient outcomes.2,7 Diagnostic tests will be needed throughout the course of HF to assess disease progression and the effectiveness of therapy.

A TTE, an ultrasound of the heart, provides information on the diagnosis, EF, cardiac structures, and HF progression, which are necessary to classify and guide treatment for patients and to rule out other diagnoses. TTEs are recommended throughout the course of HF and when the patient’s clinical status worsens or changes.

A chest X-ray is used to monitor the patient’s initial volume status and reassess changes in the patient’s symptoms reflective of volume status, such as dyspnea. For example, this imaging study may reveal cardiomegaly and causes of dyspnea, such as pulmonary edema. An ECG is part of the routine evaluation of a patient with HF and provides information on cardiac rate, rhythm, and causes contributing to a low EF and poor outcomes, such as dysrhythmias and myocardial ischemia or infarction. Patients with HF are at high risk of developing AF or experiencing ventricular dysrhythmias resulting in sudden cardiac death.3

Lab testing identifies possible causes and other comorbidities in patients with HF; such as diabetes, renal disease, thyroid disease, and anemia. A standard diagnostic evaluation of a patient with HF includes...
Management of HFrEF

Management of HFrEF is complex and requires coordination and care by a multidisciplinary team. The mainstay of treatment for Stage C HFrEF is GDMT. In addition to pharmacotherapy, there are nonpharmacologic treatment considerations.

Pharmacotherapy: Four pillars

The core pharmacologic treatment for Stage C HFrEF includes RAAS inhibition with an ARNi, angiotensin-converting enzyme inhibitor (ACEi), or angiotensin receptor blocker (ARB); BB; MRA; and SGLT2i. Additional therapies include hydralazine and isosorbide dinitrate (vasodilator), ivabradine (I, inhibitor or hyperpolarization-activated cyclic nucleotide-gated channel blocker), digoxin (cardiac glycoside), and soluble guanylate cyclase stimulators. Guideline-directed medication dosing titration is recommended, as tolerated, to achieve target doses. Using ARNI/ACEi/ARB, BB, MRA, and SGLT2i together reduces the likelihood of death. The most recent guideline has delineated GDMT to include all four classes: RAAS inhibition with an ARNi, ACEi, or ARB; BB; MRA; and SGLT2i.

Medications that inhibit RAAS reduce death. First-line therapy for patients with HFrEF is an ARNi for RAAS inhibition. An ACEi is recommended when an ARNi is not feasible, and an ARB when using an ARNi and ACEi is not feasible. For example, an ARB may be used if a patient develops a cough or angioedema. When switching between an ACEi and an ARNi, a 36-hour washout period is recommended between doses to minimize the risk of angioedema. Patients can switch among the various RAAS inhibitors (RAASi), that is, an ARNi, ACEi, or ARB in certain instances, if the patient develops an adverse reaction as mentioned above.

In patients with HFrEF, at the time of diagnosis, a BB, such as carvedilol, metoprolol succinate (extended release), and bisoprolol, is recommended to reduce death and hospitalizations unless contraindicated. BBs should be initiated at low doses and uptitrated to achieve the target doses. Even if LVEF improves, BBs should be continued to reduce the risk of LV dysfunction and thus reduce the risk of major cardiovascular events.

In patients with HFrEF and NYHA class II-IV symptoms, an MRA including spironolactone or eplerenone is recommended. The patient’s kidney function and potassium level should be monitored routinely. Adverse reactions such as gynecomastia and vaginal bleeding have been observed in patients who take spironolactone. Consider temporarily stopping MRA therapy if a patient develops diarrhea or dehydration due to the risk of worsening kidney function or abnormal potassium levels.

