Heart failure (HF) in older adults presents challenges that are different in many ways than those for younger adults. Diagnosis of HF in older adults can be delayed due to attributing early symptoms to normal changes of aging or, in the setting of a normal ejection fraction, failing to appreciate diastolic heart failure. Moreover, treatment of HF in the elderly is often complicated by comorbidities and polypharmacy. The long-term care setting can present even more challenges, yet can be made easy by following a simple mnemonic DEFEAT-HF. After making a clinical Diagnosis and determining the Etiology, Fluid volume must be assessed to achieve euvaolemia, and Ejection frAction must be determined to guide Therapy. (J Am Med Dir Assoc 2008; 9: 383–389)

Keywords: Chronic heart failure; nursing home; assessment; management

Heart failure (HF) is a complex cardiac syndrome in any age group. However, geriatric HF is more complex as most of these patients suffer from concomitant functional and cognitive limitations, multiple morbidities, and polypharmacy. Over 80% of an estimated 5 million HF patients in the United States are 65 years of age and older and HF is the leading cause of hospital admission in this age group.1 Cardiovascular disease is the single largest diagnostic category in the long-term care setting.2 Although the precise prevalence of HF in long-term care settings is unknown, a significant number of an estimated 2 million long-term care residents 65 years and older in the United States have HF. Thus, HF is also a major long-term care syndrome, made even more complex by concomitant functional and cognitive limitations, comorbidities, and polypharmacy, which are more prevalent in long-term care settings.3,4 This complexity and the lack of scientific evidence and guideline recommendations specific to this population make assessment and management of HF in the long-term care setting challenging. In addition, patient and family preferences and variations in clinician practice patterns may result in poor quality of care in elderly HF patients in the long-term care setting.5,6

LONG-TERM CARE HEART FAILURE: CASE SCENARIOS

Case 1

An 81-year-old white man with a history of hypertension and old myocardial infarction was recently hospitalized after progressive dyspnea. In the 6 months prior to hospital admission, he developed dyspnea on minimal exertion, orthopnea, and paroxysmal nocturnal dyspnea. Prior to this he was rather physically active. He also developed leg swelling and gained about 10 pounds in weight. He ignored his symptoms, did not call in for a clinic appointment or go to an emergency room. He was directly hospitalized from his primary care physician’s office after a routine visit. He denied chest pain, cough, wheezing, or dizziness. His vital signs were stable. At 45 degrees, the top of his jugular venous pulsation was about 4 cm from the sternal angle and there was a positive hepatojugular reflux. He also had a right-sided third heart sound, best appreciated at the left sternal border, and pulmonary rales at both lung bases. During his 5-day hospital stay, he once...
became somewhat confused and mostly rested in bed. He responded nicely to intravenous diuretics, regained his baseline weight, and was sent to a nursing home for reconditioning and rehabilitation. His electrocardiogram showed abnormal Q waves in the chest leads, and his initial chest x-ray finding of pulmonary congestion was cleared before discharge. An echocardiogram done in the hospital showed an ejection fraction of 35%. Before discharge his serum creatinine was 1.8 mg/dL and his serum potassium was 3.8 mEq/L. His discharge medications included furosemide 40 mg every morning, metoprolol tartrate 12.5 mg twice a day, and verapamil 180 mg once a day that he had been taking for his high blood pressure.

