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Heart Failure: Updated Treatment Guidelines

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Dr. Mona T. Thompson has no relevant financial relationships to disclose.

Goal. The goal of this lesson is to provide an overview of heart failure including signs, symptoms, types and classification, and a guideline-driven treatment algorithm for management from the American College of Cardiology Foundation/American Heart Association (ACCF/AHA) in the ambulatory care setting.

Objectives. At the completion of this activity, the participant will be able to:

1. demonstrate an understanding of the basic pathophysiology that leads to heart failure (HF);
2. recognize the classification systems utilized to describe HF patients and the pharmacological treatment recommended at each stage;
3. identify adverse events, safety concerns, and key counseling points associated with each drug class utilized for HF; and
4. demonstrate an understanding of interventions used to reduce HF-related hospitalizations and the role of the pharmacist.

Background

Heart failure (HF) is a complex and progressive clinical syndrome caused by inability of the heart to pump sufficient blood to meet the body's metabolic needs. HF can result from any disorder that reduces ventricular filling and/or myocardial contractility. Decreased contractility, also referred to as systolic dysfunction, may result from reduced muscle mass (e.g., myocardial infarction [MI]), dilated cardiomyopathies, and ventricular hypertrophy. Restricted ventricular filling, traditionally referred to as diastolic dysfunction, may occur secondary to increased ventricular stiffness, ventricular hypertrophy, myocardial ischemia, MI, mitral or tricuspid valve stenosis, and pericardial disease.

Many conditions or comorbidities are associated with an increased propensity for structural heart disease. These include hypertension, diabetes mellitus, metabolic syndrome, and atherosclerotic disease. Chronic alcoholism, cocaine abuse, some antineoplastics, anabolic steroids, as well as stimulants such as ephedra, amphetamine, and methylphenidate, are among agents that may lead to toxic cardiomyopathy. Hypertension is recognized in literature as the single most important modifiable risk factor for HF in the United States.

Primary symptoms of HF include dyspnea (particularly on exertion) and fatigue, which may limit exercise tolerance. Fluid overload can result in pulmonary congestion and peripheral edema. However, since not all patients will present with volume overload, the term "heart failure" is preferred over "congestive heart failure." Other non-specific symptoms may include nocturia, hemoptysis (coughing up blood), abdominal pain, anorexia, nausea, bloating, ascites, poor appetite, mental status changes, and weight gain.

There is no single diagnostic test for heart failure as it is a clinical diagnosis based on patient history and physical examination. Laboratory tests are useful in identifying disorders that may cause or worsen HF. Ventricular hypertrophy can be seen on a chest radiograph or electrocardiogram (ECG). Echocardiogram can identify abnormalities of the pericardium, myocardium, or heart valves, and measure left ventricular ejection fraction (LVEF) to determine if systolic or diastolic dysfunction is present.

The lifetime risk of developing HF is 20 percent for Americans ≥40 years of age, and is associated with an increased risk of incidence with age. Approximately 6.6 million persons in the U.S. have HF, with an estimated 670,000 new diagnoses each year. Along with the aging of baby-boomers and the improved survival of patients after myocardial infarction, it is anticipated that the prevalence of heart failure will further increase.

Epidemiological studies indicate that the incidence rate is highest among black men and lowest in white women. HF is the primary diagnosis in greater than one million hospitalizations annually. The total cost of HF care in the U.S. exceeds $30 billion annually, with over half of these costs from hospitalizations. While survival has improved, the absolute mortality rate for HF remains approximately 50 percent within five years of diagnosis. In addition to being

Table 1
Comparison of ACCF/AHA Stages of HF and NYHA functional classifications*

ACCF/AHA Stages of HF	NYHA Functional Classification
A High risk for HF but without structural heart disease or symptoms of HF	None
B Structural heart disease but without signs or symptoms of HF	I No limitations of physical activity. Ordinary physical activity does not cause symptoms of HF.
C Structural heart disease with prior or current symptoms of HF	I No limitations of physical activity. Ordinary physical activity does not cause symptoms of HF.
	II Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in symptoms of HF.
	III Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes symptoms of HF.
	IV Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest
D Refractory HF requiring specialized interventions	IV Unable to carry on any physical activity without symptoms of HF, or symptoms of HF at rest

*2013 ACCF/AHA Guideline for the Management of Heart Failure

a considerable economic burden, HF significantly decreases health-related quality of life.