The 2022 guideline for the management of HF added an SGLT2i for patients with symptomatic HFrEF, irrespective of the presence of type 2 DM. Novel mechanisms of an SGLT2i that may positively impact patients with HFrEF include volume regulation, metabolic effects, cardiorenal mechanisms, direct effects on cardiac contractility and ion-homeostasis, improved cardiac remodeling, inflammation reduction, and decreased oxidative stress. Serious renal adverse outcomes were infrequent, and a slower rate of decline in estimated glomerular filtration rate was observed in patients taking an SGLT2i. Both SGLT2is, dapagliflozin and empagliflozin, are initiated at their target dose of 10 mg once daily. Monitoring parameters include blood glucose levels, kidney function (at the time of initiation), and ongoing signs and symptoms of genitourinary infections, necrotizing fasciitis, hypersensitivity reactions, volume depletion, and hypotension. The SGLT2i medications are the most recent addition to GDMT and can be initiated in the hospital or outpatient setting.

Starting quadruple therapy (the four pillars) with the above medications at low doses while routinely monitoring vital signs, kidney function, serum potassium levels, and volume status is recommended once the patient is hemodynamically stable. This life-saving medication regimen should be initiated as soon as safely possible.

Other pharmacologic therapies

Diuretics are recommended for the management of fluid retention and congestive symptoms in patients with HFrEF. Loop diuretics are preferred; additional diuretics, such as metolazone or chlorothiazide, may be added to the loop diuretic regimen for patients with refractory edema. Maintenance diuretic therapy should be considered to prevent recurrent congestion. It is important to monitor kidney function and serum electrolytes. Diuretics should only be used in combination with GDMT.

Managing volume overload with diuretics is very common. However, additional drugs for special populations include hydralazine and isosorbide dinitrate, ivabradine, soluble guanylate cyclase (sGC) stimulators, cardiac glycosides, inotropes, omega-3 polyunsaturated fatty acids (PUFA), potassium binders, and intravenous iron replacement.

The combination of hydralazine and isosorbide dinitrate is considered...
Patients should be taught to monitor for Black patients with HFrEF and with NYHA class III-IV symptoms because it has been shown to reduce morbidity and mortality and improve symptoms. This combination may also be used in patients who cannot tolerate RAAS inhibition.

Ivabradine has specific restrictions related to HR. The drug can be considered for patients with NYHA class II-III symptoms, an LVEF less than or equal to 35%, who are on GDMT including a BB at maximum tolerated dose, and who are in sinus rhythm with an HR greater than or equal to 70 beats/minute at rest. Although adding ivabradine to standard GDMT for patients with HFrEF is associated with improved cardiac function, reduced HF readmission, greater HR reduction, and better exercise capacity, it has not been shown to reduce mortality.

sGC stimulators, such as vericiguat, have a unique mechanism of action compared with other therapies for HFrEF. They enhance the sensitivity of sGC to nitric oxide and thereby increase cyclic guanosine monophosphate (cGMP) production, inducing smooth muscle relaxation and vasodilation. Vericiguat may be considered for use in patients with HFrEF who have persistent symptoms despite optimization of GDMT. sGC stimulators should be avoided in patients taking long-acting nitrates or phosphodiesterase inhibitors due to potentiated vasodilation. Patients should be taught to monitor for hypotension, the most common adverse reaction. The potential niche role for sGC stimulators in the HFrEF patient population is still evolving.

Another optional therapy is digoxin, a cardiac glycoside or positive inotrope. The only large digoxin trial in patients with HFrEF who predated the current GDMT recommendations. The benefit of digoxin in patients with HFrEF is unclear because some studies have shown lack of mortality benefit or increased mortality.