Case 2

A 76-year-old African American woman who was a bed- and wheel chair–bound long-term care resident for the preceding 3 years had 2 recent hospital admissions for worsening HF. She had diastolic HF, which is clinical HF in the presence of normal or near normal ejection fraction. An echocardiogram from her last hospital admission showed her left ventricular ejection fraction to be more than 55%. She also has a history of hypertension, diabetes, atrial fibrillation, chronic kidney disease, emphysema, and right hemiparesis from prior stroke. Over the preceding several weeks, she had been showing signs of dyspnea and fatigue on minimal exertion such as moving in and out of the bed to wheel chair, or changing clothes. She had no dyspnea at rest, and reported no orthopnea or paroxysmal nocturnal dyspnea. However, she slept in a hospital bed, the head end of which was already raised about 45 degrees. Daily weights were not routinely recorded because of her difficulty getting up on a scale. On a physical examination, she was tachypneic with a blood pressure of 180/65 mm Hg and a heart rate of 98 beats per minute. She refused to be moved to the bed for an examination of her jugular venous pressure. However, her external jugular veins were distended bilaterally while she was sitting on her wheel chair. The right side did not show any pulsation, but pulsations could be seen at her left external jugular vein about 7 to 8 cm above her sternal angle. A compression of her abdomen caused distension of her left external jugular vein that lasted for most of the 10-second duration of the compression. This confirmed that her left external jugular vein was connected to the right atrium and could be reasonably used to estimate her jugular venous pressure. She also had a right-sided third heart sound but no pulmonary râles or wheezing. She had a history of chronic leg edema from venous insufficiency. An accentuated second heart sound at left fifth intercostal space suggested pulmonary hypertension, with an estimated pulmonary artery systolic pressure of 45 to 50 mm Hg. Her electrocardiogram was normal and chest x-ray showed cardiomegaly but no pulmonary congestion. Her serum creatinine and potassium were respectively 1.4 mg/dL and 4 mEq/L. In addition to her insulin and bronchodilators, she was also on torsemide 40 mg daily, potassium chloride 10 mEq twice a day, lisinopril 10 mg daily, amiodipine 5 mg daily, and digoxin 0.25 mg daily.

DEFEAT-ING HEART FAILURE IN THE LONG-TERM CARE SETTING

An understanding of the assessment and management of geriatric HF can be the basis of a solid foundation for good care of HF patients in the long-term care settings. Care for HF in older adults in the long-term care setting can be simplified by following the simple mnemonic “DEFEAT”-HF.7 DEFEAT stands for Diagnosis, Etiology, Fluid volume, Ejection fraction, Therapy, and Assessment. Diagnosis (D): A diagnosis of heart failure before ordering any test, especially an echocardiogram. A normal ejection fraction in a patient without a clinical diagnosis may confound the diagnosis process. If a clinical diagnosis is already made or patient had hospitalization due to heart failure, check how the diagnosis was established.

E = Etiology

Hypertension and myocardial infarction are the two most common causes. Expect multiple causes in elderly heart failure patients in the long-term care setting. Continued treatment of risk factors such as high blood pressure and myocardial ischemia is important to prevent disease progression.

F = Fluid volume

Single most important physical examination. Best assessed by estimating jugular venous pressure in the neck. External jugular veins are very useful if their limitations are appreciated. Being superficial veins they are subject to external pressure or internal obstruction. Thus a distended external jugular vein without visible pulsation should not generally be used to estimate venous pressure.

EA = Ejection fraction

Single most important test after a clinical diagnosis of heart failure has been made. It is a marker of prognosis (generally patients with lower ejection fraction have a poorer prognosis) and a guide to therapy.

T = Treatment

Heart failure therapy can be divided into symptom-relieving and life-prolonging therapies. The two most important life-prolonging therapies for heart failure patients with low ejection fraction (<45%) are ACE inhibitors (or an ARB if intolerant to ACE inhibitor) and beta-blockers (those approved for use in heart failure and shown to reduce mortality, namely metoprolol extended release and carvedilol). Survival benefit of these drugs in diastolic heart failure has not yet been proven. All symptomatic heart failure patients with fluid volume overload should be treated with a diuretic and once euvolemia is achieved the lowest possible dose should be used to maintain euvolemia in conjunction with salt and fluid restriction. Digitalis in low doses (0.125 mg or less per day) should be used to reduce symptoms and risk of hospitalization. In patients who cannot tolerate beta-blockers at low dosages, digitalis can also reduce mortality.