Acute decompensated heart failure, which occurs in patients with new or worsening signs or symptoms of HF, is a significant source of morbidity and mortality in the U.S. Many of the hospital readmissions for HF are considered preventable, which has led providers and hospital systems to seek novel strategies to reduce hospitalization.

Defining and Classifying Heart Failure

Ejection fraction (EF) is an important measurement in determining how well the heart is pumping blood, and in diagnosing and tracking heart failure. EF is defined as the percentage of the total amount of blood in the left ventricle that is pushed out with each heartbeat. A normal heart's ejection fraction is between 55 and 70. Because patients with heart failure may still have a normal ejection fraction, the American Heart Association (AHA) has adopted more reflective terms to describe types of HF historically referred to as diastolic HF and systolic HF. Heart failure with preserved ejection fraction (HFpEF), also referred to as diastolic HF, is defined as maintaining an EF of ≥50. Alternatively, patients with an EF ≤40 are said to have heart failure with reduced ejection fraction (HFrEF) or systolic HF.

There are two different systems used to categorize and treat HF. In the New York Heart Association Functional Classification System (NYHA-FC), physicians utilize current symptoms and exercise capacity to classify patients with HF into functional classes. For example, functional class I (FC-I) patients have no limitation in physical activity, whereas patients in FC-IV are unable to engage in physical activity without discomfort.

The American College of Cardiology Foundation/American Heart Association (ACCF/AHA) system emphasizes the development and progression of disease. Patients are placed into ACCF/AHA stages based on risk factors for HF, presence of structural heart disease, and symptoms. Table 1 provides a comparison of these two classification systems and demonstrates how they complement one another according to the 2013 guidelines from ACCF/AHA.

Clinical Evaluation of the Heart Failure Patient

The 2013 ACCF/AHA guidelines call for clinicians treating patients presenting with HF findings to complete a thorough history and physical examination to identify cardiac and non-cardiac disorders or behaviors that may cause or accelerate the development or progression of HF. Reviewing medications that may exacerbate HF and assessing for adherence are important when an extensive history is conducted. The guidelines also provide comprehensive recommendations on using risk scoring to help guide therapeutic decision making, diagnostic testing, use of biomarkers, and cardiac imaging which will not be covered in this lesson. This information is used by clinicians to assign patients to HF stages and direct evidence-based treatment decisions.

Treatment Algorithm for Heart Failure

Treatment at each stage of HF will be discussed in general, with an emphasis on the goal of treatment and the drug classes recommended at each stage. First and foremost, the etiology or precipitating factors, if known, should be treated to minimize the development or progression of HF.