### Drug classes for the treatment of HFrEF

| Drug Class/Target Dose | Mechanism of Action |
|------------------------|---------------------|
| **Renin-Angiotensin System Inhibitors** | |
| Angiotensin Receptor | Sacubitril: |
| Neprilsin inhibitor (ARNI) | • Inhibits neprilsin (breaks down NPs), leading to increased levels of natriuretic peptides |
| Sacubitril-Valvartan | • Induces vasodilation and natriuresis |
| 97 mg Sacubitril | Valsartan: |
| 103 mg Valvartan twice daily | • See ARB MOA below |
| **Angiotensin-Converting Enzyme inhibitor (ACEi)** | |
| Captopril | 50 mg 3 times daily |
| Enalapril | 10-20 mg twice daily |
| Lisinopril | 20-40 mg daily |
| **Angiotensin Receptor Blocker (ARB)** | |
| Valsartan | Blocks the vasoconstricting and aldosterone-secreting effects of angiotensin II |
| 160 mg twice daily | • Decreases the response to bradykinin |
| Losartan | • Decreases non-renin-angiotensin effects (cough) |
| 50-150 mg daily | |
| **Beta Blocker (BB)** | |
| Bisoprolol | • Inhibits beta-adrenergic receptor activation |
| 10 mg daily | • Decreases heart rate |
| Carvedilol | • Decreases pulmonary pressure |
| 25-50 mg twice daily | • Decreases systemic vascular resistance |
| Carvedilol CR | • Increases stroke volume |
| 80 mg daily | |
| Metoprolol succinate | 200 mg daily |
| **Mineralocorticoid Receptor Antagonist (MRA)** | |
| Spironolactone | • Competes with aldosterone for receptor sites in the distal tubules |
| 25-50 mg daily | • Increases sodium chloride and water excretion while conserving potassium and hydrogen ions |
| Eplerenone | 50 mg daily |
| **Sodium-glucose Cotransporter 2 Inhibitor (SGLT2i)** | |
| Dapagliflozin | • Inhibits SGLT2 in the proximal renal tubules |
| 10 mg daily | • Reduces sodium absorption |
| Empagliflozin | • Increases sodium delivery to the distal tubule |
| 10 mg daily | • Decreases cardiac preload/aftload |
| **Other therapies** | |
| Direct-acting Vasodilator | Isosorbide Dinitrate: |
| Fixed dose combination of 40 mg isosorbide dinitrate and 75 mg of hydralazine 3 times daily | • Relaxes smooth muscle of arterial and venous vasculature |
| 120 mg of isosorbide dinitrate total daily in divided doses and 300 mg of hydralazine total daily in divided doses | • Reduces cardiac oxygen demand by decreasing preload |
| Hydralazine: | Hydralazine: |
| • Vasodilates arterioles | • In decreasing systemic resistance |
| (Continues) | |

(Continues)
potassium binders may increase risk of cardiovascular complications. Potassium binders make hyperkalemia common in patients with HFrEF; an elevated serum potassium level is associated with a poorer prognosis and increased risk of cardiovascular complications. Potassium binders may be considered for these patients; however, the benefit of using a potassium binder to facilitate the continuation of RAASI is uncertain. Whether potassium binders improve clinical outcomes for these patients is under investigation.

Anemia is independently associated with mortality and disease severity in HF and is associated with poorer quality of life, prognosis, and exercise capacity in patients with HFrEF. For patients who have iron deficiency and HFrEF, with or without anemia, IV iron replacement may be considered to improve quality of life and functional status. Oral iron is inadequate to treat iron-deficiency anemia in patients with HFrEF due to poor absorption and slow iron repletion in these patients (see Drug classes for the treatment of HFrEF).

Nonpharmacologic treatment

Dietary sodium restriction has been a common nonpharmacologic treatment recommendation for patients with HFrEF and congestive symptoms. However, conflicting evidence and concerns about the quality and representativeness of data have led experts to question this recommendation. The AHA recommends a daily sodium intake of less than 2,300 mg for general health promotion and is moving toward a limit of no more than 1,500 mg per day for most adults. However, this is not specific to patients with HFrEF as the current guidelines recommend less than 3,000 mg per day. The Dietary Approaches to Stop Hypertension (DASH) diet can achieve sodium restriction without affecting nutritional quality and may reduce hospitalizations for HF.

Exercise has several benefits for patients with HFrEF such as improving functional status and quality of life. Exercise training in patients with HFrEF is safe and does not increase mortality. All patients should be encouraged to stay physically active once they are hemodynamically stable and on GDMT. Cardiac rehabilitation improves exercise capacity and quality of life in patients with HFrEF, but there was no association between cardiac rehabilitation and mortality or hospitalization. For patients with HFrEF who were hospitalized for worsening HF, an early, transitional, tailored rehabilitation program resulted in improved physical function.