Table 1. DEFEAT Heart Failure

| D = Diagnosis | Make a clinical diagnosis of heart failure before ordering any test, especially an echocardiogram. A normal ejection fraction in a patient without a clinical diagnosis may confound the diagnosis process. If a clinical diagnosis is already made or patient had hospitalization due to heart failure, check how the diagnosis was established. |
| E = Etiology | Hypertension and myocardial infarction are the two most common causes. Expect multiple causes in elderly heart failure patients in the long-term care setting. Continued treatment of risk factors such as high blood pressure and myocardial ischemia is important to prevent disease progression. |
| F = Fluid volume | Single most important physical examination. Best assessed by estimating jugular venous pressure in the neck. External jugular veins are very useful if their limitations are appreciated. Being superficial veins they are subject to external pressure or internal obstruction. Thus a distended external jugular vein without visible pulsation should not generally be used to estimate venous pressure. |
| EA = Ejection fraction | Single most important test after a clinical diagnosis of heart failure has been made. It is a marker of prognosis (generally patients with lower ejection fraction have a poorer prognosis) and a guide to therapy. |
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tion frAction, and Treatment (Table 1). The process of HF care in the long-term care setting should begin with a clinical Diagnosis of HF, followed by an assessment of Etiologies for HF. Assessing Fluid volume and achieving fluid balance is the most important clinical goal for both patients’ quality of life and reducing hospital admission. Ordering an echocardiogram to know the left ventricular Ejection frAction is the single most important laboratory test once a diagnosis of HF is established. Although major guidelines provide few evidence-based recommendations for HF patients in long-term care settings, and Treatment must be individualized, it is important to be familiar with one of the major HF guidelines.6,8,9

DIAGNOSIS OF HEART FAILURE

HF is a clinical diagnosis. HF cannot be definitively diagnosed by any laboratory tests. This makes the diagnosis of HF challenging. A clinical diagnosis of HF depends on a constellation of symptoms, signs, and other findings. Our Case 1 was a rather newly diagnosed HF patient who was also new in the long-term care setting. He already had one episode of HF requiring hospitalization, thus a diagnosis of HF had already been established. More importantly, the collection of his symptoms and signs before his hospital admission were suggestive of clinical HF. However, whenever possible it is useful to verify the history of initial HF symptoms. Our Case 2 also had known HF, a frequent diagnosis in the long-term care setting. Therefore, the process of establishing a new clinical diagnosis is often not necessary. However, residents of long-term care facilities without HF may develop new-onset HF, requiring the clinician to make a diagnosis. Because long-term care residents may not often be physically active, a history of dyspnea on exertion may not always be available, and their symptoms may progress to dyspnea at rest.3,10 A history of dyspnea on exertion or fatigue may need to be elicited by asking questions about their routine daily activities such as moving in and out of the wheel chair instead of how many blocks one can walk.

ETIOLOGY OF HEART FAILURE

Hypertension and coronary artery disease are the 2 most common risk factors for HF in older adults.11 Other less common risk factors are chronic kidney disease, diabetes, atrial fibrillation, stroke, and valvular heart disease. Because HF is a syndrome and not a disease, almost always it is caused by one of these risk factors, which can often be determined during history or chart review. Our Case 1 had a history of hypertension and myocardial infarction, both potential risk factors. Our Case 2 had hypertension, diabetes, stroke, and atrial fibrillation, all potential causes of HF. It is important to identify underlying risk factors even if they are not the specific etiology of the HF. For example, uncontrolled blood pressure or ongoing myocardial ischemia that may or may not have caused the initial HF may cause significant myocardial damage to the failing heart. This may lead to disease progression, precipitate acute HF, and increase risks of death and hospitalizations.

Normally, when an underlying etiology cannot be found or an ongoing insult such as myocardial ischemia is identified, it is recommended that patients should be referred to a cardiologist. However, in the long-term care setting, a decision of cardiology referral for identification of an etiology needs to be individualized depending on patient and family preferences, and the patient’s overall health and prognosis. However, for patients with ongoing myocardial ischemia, medical therapy should be optimized even if they refuse invasive diagnostic and/or therapeutic procedures, or a cardiology referral is not made for some other reason. Nonadherence to instructions for salt and fluid restriction, and medications may precipitate acute HF. In particular the use of table salt should be discouraged. However, a too restrictive salt regimen may lead to malnutrition and weight loss and should be carefully avoided. Efforts to identify such exacerbating factors should be done in patients with repeated hospitalizations as in Case 2.

FLUID VOLUME MANAGEMENT IN HEART FAILURE

HF patients with fluid volume overload often experience dyspnea and fatigue, and have poor quality of life. Protracted cases of fluid volume overload may lead to decompensation and hospital admission. Moreover, patients with fluid volume overload often cannot tolerate initiation or continuation of life-saving drugs such as a beta blocker or an angiotensin-converting enzyme (ACE) inhibitor.

Euvolemia is a key goal in HF management and can often be achieved with proper assessment of fluid volume and appropriate diuresis. However, a proper fluid volume assessment is essential for optimal fluid volume management. While an elevated jugular venous pressure is a sensitive marker of fluid overload,9 this important component of the physical examination is often done improperly, in part due to several myths.