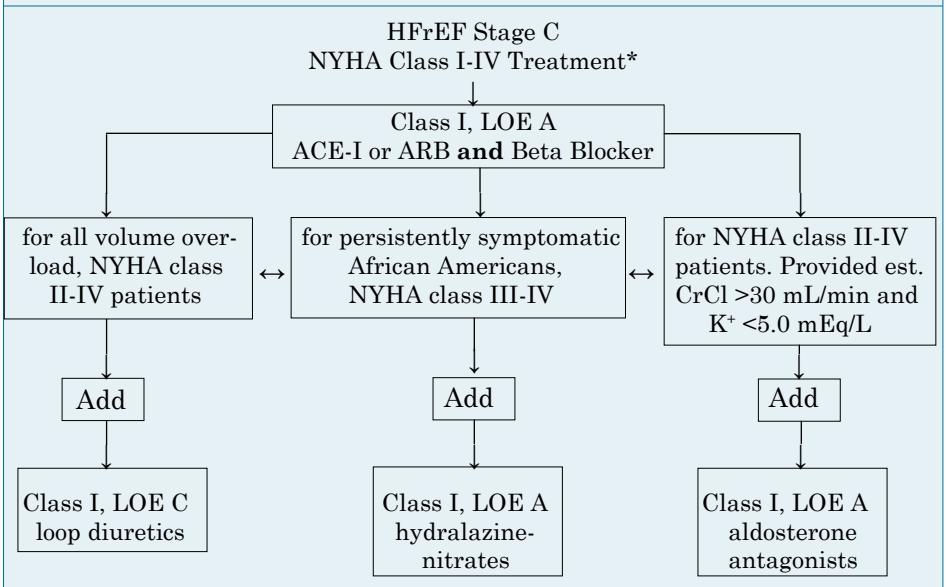
Patients classified as ACCF/

AHA **Stage A** are at high risk for developing heart failure. In Stage A, the emphasis is on treating or avoiding modifiable risk factors that may lead to or contribute to HF, such as obesity, diabetes mellitus, dyslipidemia, alcohol or tobacco use, and known cardiotoxins. Hypertension and lipid disorders should be treated in accordance with evidence-based treatment guidelines to lower risk of HF. Long-term treatment of both systolic and diastolic hypertension has been shown to reduce the incidence of HF by approximately 50 percent. In patients with type 2 diabetes mellitus, angiotensin-converting enzyme inhibitors (ACE-I) or angiotensin receptor blockers (ARBs) significantly reduce the incidence of HF. Patients with atrial fibrillation should be adequately treated for rate control, as uncontrolled heart rate is clearly associated with the development of HF.

Patients with structural heart disease, but no signs of HF or symptoms are said to have **Stage B** heart failure. The objective of treatment is to minimize additional injury, and to prevent or slow the cardiac remodeling (alteration in the dimensions, mass, or shape of the heart) process. In general, all recommendations for patients with Stage A HF also apply to those with Stage B HF. ACCF/AHA guidelines recommend that in all patients with a recent or remote history of MI or ACS (acute coronary syndrome) and reduced EF, ACE-I (or ARBs) and beta blockers should be used. Furthermore patients with reduced EF (HFrEF), even if they do not have a history of MI, should receive ACE-I and beta blockers. Treatment of hypertension and lipid control with statin therapy remains imperative to prevent symptomatic HF and cardiovascular events.

Nonpharmacologic interventions include recommendations to educate patients on heart failure self-care. This includes teaching patients how to monitor their symptoms and weight fluctuations, restrict sodium intake, take medications as prescribed and stay physically active. Exercise training in patients with HF can be safe and has numerous benefits. Meta-analyses show that cardiac rehabilitation reduces mortality; improves functional capacity, exercise duration, health-related quality of life; and reduces hospitalizations.

Table 2
Pharmacologic Therapy for Stage C HFrEF



*Adapted from *Circulation*, October 15, 2013. ACE-I = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; HFrEF = heart failure with reduced ejection fraction; hydralazine-nitrate = hydralazine and isosorbide dinitrate; LOE = Level of Evidence; NYHA = New York Heart Association.

Patients with **Stage C** heart failure have structural heart disease and previous or current HF symptoms. At Stage C, the treatment of HF becomes more complex. Table 2 summarizes the drug therapy recommendations for patients with Stage C systolic HF or HFrEF according to ACCF/AHA guidelines. In addition to the treatments outlined for Stage A and B (ACE-I or ARB + beta blocker), diuretics, digoxin, hydralazine/isosorbide dinitrate, or aldosterone antagonist agents may be indicated. Specific prescribing information about each of the drug classes will be discussed in more detail in the next section of this lesson.

In patients with preserved ejection fraction (HFpEF), controlling blood pressure and utilizing diuretics to relieve symptoms due to volume overload continue to be recommended. According to the ACCF/AHA guidelines, beta blockers, ACE-I, and ARBs are reasonable options to control hypertension in patients with HFpEF. In patients with atrial fibrillation, management according to guidelines may also improve symptomatic HF. At the time of writing this lesson, there are no evidence-based treatments to decrease readmission or mortality in patients with HFpEF.

Nonpharmacologic interventions such as cardiac rehabilitation and restriction of fluid intake and dietary sodium continue to be recommended as in previous stages.

Stage D HF describes patients with chronic HF who continue to progress and develop persistent severe symptoms regardless of medical treatment. Various terms for this stage include "refractory," "end-stage HF," or "advanced HF." These patients may be considered for specialized therapies that include mechanical circulatory support, procedures to facilitate fluid removal, continuous IV positive

inotropic therapy, cardiac transplantation, or hospice care when appropriate. Inotropic support may be recommended for patients with cardiogenic shock on a temporary basis, until definitive therapy is determined or resolution of the acute precipitating problem, in order to maintain systemic perfusion and preserve end-organ performance. Inotrope therapy may also serve as a bridge for patients awaiting a mechanical circulatory support device or cardiac transplantation. In some cases, inotropes may be used long-term as palliative therapy for symptom control in select patients. Intravenous inotropes used in the management of HF include dopamine, dobutamine, and milrinone.

