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Chronic Heart Failure in Older Adults

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Most of the estimated 6 million heart failure (HF) patients in the United States are 65 years and older. The vast majority of the nearly 700,000 patients who are newly diagnosed with HF every year are also older adults, and the incidence of HF increases with aging, approaching 10 per 1000 persons for those 65 years and older.¹ The annual cost of HF was estimated to be \$40 billion in 2010, nearly half of which was spent for inpatient care.² HF is the leading cause of hospitalization among Medicare beneficiaries and it is listed as the primary discharge diagnosis for an estimated 1 million hospitalizations in the United States. HF is responsible for about 60,000 deaths annually in the United States, and those 65 years and older carry most of the brunt of this mortality. HF is listed as a secondary diagnosis in more than 200,000 other deaths annually.¹

HF is a clinical syndrome and is often difficult to diagnose, because unlike acute myocardial infarction (AMI) or stroke, there is no single test or procedure that can definitively confirm or rule out HF. The diagnosis and management of HF in older adults, who suffer from multiple morbidities and polypharmacy, are particularly difficult.³ A typical geriatric HF patient is an older woman with diastolic HF or clinical HF with normal or near-normal left ventricular ejection fraction (LVEF), often with a history of hypertension. Often these patients have other morbidities such as coronary artery disease (CAD), atrial fibrillation, diabetes mellitus, arthritis, chronic kidney disease (CKD), and depression, and take multiple medications. Yet, the clinical presentation of HF is similar, regardless of the LVEF.⁴ The diagnosis and management of HF in an older adult is clearly more complex and challenging than in a younger HF patient, who is likely to be a man with impaired LVEF, often with a history of CAD and/ or AMI. The diagnosis and management of HF in older adults is made more difficult by the fact that major randomized clinical trials (RCT) of HF have often excluded older adults and those with diastolic HF.^{5,6}

However, the diagnosis and management of HF in older adults can be simplified by a clear understanding of the key facts, and following a structured protocol that covers all the key areas of the process. In this article, the author presents several cases of geriatric HF to illustrate the heterogeneity and complexity of HF in older adults, and demonstrate how the

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process of diagnosis and management can be simplified by the use of the mnemonic DEFEAT-HF: Diagnosis, Etiology, Fluid volume, Ejection fraction, And Treatment of Heart Failure (Table 1).^{3,7,8}

CASE PRESENTATIONS

Case 1

A 79-year-old man without a history of HF presented with progressive dyspnea on exertion (DOE) and leg swelling for 6 months (Table 2). He had no history of dyspnea at rest, orthopnea, paroxysmal nocturnal dyspnea (PND), cough, wheezing, or chest pain. He was receiving lisinopril and propranolol. There was no history of emergency department visits or hospitalizations due to HF or dyspnea. His vital signs were within normal limits. His physical examination was remarkable for mild pitting edema around his ankles and lower leg areas. He had normal jugular venous pressure (JVP), no hepatjugular reflux (HJR), no third heart sound (S3), and no pulmonary râles. His electrocardiogram and chest radiograph findings were within normal limits. His medical history revealed 2 etiologic risk factors for HF: a history of generally well-controlled hypertension and CAD with a known old AMI (Table 3).

At a 45° incline, no jugular pulsation could be observed in his neck. Further, when the head of his bed was lowered to at about 35°, there was no visible pulsation of his internal jugular veins (IJV). However, at this position the top of the pulsations of his external jugular veins (EJV) was visible in the lower neck area. When the head of the bed was lowered to about 30°, the pulsation of his EJV was now visible in the middle of his neck. The top of his EJV pulsation was about 2 cm vertically below his sternal angle (Table 4). In this near-supine position, a firm pressure on his abdomen slightly raised the top of his EJV pulsation by about 1 cm that lasted a few seconds while the abdominal pressure was maintained. An echocardiogram showed an LVEF of 35%.

Case 2

An 86-year-old woman with a history of HF (LVEF unknown) presented with a history of fatigue and dyspnea on minimal exertion of 1 month's duration (Table 2). Her DOE was so extreme that she reported dyspnea on turning sides in bed. She was sleeping in a recliner to avoid orthopnea, and her occasional PND had ceased since she started sleeping on the recliner. She reported no chest pain. She also complained of fatigue on minimal exertion, right upper quadrant pain, nausea but no vomiting, loss of appetite, and severe leg swelling. Her clothes were wet from the oozing of clear liquid from her swollen legs, and she had multiple blisters on both legs. She was receiving digoxin, atenolol, and spironolactone. Despite her symptoms, she had not visited the emergency department.