The first myth is that only the internal jugular vein is useful in the estimation of jugular venous pressure. From an anatomic point of view, the internal jugular veins would be ideal for the estimation of jugular venous pressure. However, also from an anatomic point of view, internal jugular veins are very difficult to use for the estimation of jugular venous pressure in chronic HF because for most of their course in the neck they lie behind the largest group of muscles in the neck, the sternocleidomastoids. Therefore, internal jugular veins tend to underestimate jugular venous pressure in both chronic and acute HF.12–14 External jugular veins, on the other hand, are easily visible, and can be a more practical alternative vein for the estimation of jugular venous pressure as long its limitations are understood.15

The second myth about jugular venous pressure estimation is that patients need to be positioned at 45 degrees incline and that the distance between the right atrium and sternal angle is always 5 cm regardless of body position. While a visible jugular pulsation in the neck at 45 degrees would suggest elevated jugular venous pressure, it is not very useful when the pressure is too high (when the top of the jugular pulsation may be behind the angle of jaw) or too low (when the top of the jugular pulsation would be behind the clavicle). Therefore, patients need to be in a supine or sitting position or any other position in between, so that the top of the venous pulsation can be seen and its distance from the right atrium can be estimated. Once the top of the jugular venous pula-
tion is identified, its distance from the sternal angle should be estimated (Figure 1). If the top of the jugular pulsation is above the level of the sternal angle, that distance should be added to the estimated distance between the sternal angle and right atrium. However, if the top of the jugular pulsation is below the level of the sternal angle, this distance should be subtracted from the estimated distance between the sternal angle and right atrium.

The third myth is about the distance between the right atrium and the sternal angle. It has often been taught in textbooks, classrooms, and at bedside that the distance between the right atrium and sternal angle is 5 cm regardless of body position. However, a study based on chest computed tomography (CT) scans of 160 patients has determined that the distance between the right atrium and sternal angle would be 5 cm only in the supine position.16 However, at 30 and at more than 45 degrees of elevation, this distance would be 8 and 10 cm respectively (Figure 1).

For example, in Case 1, the external jugular venous pulsation was about 4 cm above the sternal angle at 45-degree elevation. At this elevation, the distance between the right atrium and the sternal angle is about 10 cm. So, the jugular venous pressure would be estimated at about 14 cm of water, which was rather elevated, and was consistent with his symptoms. In Case 2, the top of the jugular venous pulsation in the mid-neck area in the sitting position was 7 to 8 cm from the sternal angle. Adding 10 cm (the distance between right atrium and sternal angle in sitting position) to this, her jugular venous pressure was about 17 to 18 cm of water (Figure 1).

**LEFT VENTRICULAR EJECTION FRACTION IN HEART FAILURE**

After a clinical diagnosis has been established, an echocardiogram should be ordered to determine left ventricular ejection fraction. This is the single most important test for an HF patient. It is important because it assists in guiding medical management and provides prognostic information about HF patients. Generally speaking, patients with low ejection fraction, as in Case 1, have poorer prognosis than those with normal ejection fraction, such as Case 2.17 However, the good news is that the prognosis in HF patients with low ejection fraction can be improved with neurohormonal antagonists, such as ACE inhibitors or angiotensin receptor blockers, use of which is considered a marker of good quality of care. For most HF patients in the long-term care setting, ejection fraction will likely already be known, as was the case with our Case 1 and Case 2. If this information is not available, an echocardiogram should be ordered, unless contraindicated by patient and family preferences, and life expectancy of the patients. However, with increasing use of mobile echocardiography services, left ventricular ejection fraction should be obtainable for most HF patients in the long-term care setting.

**TREATMENT OF HEART FAILURE IN THE LONG-TERM CARE SETTING**

Treatment of HF in the long-term care setting can be divided into symptom-relieving and life-prolonging therapies. As symptoms of HF are similar regardless of ejection fraction, symptom-relieving therapies are very similar for both systolic and diastolic HF. However, life-prolonging therapies are generally indicated for systolic HF patients.