Acute decompensated heart failure involves patients with new or worsening signs or symptoms (often resulting from volume overload and/or hypoperfusion). Additional medical care such as emergency department visits and hospitalizations may be required. Treatment may include intravenous diuretics, vasodilators, and inotropes in order to relieve congestive symptoms, restore optimal volume status, and improve cardiac output.

Drug Classes for Heart Failure

ACE Inhibitors/ARBs. Clinical trials have left no doubt that ACE-I improve symptoms, slow disease progression, and decrease mortality in patients with HF and reduced LVEF. Their primary mechanism of action, decreasing angiotensin II and aldosterone, reduces the damaging ramifications that lead to cardiac remodeling. One study reported that, at a three-year follow-up, patients treated with ACE-I demonstrated reduced hospitalization or death, and that the benefit extended up to a 12-year follow-up. Unless contraindications exist, ACE-I are used in most patients with Stage A, B, or C HF. Cough and angioedema are the most common causes of ACE-I intolerance.

ARBs work in a similar fashion to ACE-I, inhibiting negative consequences of angiotensin II by blocking the angiotensin II receptor. However, they do not affect bradykinin and are not associated with cough that can result from ACE-I use and intolerance. While there are seven ARBs currently marketed in the United States, candesartan and valsartan are preferred as they are FDA-approved for the treatment of HF. Literature and AHA guidelines support ARBs as reasonable alternatives to ACE-I for patients who cannot tolerate them or who have contraindications. However, caution should be exercised when ARBs are used in patients with a history of angioedema due to ACE-I use because cross sensitivity has been reported.

Initial doses of ACE-I or ARBs are low, then gradually titrated upward to goal doses used in clinical trials. If the target dose cannot be tolerated, then the maximum tolerable dose should be used. For both medication classes, blood pressure, renal function, and serum potassium levels should be monitored during the initiation phase and during dosage increases. Some Stage C patients may be candidates for combination ACE-I and ARB therapy (in addition to beta blocker therapy), but only when aldosterone receptor antagonists are not indicated or not tolerated.

Beta Blockers. Certain beta blockers have convincingly shown slowed disease progression, decreased hospitalizations, and reduced mortality in patients with systolic HF. Carvedilol, metoprolol XL, and bisoprolol are the only beta blockers shown to reduce mortality in large HF trials. Unless contraindicated, use of one of the above beta blockers is recommended for patients with current or prior symptoms of HFrEF.

Studies indicate that beta blockers should be introduced early to prevent further remodeling in an already structurally abnormal left ventricle. They reduce LV chamber volume and improve ejection fraction. It is not necessary to maximize the ACE-I/ARB dose before a beta blocker is added to therapy. Because of their negative inotropic effects, beta blockers should be started at low doses and slowly titrated upward to avoid symptom worsening or acute decompensation.

Initiation of beta blocker treatment may produce fluid retention and worsening HF, fatigue, bradycardia or heart block, and hypotension. These are not considered absolute contraindications for future beta blocker use. Management of these adverse events includes: reducing the dose of beta blocker, separating administration time from the ACE-I, or adjusting diuretic dose. If hypotension is accompanied by hypoperfusion, beta blocker therapy may be decreased or discontinued. Abrupt withdrawal of treatment with a beta blocker should be avoided.

Diuretics. Diuretic therapy is recommended for patients with Stage C HF who have clinical evidence of fluid retention. However, because they do not alter disease progression or prolong survival, they are not recommended for patients without fluid retention. Their use is associated with a reduction in symptoms and increase in exercise tolerance.

Loop diuretics (furosemide, bumetanide, and torsemide) are potent and are most often used to restore and maintain euvolemia in HF. Additionally, loop diuretics maintain effectiveness in the presence of impaired renal function.

Thiazide diuretics (e.g., hydrochlorothiazide) and the thiazide-like diuretic metolazone are not very effective in patients with impaired renal function. Thiazides are preferred in patients with mild congestion who benefit from their anti-hypertensive properties. Thiazides are sometimes used in conjunction with loop diuretics to potentiate diuresis.