On physical examination she had elevated JVP, positive HJR, a right-sided S3, no pulmonary râles or wheezing, an enlarged soft tender liver, and severe bilateral lower extremity edema up to mid-thigh with brown pigmentation and induration of skin, and multiple blisters over the lower legs. An accentuated second heart sound at the left fifth intercostal space suggested pulmonary hypertension, with an estimated pulmonary artery systolic pressure of 50 to 55 mm Hg. She had a normal electrocardiogram. A chest radiograph revealed marked cardiomegaly, pulmonary venous congestion, and mild pulmonary edema. Except for a history of hypertension and atrial fibrillation, she had no other known etiologic risk factors for HF; the control status of her hypertension was not well known (Table 3).

At a 45° incline, her EJV could be seen distended, but there was no visible pulsation. However, when the head of her bed was raised to about 60° incline, the top of her EJV

pulsations was visible in her upper neck area. When the head of her bed was further raised to a near-sitting position, the top of her EJV was visible in the middle of her neck (Table 4). At this position, the top of her EJV pulsation was vertically nearly 8 cm above her sternal angle. A firm pressure on her abdomen made her EJV be more distended and the top of her EJV pulsation to rise by about 3 cm and be invisible behind the angle of her jaws, and remained so for about 10 seconds as the abdominal pressure was maintained. She had no visible pulsation of her IJV on either side of her neck. An echocardiogram later showed an LVEF of greater than 55%.

Case 3

An 84-year-old man presented with worsening DOE and fatigue of 2 months' duration (Table 2). He had occasional orthopnea but no PND. He had no chest pain but reported dizziness. He was recently hospitalized with a syncopal episode. Before that hospitalization, he was physically active. On further questioning, he admitted some mild DOE prior to hospitalization, but denied any dyspnea at rest, orthopnea, PND, or chest pain. A workup during hospitalization demonstrated an enlarged heart by chest radiograph, an LVEF of 25% by echocardiogram, but a normal coronary angiogram. He was discharged on furosemide, 80 mg daily. His DOE subsequently worsened, and his furosemide was increased to 160 mg daily. Over the next 2 weeks he lost more than 20 lbs. (9 kg) and his symptoms improved. His serum B-type natriuretic peptide (BNP) level was about 400 (normal <100) pg/mL. He continued on a diuretic regimen of furosemide, 160 mg a day. Four weeks later he presented with worsening fatigue and DOE. His physical examination was remarkable for a systolic blood pressure of 95 mm Hg and a low JVP. His medical history revealed hypertension and diabetes, but he had no other known etiologic risk factors for HF, including CAD (Table 3). He said his hypertension had generally been well controlled. He quit smoking about 15 years ago after about 35 pack-years of smoking.

At a 45° incline, he had no visible IJV or EJV pulsation in his neck. However, when the head of the examination table was lowered to a supine position, the top of his EJV pulsations was visible in his mid-neck, which was vertically nearly 2 cm below his sternal angle (Table 4). A firm pressure on his abdomen raised the top of his EJV by about 1 cm for about 2 second. He had no visible IJV pulsation. As already mentioned, an echocardiogram a few months earlier had shown an LVEF of 25%.

Case 4

A 76-year-old man presented with history of persistent chronic DOE and cough of more than 5 years' duration that had become worse in recent months (Table 2). He slept on a couch to avoid orthopnea, but denied dyspnea at rest or PND. The swelling of his feet, ankles, and lower leg had also worsened in recent months. He stated that he had to cut a split in the top of his socks so that he could wear them. He was not aware of a history of HF and denied any HF-related hospitalization. He was receiving furosemide 80 mg every morning. He also received bronchodilator inhalation for suspected emphysema. However, his pulmonary function test showed no obstructive lung disease and was remarkable for only mild restrictive lung disease. A review of his medical record revealed 3 values of BNP levels over the past 12 months, all of which were less than 50 (normal <100) pg/mL.