**ACE Inhibitors**

All systolic HF patients, whether or not they are symptomatic, should be treated with an ACE inhibitor. If they are intolerant of ACE inhibitors, an angiotensin receptor blocker is an alternative. Cough is the most common reason for intolerance of ACE inhibitors and can less commonly occur with angiotensin receptor blockers. Case 1 was not receiving an ACE inhibitor and unless he had a history of prior allergic reaction, he should be offered these drugs. However, a common reason for underuse of ACE inhibitors in HF patients is...
kidney function. \(5,18,19\) Generally a rise in serum creatinine is small and transient and should not be a reason for non-initiation or discontinuation of an ACE inhibitor. HF patients with kidney disease have poor prognosis, and evidence suggests that these patients are equally likely to benefit from these drugs. Although the beneficial effect of these drugs has not been proven in patients with diastolic HF, they may provide renoprotection for diastolic HF patients with chronic kidney disease. Data from younger HF patients suggest that the effect of kidney disease may be worse in diastolic than in systolic HF. \(20\) If our Case 2 had not been receiving an ACE inhibitor, it could be considered given her kidney disease. Hyperkalemia is not uncommon in elderly HF patients and ACE inhibitors may be partly responsible. However, before stopping ACE inhibitors, one should look for other sources of potassium including potassium supplements. Serum potassium levels can be kept within the normal range by once or twice weekly use of small doses of cation-exchange resins such as kayexalate. This may allow continuation of ACE inhibitors; however, the long-term effect of this approach is not known.

**Beta-Blockers**

All systolic HF patients should also be prescribed a beta-adrenergic blocker approved for HF unless contraindicated. Metoprolol extended release is a beta-1 selective blocker with little effect on blood pressure and may be more suitable for frail elderly patients with low blood pressure. \(21,22\) Carvedilol, on the other hand, may be more suitable for those with concomitant high blood pressure. There is no need to wait to maximize the dose of ACE inhibitors before a beta-blocker is initiated. Regular metoprolol tartrate may not be as effective as metoprolol extended release and should not be used in systolic HF. \(23\) Case 1 was receiving metoprolol tartrate, which should be switched to extended release metoprolol succinate and titrated upward as tolerated. Extended release metoprolol succinate can be broken in half but should not be crushed. For patients who need their beta-blockers crushed, carvedilol may be used. If carvedilol cannot be used due to low blood pressure, and extended release metoprolol succinate must be crushed, it should be given twice a day. The concomitant use of beta-blockers and calcium channel blockers may increase the risk of cardiac bradyarrhythmias and should be avoided. However, more importantly, given the low ejection fraction of Case 1, his verapamil should be discontinued for its potential negative inotropic effects. Dihydropyridine calcium channel blockers such as amlopidine or felodipine may be safely used if needed for better blood pressure control in systolic HF. \(24\) However, if his blood pressure is not well controlled, his extended release metoprolol should be switched to carvedilol, which is a better antihypertensive drug than metoprolol. This may obviate the need for another antihypertensive drug, an important consideration in patients already receiving many drugs. Case 2 was not receiving a beta-blocker. However, given her high blood pressure and relatively high pulse rate, a beta-blocker, preferably one with better antihypertensive properties, such as carvedilol or the newly approved beta-blocker nebivolol may be a better choice and may later allow discontinuation of her amlopidine. For patients showing signs of orthostasis, a sitting blood pressure, instead of a supine blood pressure, should be used to guide antihypertensive therapy.

**Aldosterone Antagonists**

Spironolactone may be cautiously used in advanced and symptomatic systolic HF patients. Elderly HF patients with declining kidney function who are also using ACE inhibitors are particularly at risk of hyperkalemia from spironolactone. This drug should also be avoided in patients with kidney disease and high serum potassium, and serum creatinine and potassium should be monitored closely in other patients. \(25\)