Diuretic therapy is generally started at low doses and titrated upward until urine output increases followed by secondary weight decreases. Careful titration is important to avoid volume contrac-

tion, hypotension, renal insufficiency, and electrolyte abnormalities. Hypomagnesemia and hypokalemia can predispose patients to serious cardiac abnormalities. Loop diuretics can also cause rare side effects such as ototoxicity and hypersensitivity reactions related to their sulfonamide structure. Increased diuretic doses may be needed as HF progresses. Parenteral administration, use of two or more diuretics in combination or the addition of drugs that increase renal blood flow may be utilized to overcome diuretic resistance.

Aldosterone Antagonists. Despite inhibition of angiotensin converting enzyme by ACE-I therapy, increased plasma levels of angiotensin are present in HF patients. In order to combat aldosterone's detrimental effects in these patients, aldosterone receptor antagonists may be added for patients with NYHA class II-IV, and those who have LVEF <35 percent (Stage C), to reduce morbidity and mortality. The two aldosterone receptor antagonists, also referred to as mineralocorticoid receptor antagonists (MRAs), currently on the market are spironolactone and eplerenone. Clinical trials with both agents have demonstrated a decrease in cardiovascular death and heart failure hospitalization.

However, MRAs are only recommended in men with a creatinine less than 2.5 mg/dL, or less than 2.0 mg/dL in women (should have estimated GFR >30mL/min/1.73m^2), as well as potassium level less than 5.0 mEq/L. In elderly patients or others with low muscle mass in whom serum creatinine does not accurately reflect GFR, determination of GFR or CrCl >30mL/min is recommended. Guidelines caution that inappropriate use of aldosterone receptor antagonists can lead to life-threatening hyperkalemia or renal insufficiency.

The initial dose for spironolactone is 12.5-25 mg daily, and for eplerenone is 25 mg/day. The eplerenone dose should be titrated upward to the target dose of 50 mg once daily. Every other day dosing can also be used in patients with compromised renal function (CrCl 30-50 mL/min) or a history of hyperkalemia. Careful monitoring of serum potassium, renal function, and diuretic dosing should be performed at initiation and closely followed thereafter. The risk of hyperkalemia is increased with concomitant use of higher doses of ACE-I. In most circumstances, potassium supplements are discontinued or reduced when initiating aldosterone antagonists.

Available literature neither supports nor refutes that spironolactone and eplerenone are interchangeable due to differences among aldosterone receptor antagonism selectivity. In clinical trials (not head-to-head), the incidence of gynecomastia or breast pain was higher with spironolactone (10 percent) than with eplerenone (<1 percent).

Digoxin. Digoxin possesses both positive inotropic effects and sympatho-inhibitory effects resulting in decreases in norepinephrine, renin, and possibly aldosterone levels. Treatment with digoxin has been associated with symptom improvement, improved health-related quality of life and increased exercise tolerance in patients with mild to moderate HF. In patients with HFrEF who are symptomatic despite optimal therapies, digoxin may be added. It can be used in these patients to decrease hospitalizations, but does not improve survival rates.

Digoxin dosage should be adjusted to obtain serum levels between 0.6-1.2 ng/mL. For most patients with normal renal function, this can be achieved with a dose of 0.125-0.25 mg/day. Patients who are >70 years, have impaired renal function, or have a low lean body mass, or those receiving interacting drugs may require lower doses of 0.125 mg daily or every other day. The concomitant use of clarithromycin, dronedarone, erythromycin, amiodarone, itraconazole, cyclosporine, propafenone, verapamil, or quinidine can increase the likelihood of digoxin toxicity. Common side effects include cardiac arrhythmias (heart block), gastrointestinal symptoms (anorexia, nausea and vomiting), and neurological complaints (visual disturbances, disorientation and confusion). While digoxin toxicity is usually associated with high serum levels (>2 ng/mL), it may occur with lower levels, especially in conjunction with electrolyte imbalances.

Oral Vasodilators. Nitrates and hydralazine have complementary hemodynamic actions. Nitrates are transformed in smooth muscle cells into nitric oxide causing vasodilation which produces reductions in preload. Hydralazine produces arterial vasodilation and reductions in systemic vascular resistance by increasing intracellular cyclic guanosine monophosphate and promoting smooth muscle relaxation.