His physical examination was remarkable for elevated JVP, positive HJR, rare pulmonary rales, and severe bilateral lower extremity edema, surrounded by socks with cut edges. His electrocardiogram showed atrial fibrillation with controlled ventricular rate. His chest radiograph revealed mild cardiomegaly and pulmonary congestion but no pulmonary edema. In addition to atrial fibrillation, he also had hypertension and CAD, 2 key etiologic risk factors for HF (Table 3).

At a 45° incline, he had both his IJV and EJV visible in upper neck area. The top of his EJV pulsation was vertically 4 cm above his sternal angle and the top of his IJV was vertically 6 cm above his sternal angle. A firm pressure on his abdomen raised both IJV and EJV pulsation by 3 cm, which remained elevated for about 12 seconds as the abdominal pressure was sustained. According to a recent echocardiogram, his LVEF was greater than 55%.

Case 5

An 82-year-old woman presented with a 1-year history of dyspnea, chest tightness, and dizziness, all of which occurred at rest (Table 2). She denies orthopnea, PND, or leg swelling, and more importantly she reported no DOE. She visited the emergency department 3 times during the previous 12 months, and was hospitalized each time. Extensive tests and procedures during these hospitalizations included echocardiogram, cardiac catheterization, and magnetic resonance imaging of brain, none of which revealed any pathology. A review of her medical record revealed a history of hypertension (Table 3). Her physical examination was unremarkable. She had no evidence of clinical HF, and specifically, she had no JVP elevation or HJR.

At a 45° incline, neither her IJV nor her EJV was visible in the neck. When her head was lowered to about 30°, the top of her EJV pulsation could be seen at the base of her neck, which was 2 cm below her sternal angle (Table 4). The top of her EJV pulsation briskly moved up for a second when a firm pressure was applied on her abdomen. A repeat echocardiogram revealed normal LVEF.

DIAGNOSIS OF HEART FAILURE

HF is a clinical syndrome that is characterized by dyspnea, fatigue and swelling, most of which are manifestations of a reduced cardiac output, the hallmark pathology of HF (Table 5). Because there is no single test or procedure that can definitively diagnose or rule out HF, it is imperative that clinicians become familiar with the symptoms and signs of HF. It is also important to remember that the diagnosis of chronic HF may be a process that can span over days to months. A clinical diagnosis of HF should ideally be made before ordering an echocardiogram, as nearly half of all geriatric HF patients have normal LVEF.^{9–11} However, not infrequently, a patient may present with mild DOE and a normal LVEF from a recent echocardiogram performed in another hospital or clinic. In those circumstances, the key is to not rule out HF because the LVEF is normal. With that understanding in mind, it may be appropriate to order an echocardiogram so that LVEF data can be incorporated into the diagnostic process.

For example, Case 1 had insufficient symptoms and signs of HF to make a clinical diagnosis of HF (Table 3). However, in the context of a low LVEF, his presentation was sufficient to establish the clinical diagnosis of HF. An advantage of this approach was that it allowed early initiation of therapy with life-saving drugs such as an angiotensin-converting enzyme (ACE) inhibitor and a beta-blocker. A clinical diagnosis of HF probably could not be made until months later, by which time his HF would have further progressed and he would have developed more classic symptoms and signs of HF. Because HF is a clinical syndrome, it is almost inevitable that all HF patients will at some point in time develop overt HF. However, the nature and duration of this process is often difficult to predict. Case 2 had nearly all textbook symptoms and signs of HF. However, most HF patients fall somewhere between these two cases in their clinical presentations. Because diagnosis of HF may be difficult, it is possible that HF may have been misdiagnosed in the past. Therefore, it is important to confirm the diagnosis of HF during initial evaluation of a patient with known chronic HF. For patients who are currently euvolemic and have no clinical HF, this can often be done by

reviewing symptoms and signs of HF in the past, response to diuretics, hospitalization due to HF, and the use of current HF medications.

Dyspnea or fatigue on exertion is the most common early symptoms of HF, often accompanied by some degree of lower extremity edema (Case 1). However, both are rather nonspecific symptoms, even when jointly present, and are often not sufficient to make a clinical diagnosis of HF. Case 1 was a very active person before his dyspnea began, and as such he was bothered by his dyspnea. However, many older adults attribute their early mild symptoms of dyspnea or fatigue to older age, and respond by restricting their physical activities, which may result in delayed diagnosis, or delayed care, as in Case 2.