**Diuretics**

Most HF patients, regardless of ejection fraction, need diuretics to achieve and maintain euvolemia. While it is known that diuretics may activate neurohormonal pathways that can have adverse consequences, \(26\) for symptom control related to volume overload, diuretics are essential. \(9\) Many elderly HF patients do not receive adequate dosages of diuretics needed to achieve euvolemia. Both Cases 1 and 2 were receiving inadequate dosages of diuretics. This was apparent from their symptoms and signs of fluid volume overload. In addition, diuretics are generally less effective in the presence of kidney disease and larger doses are needed. Based on the Modification of Diet in Renal Disease or MDRD formula, both Case 1 and 2 had chronic kidney disease with estimated glomerular filtration rates of 39 and 47 mL/min/1.73 m\(^2\) respectively. \(27\) The effectiveness of diuretics can also be assessed by asking patients if they have increased urinary frequency after taking diuretics and by monitoring daily weight. Due to practical logistical difficulties with daily weight in the long-term care setting, it should be restricted to residents with unstable HF and frequent hospitalizations. Once euvoelmia is achieved, the lowest possible dose of diuretic should be used to maintain euvoelmia and avoid complications such as electrolyte imbalances, dehydration, hypovolemia, hypoperfusion, orthostasis, and potential falls. The use of diuretics should be combined with patient education to adhere to a low-salt diet and fluid restriction of less than 2 liters per 24 hours. This should generally not be an issue as residents in long-term care may be more prone to dehydration than volume overload. Torsemide is a less potent activator of aldosterone than furosemide, and less likely to cause hyperkalemia. \(28\) However, despite being a generic drug, it is expensive. The long-term effect of neither diuretic has been tested in large randomized clinical trials.

**Digitalis**

All systolic and diastolic HF patients in the long-term care setting who are symptomatic despite therapy with the above drugs, should be prescribed digoxin in low doses. Digoxin is known to reduce symptoms and hospitalizations in all HF patients regardless of ejection fraction. \(29,30\) However, in low doses (\(\leq 0.125 \text{ mg/day}\)) digoxin is more likely to result in low serum digoxin concentrations (\(<1.0 \text{ ng/mL}\)), which has been shown to reduce mortality. \(29,31\) HF patients who cannot tolerate beta-blockers should be given digoxin, as it has been

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shown to reduce mortality in low doses in patients already receiving an ACE inhibitor. For most HF patients in the long-term care setting a starting dose of 0.125 mg/day or less will suffice. However, in patients with advanced age and in those with kidney disease, such as our Case 1 and Case 2, digoxin should be prescribed as 0.0625 mg daily or 0.125 mg every other day. The dose of digoxin should be appropriately reduced in our Case 2 and if there is any clinical evidence of digoxin toxicity, her serum digoxin level should be checked. If prescribed in low doses, the risk of digoxin toxicity is low and there is no need for routine or frequent serum digoxin monitoring. However, serum digoxin levels should be checked if clinical digoxin toxicity is suspected and before increasing the dose of digoxin for patients who remain symptomatic despite the initial low dosages.

Management of Complications

Hypokalemia is common in HF, in part due to neurohormonal activation, and also caused by diuretics. Serum potassium levels less than 4 mEq/L have been associated with increased morbidity. Therefore, potassium levels should be maintained at least 4 mEq/L and preferably to around 4.5 mEq/L. Oral potassium supplements are generally effective in achieving normokalemia. However, spironolactone may be preferable over potassium supplement for maintenance of normokalemia. Potassium supplements are not effective in correcting other electrolyte imbalances such as hypomagnesemia, commonly associated with hypokalemia. Case 1 should be prescribed oral potassium supplement to achieve normokalemia. Oral potassium in Case 2 may be replaced with spironolactone 12.5 or 25 mg daily, which will not only likely correct her potassium balance but also suppress aldosterone, which is being activated both by her HF and diuretic therapy. Aldosterone is a key neurohormone in HF that is associated with myocardial fibrosis, disease progression, and poor prognosis.

Management of Comorbidities

Common cardiovascular comorbidities such as hypertension, coronary artery disease, hypercholesterolemia, chronic kidney disease, peripheral arterial disease, cerebrovascular disease, and atrial fibrillation should be adequately treated. Treatment of noncardiovascular comorbidities is also important. Depression is common in the elderly and may present atypically. Depression is associated with poor outcomes including increased risk of placement in the long-term care settings. Poor physical activity may be a negative prognostic marker in HF. Pain from osteoarthritis or other causes should be optimally treated to allow optimal physical activity.

CONCLUSIONS

HF in the long-term care setting is a complex syndrome, the management of which can be challenging. However, following a simple 5-step protocol called “DEFEAT-HP” one can organize the process: Starting with a clinical Diagnosis and establishing an Etiology, one should assess Fluid volume and achieve euvolemia and determine Ejection frAction to guide Therapy. Guidelines provide little specific information for HF patients in the long-term care setting. However, familiarization with a major national guideline may provide a foundation on which HF treatment in the long-term care setting can be individualized.

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