Combination therapy with hydralazine and nitrates is recommended to reduce morbidity and mortality for patients 1) self described as African Americans with NYHA Class III-IV HFrEF receiving optimal therapy with ACE-I and beta blockers, unless contraindicated; or 2) in patients with current or prior symptomatic HFrEF who cannot be given an ACE-I or ARB because of drug intolerance, hypotension, or renal insufficiency, unless otherwise contraindicated. Adverse effects of these vasodilators include headache, dizziness, and gastrointestinal complaints. Hypotension can be a limitation for use in the elderly.

Hydralazine and isosorbide dinitrate may be given together as a fixed combination tablet (37.5 mg/20 mg) or separately, three times a day. As with other therapies, an initial low dose and slow titration upward is recommended to enhance tolerance. The large pill burden, frequency of administration, and high incidence of adverse reactions may lead to poor adherence.

Drugs that are known to adversely effect the clinical status

of patients with current or prior symptoms of HFrEF are potentially harmful and should be avoided or withdrawn when possible. These include most antiarrhythmic drugs, most calcium channel-blocking drugs (except amlodipine), NSAIDs, and thiazolidinediones.

Reducing Hospitalizations in HF Patients

As previously established, heart failure is one of the most common reasons for hospital admissions, particularly in patients aged 65 years and older. HF readmission rates have been reported to be as high as 44 percent within the first six months. Data from Medicare patients indicate that readmission rates are almost 20 percent within 30 days. The Centers for Medicare and Medicaid Services (CMS) has mandated that health systems report their heart failure quality measures. The Affordable Care Act's Readmission Reduction Program allows CMS to adjust payments to hospitals based on the system's readmission performance compared to the national average. Penalties may be up to a 2 percent payment reduction.

There are numerous specific interventions showing decreased readmission for heart failure that have been published in the literature. However, they are narrow in scope and may focus on just one aspect of patient care. In a recent article by Sperry, Ruiz, and Najjar, the authors propose that interventions may be placed into six categories: 1) quality of medical management, 2) early reassessment, 3) health literacy, 4) neuropsychological status, 5) financial means, and 6) functional status. The authors suggest that combining multiple interventions into a comprehensive patient-centered model is ideal. Health systems are encouraged to develop a disease state management program that meets the unique needs of the system and population.

Medical management interventions are related to aggressively treating acute congestion and implementing evidence-based maintenance medications. In the OPTIMIZE-HF trial, inpatients were managed with the help of protocols to enact evidence-based guidelines before hospital discharge. The study resulted in a trend toward reductions in rehospitalizations and post discharge death.

Early reassessment of recently discharged patients is vital. Higher early follow-up rates are correlated with a lower risk of 30-day readmission. Debate continues on the ideal type and frequency of outpatient contact to best manage heart failure and readmission rates. Methods of contact may include phone calls, office appointments, home visits, and telemonitoring.

Considering a patient's health literacy is important as it may provide a measure of the patient's ability to read, understand, and manage their health care. Basic heart failure education for all patients is recommended in the guidelines, and has been proven to be a successful component of disease management programs. However, there will be variability among patients. Some patients will have a firm grasp of their disease. These patients may understand the usefulness of medications and expected side effects, and self-monitoring strategies, and appropriate diet and exercise recommendations. Others will have difficulty with medication and diet compliance and adherence.

Interventions that address psychiatric disorders (i.e., depression and anxiety), dementia, and social support structures may be considered. Financial limitations as a barrier to achieving quality heart failure care should also be considered. Economic hardships are often coupled with deficits in education, cognition, mood, or functional status.

The Pharmacist's Role in Heart Failure

Pharmacist involvement in the management of heart failure patients has been shown to reduce heart failure hospitalizations with trends towards a reduction in mortality. Nonadherence to either medication or lifestyle modifications is a major cause for readmission. Medication nonadherence rates are estimated to be between 40 to 60 percent. Pharmacists are well suited to optimize drug therapy regimens, and identify barriers to medication adherence, as well as educate patients and the health care team about medication-related topics.