Orthopnea and PND are more specific HF symptoms. However, patients often sleep with their head raised to avoid orthopnea, which may also reduce the occurrence of PND. Therefore, asking about the number of pillows currently used, whether currently sleeping on a couch or in a recliner, and past episodes of orthopnea may help elicit otherwise underreported orthopnea. Orthopnea and PND are 2 major Framingham criteria for the diagnosis of HF, and their presence suggests a diagnosis of HF. However, an analysis of the Cardiovascular Health Study (CHS) suggested that many older adults with both these symptoms may not have HF.¹² In that study, 7% (388/5771) of the community-dwelling adults 65 years or older had both orthopnea and PND, yet only 20% (76/388) of those with both orthopnea and PND had centrally adjudicated HF.

Lower extremity edema is often present in HF but needs to be distinguished from pitting edema due to venous insufficiency, which may be unilateral and may predate the onset of dyspnea. Unpublished data based on the analysis by this author of the public-use copy of the CHS data obtained from the National Heart, Lung, and Blood Institute suggest that only about one-third of community-dwelling older adults with orthopnea, PND, and lower extremity edema had centrally adjudicated HF. These findings suggest that the diagnosis of HF may not be made merely on the basis of a few symptoms or signs, even if they appear rather specific or suggestive of HF, and that a clinical diagnosis of HF at times may be an ongoing process whereby an initial diagnosis may need to be reevaluated based on other accumulating evidence of HF.

An evidence of fluid volume retention in the context of classic symptoms of HF such as dyspnea and fatigue on exertion, orthopnea, PND, and lower extremity edema is most helpful in making an early clinical diagnosis of HF. An elevated JVP is the most specific sign of fluid retention in HF and is likely the most important physical examination in the process of diagnosis of HF in older adults.¹³ While most textbooks of physical examination suggest that IJV should be used for JVP estimation, many clinicians find this difficult to appreciate. In part this is because for the most part of its course in the neck, the IJV lies behind the large sternocleidomastoid muscles. Because unlike the carotid pulsation, the jugular pulsation is relatively much weaker (10 cm of water 5 7.4 mm Hg), IJV pulsations often are not transmitted well for JVP estimation. This finding is confirmed by the results from several studies that suggest underestimation of the prevalence of JVP elevation in HF.

Findings from nearly 10,000 chronic systolic HF patients enrolled in 2 major RCTs and examined by cardiologists demonstrated that slightly more than 10% had elevated JVP.^{14,15} One might argue that the low prevalence of JVP in those patients is likely attributable to the fact that most of those patients with chronic HF were euvolemic. However, the prevalence of JVP elevation was similar in a group of older adults with dyspnea who presented at the emergency department.¹⁶ The prevalence of JVP elevation would be expected to be higher in those patients. One would expect that nearly all hospitalized acute HF patients would have elevated JVP. However, findings from the OPTIMIZE-HF registry demonstrated that

only about one-third of more than 40,000 hospitalized older adults with acute decompensated HF had JVP elevation.⁴ This systematic underestimation of the prevalence of JVP elevation in chronic HF may in part be attributed to reliance on IJV for JVP estimation.

Therefore, when a clear IJV pulsation cannot be seen, EJV pulsation should be used to estimate JVP.¹⁷ The key limitation of the EJV in estimating JVP is that it is a superficial vein, and as such it is vulnerable to external pressure (e.g., contraction of the overlying platysma muscle) or internal obstruction (thrombosis or sclerosis of valves). Therefore, a distended EJV should never be used as a marker of JVP elevation or to estimate JVP. However, at times a patent EJV may also look distended, which can happen if the JVP is very high and the patient is at a 45° incline. The top of the EJV pulsation may be behind the angle of mandible. In that case, raising the head of the bed or sitting the patient make the EJV pulsation visible in the middle of the neck. Using both IJV and EJV and proper techniques, JVP can be estimated in 90% to 95% of all HF patients.