A cardiac rehabilitation program for patients with HFrEF typically includes a medical evaluation, education, dietary recommendations, psychosocial support, and an exercise training program. A prescribed exercise program is recommended for patients with HFrEF in conjunction with evidence-based therapy, such as GDMT. Device-based therapies for HFrEF include cardiac implantable devices and cardiac resynchronization therapy (CRT). For patients who meet certain criteria, ICD therapy is recommended to reduce mortality from sudden cardiac death due to ventricular dysrhythmias. For some patient populations, CRT is recommended to help the ventricles contract more efficiently. Synchronizing ventricular contraction may reduce mortality and hospitalizations and improve quality of life and symptoms. An important point is to optimize GDMT before any device implantation.

Nursing considerations

HF management and coordination of services are complex. Ideally, early identification of those at risk may help limit the number of new cases. Care by multidisciplinary teams, including

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**Drug classes for the treatment of HFrEF**

| Drug Class/Target Dose | Mechanism of Action |
|------------------------|----------------------|
| I inhibitor (pacemaker current inhibitor) | Inhibits ion channels within the sinoatrial node, Disrupts the I, ion current flow, Increases diastolic filling time, Slows SA node firing, Reduces heart rate |
| Soluble Guanylate Cyclase Stimulator (sGC stimulator) | Enhances sensitivity of sGC, the primary receptor for nitric oxide |
| Cardiac Glycoside (positive inotrope) | Inhibits sodium/potassium ATPase pump in myocardial cells, Increases intracellular sodium, Increases cardiac contractility by promoting calcium influx |

**Abbreviations:** ATP, adenosine triphosphatase; HFrEF, heart failure with a reduced ejection fraction; MOA, mechanism of action; mg, milligrams; ng/mL, nanograms per milliliter; SA, sinoatrial
nurses, may help improve patients’ outcomes and quality of life. Multidisciplinary HF teams provide integrated, comprehensive care for the management of HF and associated comorbidities. Nurses play an important role in educating patients during their hospital admission by helping them navigate symptom recognition and the management processes. Nurses must educate patients continually throughout their hospitalization on self-management and self-care, strategies to prevent readmission, and other options such as palliative care (see Patient, family, and caregiver education topics). Discharge teaching is important, but nurses should take the opportunity to educate patients and caregivers at every healthcare encounter, helping to reinforce knowledge and understanding.

**Patient, family, and caregiver education topics**

**General Education**
- Definition of HF
- Cause/probable cause of HF
- Reason for signs and symptoms
- Expected signs and symptoms
- Signs and symptoms of worsening HF and what to do if they occur
- Self-monitoring, including daily weights
- Treatment care and plan
- Patient and caregiver responsibilities
- Role(s) of family and caregivers in treatment care and plan
- Qualified local support group and online resource availability and importance
- Importance of tobacco use cessation
- Value of preventive health behaviors, including influenza and pneumococcal vaccinations

**Prognosis**
- Life expectancy
- Educate family/caregiver about what to do in the event of sudden death
- Advance directives

**Recommended Activities**
- Exercise
- Work, leisure, and recreational activity
- Sexual activity and difficulties and how to cope

**Recommended Diet**
- Calorie-appropriate diet
- Consistent and restricted sodium intake of less than 3,000 mg per day
- Correlation between sodium intake and HF signs or symptoms or weight gain
- Small, frequent meals
- Fluid intake moderation
- Alcohol moderation or restriction if HF is secondary to alcohol consumption

**Medication**
- Medication dosing
- Effects of medications on quality of life and survival
- Potential adverse reactions and what to do if they occur
- Coping with complicated medical regimens
- Accessibility of lower-cost medications or financial assistance
- Avoiding dangerous interactions with OTC medication, home remedies, and herbal supplements

**Conclusion**

The case study involving JW highlights the importance of a team effort to manage HF and the vital role nurses play in identifying signs and symptoms and educating patients. Heart failure, as delineated above, is a complex disease process; evidence-based care can improve outcomes.

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