Most of the pharmacist intervention studies with a medication adherence outcome demonstrated significant improvements when compared with a control group. Pharmacist intervention studies included pharmacist counseling, pharmacy education, pharmacy consultation, pharmacists as part of a multi-disciplinary team, postdischarge pharmaceutical care plus telemanagement, medication reconciliation services, pharmacist discharge services, and pharmacist home visits in varying capacities and intervals.

In a study by Davis *et al.*, the authors discussed significant predictors for nonadherence to HF medications. These include those with more severe HF, a greater number of concomitant illnesses, living alone, no health insurance, higher medication copays, or hospitalization within six months of the initial HF admission. Health care providers, including pharmacists, may utilize these predictors to identify patients at risk and those who may benefit from more intense monitoring and education.

Not only can pharmacists increase adherence to HF medications, but they can assist in the avoidance of medications that may exacerbate heart failure symptoms. A large number of HF patients are elderly with other disease states, and are at risk for polypharmacy. Pharmacists may identify these patients through medication reconciliation or medication therapy management services.

Summary

Heart failure is a complex and progressive clinical syndrome that results in diminished quality of life, significant morbidity and mortality, and substantial economic burden. In general, the goals of treatment are to minimize structural changes and slow the progression of HF in order to maintain functionality and improve survival. HF Stage-based guidelines are available to direct pharmacologic treatment. Studies have shown that pharmacists improve medication adherence which can positively impact hospital readmission rates.

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This lesson is a knowledge-based CE activity and is targeted to pharmacists in all practice settings.

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1. Heart failure (HF) can result from any disorder that:
   a. increases ventricular filling.
   b. reduces ventricular filling.

2. What is the single most important modifiable risk factor for HF in the United States?
   a. Diabetes mellitus
   b. Atherosclerosis
   c. Chronic alcoholism
   d. Hypertension

3. All patients with HF present with volume overload.
   a. True
   b. False

4. According to the American Heart Association, what type of HF do patients with an ejection fraction of ≤40 have?
   a. HFrEF
   b. HFpEF
   c. Diastolic HF
   d. Stage A HF

5. Patients with structural heart disease, but no signs of HF or symptoms have:
   a. Stage A HF.
   b. Stage B HF.
   c. Stage C HF.
   d. Stage D HF.

6. All of the following are reasonable options to control hypertension in patients with HFpEF EXCEPT:
   a. ACE inhibitors.
   b. ARBs.
   c. digoxin.
   d. beta blockers.

7. The most common causes of ACE inhibitor intolerance are:
   a. hypotension and hypoperfusion.
   b. visual disturbances.
   c. hyperkalemia.
   d. cough and angioedema.

8. All of the following beta blockers have been shown to reduce mortality in large HF trials EXCEPT:
   a. atenolol.
   b. bisoprolol.
   c. carvedilol.
   d. metoprolol XL.

9. Which of the following classes of drugs is recommended only for patients with Stage C HF who have clinical evidence of fluid retention?
   a. ACE inhibitors
   b. Beta blockers
   c. Diuretics
   d. ARBs

10. Which diuretics are most often used to restore and maintain euvolemia in HF?
   a. Thiazide diuretics
   b. Loop diuretics

11. Guidelines caution that inappropriate use of aldosterone receptor antagonists can lead to life threatening:
   a. hypokalemia.
   b. hyperkalemia.

12. In clinical trials, gynecomastia was reported to be higher with:
   a. spironolactone.
   b. eplerenone.

13. All of the following can increase the likelihood of digoxin toxicity with concomitant use EXCEPT:
   a. clarithromycin.
   b. amiodarone.
   c. phenytoin.
   d. cyclosporine.

14. Which of the following may be given to reduce morbidity and mortality in patients with current or prior HFrEF who cannot take an ACE inhibitor or ARB?
   a. Oral vasodilators
   b. Beta blocker
   c. Digoxin
   d. Diuretic

15. In one study, significant predictors for nonadherence to HF medications included all of the following EXCEPT:
   a. no health insurance.
   b. many concomitant diseases.
   c. mild HF.
   d. living alone.

Completely fill in the lettered box corresponding to your answer.

1.	[a]	[b]		6.	[a]	[b]	[c]	[d]	11. [a] [b]
2.	[a]	[b]	[c] [d]	7.	[a]	[b]	[c]	[d]	12. [a] [b]
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5.	[a]	[b]	[c] [d]	10.	[a]	[b]			15. [a] [b] [c] [d]

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