Although popularized in the medical literature and promoted by the industry, serum BNP and N-terminal pro-BNP (NT-proBNP) are rarely needed by skilled clinicians to establish a clinical diagnosis of chronic HF in older adults. These neurohormones are secreted by the failing ventricle in HF in response to myocardial wall stress. However, they may also be elevated in many conditions other than HF including aging, infection, and renal insufficiency.¹⁸ In one prospective study of 1586 patients (mean age, 64 years) with acute dyspnea, clinical diagnosis of HF was adjudicated in 744 (47%) patients by 2 independent cardiologists.¹⁹ In that study, bedside BNP data were collected on all participants in the emergency department, and values of less than 100 and less than 50 pg/mL were shown to have negative predictive values of 89% and 96%, respectively.¹⁹ However, currently there are no standard cutoffs for these neurohormones to confirm or rule out HF in general, and even less is known about their usefulness in older adults.^{18,20} In the aforementioned study, BNP was observed to be more accurate than physical signs in identifying HF in patients presenting with acute dyspnea. However, only 22% of patients in that study had JVP elevation, whereas 47% of the patients had adjudicated HF.¹⁹ This finding underscores the importance of proper estimation of JVP in older adults with HF. The limitations of the use of BNP are also illustrated in Cases 3 and 4. Therefore, a careful and proper clinical estimation of JVP is indispensable for a clinical diagnosis of HF.

ETIOLOGY OF HEART FAILURE

Because HF is a syndrome, an underlying etiology for HF must be established for all HF patients. In older adults, more than one etiologic factor may be involved. CAD and hypertension are the 2 most common risk factors (Table 4), and can often be identified during history and physical examination, sometimes with the help of a few additional tests. This identification has important secondary prevention values, as the ongoing presence of an underlying cause, such as myocardial ischemia or uncontrolled hypertension, may lead to continuing myocardial damage, thus adversely affecting disease progression and prognosis. The presence of one or more risk factors does not necessarily mean that they have caused HF or that other factors did not play any etiologic role. However, the absence of any known risk factors mandates that HF patients be referred to a cardiologist for additional investigations. As a diagnosis of fever is not sufficient for the proper management of fever, a diagnosis of HF must be accompanied by the identification and management of potential etiologic risk factors.

FLUID VOLUME ASSESSMENT IN HEART FAILURE

As already mentioned, the documentation of an elevated JVP is essential in making a clinical diagnosis of HF. An elevated JVP is also the most specific sign of fluid overload in patients with established HF. The estimation of JVP is probably the most important physical examination in HF, during both initial and subsequent visits. Yet physicians often find it challenging, and traditional textbook and bedside teaching have not been helpful. To understand the problem of underestimation of the prevalence of JVP elevation in HF,^{4,14–16} one needs to recognize the 3 myths associated with the traditional approach to JVP estimation: (1) the IJV myth, (2) the 45° myth, and (3) the 5 cm myth. As mentioned earlier, the singular use of IJV to determine JVP is likely to substantially underestimate the prevalence of JVP elevation in HF.^{4,14–16} Therefore, both IJV and EJV should be used to estimate JVP in HF, and JVP should be estimated even when it seems normal. For example, Case 3 had a JVP of 3 cm of water. At a 45° incline, there would be no IJV or EJV pulsation in his neck, and it would be correct to state that he had no JVP elevation. While that may be enough in most healthy individuals and even in those presenting with new-onset HF, it is not sufficient for patients with established HF. Therefore, EJV or IJV pulsation should be looked for at lower inclines (including the supine position if necessary) to identify low JVP. Clinicians need to remember one key point when estimating low JVP in HF. The top of the jugular venous pulsation visible in the middle of the neck in a near-supine position would likely be below the level of the sternal angle. Therefore, the “sternal angle to top of the jugular pulsation” distance would be negative, and as such, when estimating JVP, the “sternal angle to top of the jugular pulsation” distance should be subtracted from the “right atrium to sternal angle” distance.

Proper estimation of JVP requires identification of the jugular venous pulsation in the middle of the neck (Fig. 1). The examination can begin in any position, but the neck should be examined for venous pulsation in all possible positions between supine and sitting until the jugular pulsation is visualized. EJV's, like the veins on the dorsum of one's hands, are superficial and subcutaneous, and thus clearly visible without any tangential light. The position of the EJV's in the neck may be identified by lowering the head of the bed, eliciting the HJR, or exerting a gentle pressure at the root of the neck with a finger. In some older adults, EJV's may be stiff and their contours are visible without those maneuvers. For optimal visualization of EJV pulsation, the head needs to be turned slightly (10 to 20 degrees) to the opposite direction. This allows the skin folds in the neck to be straightened enough for visualization but not tight enough to obliterate it.

The IJV's, on the other hand, lie deep and their contours cannot be seen. The IJV pulsation is best visualized in the upper neck area, when the head of the patient is turned laterally (optimally between 50 to 80 degrees) to the opposite direction. The IJV pulsation can be confused with that of the carotid artery, which can be easily distinguished from the latter by several observations: (1) the jugular pulsation has double undulation (vs. the single upward stroke of carotid pulsation), (2) the jugular pulsation cannot be palpated, and (3) the jugular pulsation is responsive to gravity (body positions) and abdominal pressure. Once the top of the jugular pulsation has been identified in the middle of the neck, the vertical “sternal angle to top of the jugular pulsation” distance should be estimated in centimeters (Fig. 1; Fig. 2). Then, the estimated “right atrium to sternal angle” distance appropriate for the body position should be added (subtracted if the vertical “sternal angle to top of the jugular pulsation” distance is negative; Fig. 2). The “right atrium to sternal angle” distance has been traditionally thought to be 5 cm regardless of body position. However, estimates based on computer tomographic scans of chest suggest that the “right atrium to sternal angle” varies with the position of the body (Fig. 2).²¹ The “right atrium to sternal angle” is 5 cm only during supine position. It increases to 8 at about 30 degrees of head elevation, and to about

10 cm at 45 degrees of head elevation (Fig. 2).²¹ The “right atrium to sternal angle” distance remains about 10 cm despite further elevation, so that it would be 10 cm in the sitting position (Fig. 1).

HJR is often positive in patients with clinical HF. A positive HJR is one in which the JVP rises by 2 to 3 cm and remains elevated for about 10 seconds when a firm sustained pressure is applied to the upper to middle abdomen area. The cause of the positive HJR is poorly understood, and is believed to reflect right ventricular noncompliance. In one study, a positive HJR predicted right atrial pressure greater than 9 mm Hg, with high sensitivity (100%) and specificity (85%).²² A positive HJR is often more helpful in identifying EJVP and determining its patency than establishing fluid volume retention, as those with positive HJR usually will also have an elevated JVP. Not infrequently, HJR may be positive in HF patients who have normal or low JVP, which may indicate early fluid retention or mild residual fluid overload. Many of these patients are stable and have no bothersome dyspnea, and thus maybe be clinically insignificant.

EJECTION FRACTION IN HEART FAILURE

Estimation of LVEF by an echocardiogram is the most important test after a clinical diagnosis of HF has been made, as it has both prognostic and therapeutic implications. Patients with clinical HF have diastolic HF if they have normal or near-normal LVEF and systolic HF if they have reduced LVEF (Table 5). Although various LVEF cutoffs have been used to define systolic and diastolic HF, an LVEF of greater than 55% is often considered normal and an LVEF of less than 45% is often considered reduced. Baseline characteristics and prognosis of HF patients with LVEF between 45% and 55% are more similar to those with normal LVEF than reduced LVEF.⁴ In general, older adults with systolic HF are at higher risk of mortality and hospitalization than those with diastolic HF.²³ Despite poor outcomes, systolic HF patients are more likely to benefit from life-prolonging therapy using neurohormonal blockade. On the other hand, the benefit of these drugs in diastolic HF has not been well established.

TREATMENT OF HEART FAILURE

Treatment of HF can be summarized into two broader groups: symptom-relieving therapy and life-prolonging therapy (Table 6). Because symptoms of HF are similar in systolic and diastolic HF, therapy to relieve symptoms and prevent hospitalization because of worsening HF is also rather similar. This therapy is primarily based on judicious use of diuretics and digoxin. Most HF patients with symptoms need loop diuretics to stay euvoletic. Furosemide is the most popular loop diuretic, followed by torsemide and bumetenide. Furosemide 40 mg is equivalent to torsemide 20 mg and bumetenide 1 mg. It is preferable to use diuretics as a single morning dose to avoid sleep disturbances. Large doses of diuretics (e.g., furosemide 480 mg daily) may be given twice a day: morning and early afternoon. The long-term effects of prolonged loop diuretic therapy on outcomes in HF patients have not been studied in large RCTs. However, findings from propensity-matched studies suggest that prolonged diuretic use may be associated with increased mortality and hospitalization.^{24,25} Although these findings may represent bias by indications associated with the use of diuretics, they have also been attributed to hypokalemia and neurohormonal activation, both of which are associated with diuretic use and poor outcomes.^{26–29} Therefore, after euvoletmia has been achieved, lower doses of diuretics should be tried to maintain euvoletmia.

Although the effects of torsemide and furosemide have not been examined in large RCT, findings from laboratory studies and small clinical trials suggest that the neurohormonal activation and hypokalemia associated with torsemide may be milder and that its use may be

associated with better outcomes.^{30–32} Therefore, torsemide may be preferred over furosemide, especially when large doses are used. However, despite being generic for many decades, torsemide is more expensive than furosemide. Serum potassium should be monitored to keep between 4 and 5 mEq/L.^{26,33} Whereas potassium supplement may be used to correct hypokalemia, spironolactone may be preferable for patients with persistent hypokalemia who need chronic potassium supplement to remain normokalemic.^{34,35} Because judicious use of diuretics plays an important role in achieving euvolemia, clinicians should be familiar with the use of all 3 loop diuretics. Although not clearly understood why, when response to a particular diuretic is poor, a better response may be observed with another diuretic. Finally, there is no evidence suggesting that a large dose of a loop diuretic (e.g., furosemide 320 mg daily) is superior to a combination of a lower dose of diuretic (e.g., furosemide 160 mg) and a thiazide-like diuretic (e.g., metolazone 2.5 mg daily). However, the combination regimen has been suggested to cause more electrolyte imbalance, requiring a closer monitoring of serum electrolytes, and may also be more inconvenient for older adults.

Digoxin has been shown to reduce HF hospitalization in both systolic and diastolic HF.^{36–40} However, in diastolic HF, the use of digoxin was also associated with a significant increase in hospitalization due to unstable angina.³⁸ Because these patients were not receiving beta-blockers, it is unknown if hospitalization due to unstable angina would be lower in contemporary HF patients, many of whom receive beta-blockers. In systolic HF, when used in low doses (0.125 mg or less per day), leading to low serum digoxin concentrations (0.5–0.9 ng/ml), digoxin has also been associated with reduction in mortality.^{37,39,40} When used in low doses, there is no need to routinely check serum digoxin concentration, though it may be checked if there is clinical evidence of digoxin toxicity and if an increase in the dose of digoxin is being considered in a symptomatic patient unresponsive to lower doses. Digoxin should be prescribed to systolic HF patients with or without atrial fibrillation who continue to remain symptomatic despite therapy with ACE inhibitors or angiotensin receptor blockers (ARBs), and beta-blockers, or for those who cannot afford or tolerate these drugs.³⁷ Digoxin should also be tried before considering cardiac resynchronization therapy (CRT), which is invasive, costly, and indicated for systolic HF patients with a wide QRS complex, but the response is often unpredictable.³⁷

Life-prolonging therapy for HF is limited to the use of an ACE inhibitor or an ARB for patients who cannot tolerate an ACE inhibitor (mostly due to cough, but allergic reactions are not uncommon), and any of the 3 beta-blockers approved for use in HF (carvedilol, metoprolol succinate long-acting, and bisoprolol) in patients with systolic HF (Table 6). There is no evidence of additional mortality reduction from the addition of an ARB to systolic HF patients receiving ACE inhibitors. In addition, patients receiving both an ACE inhibitor and an ARB are more likely to experience adverse side effects such as worsening kidney function and hyperkalemia.⁴¹ There is no need to maximize the dose of ACE inhibitors before beta-blocker therapy is initiated.⁴² Because metoprolol succinate is a beta-1 selective blocker and has little effect on blood pressure, it may be preferred as the beta-blocker of initial choice over carvedilol, a nonselective beta-blocker with a potential antihypertensive effect.⁴³ However, in older adults with systolic HF and uncontrolled hypertension, switching metoprolol succinate to carvedilol should be considered before adding another antihypertensive agent. The indication of aldosterone antagonists such as spironolactone is limited to symptomatic patients with advanced systolic HF without renal insufficiency or hyperkalemia.³⁵ Eplerenone may be prescribed to HF patients who cannot tolerate spironolactone (mostly because of painful gynecomastia).^{44,45}

There is little evidence from RCT's that therapy with ACE inhibitors or ARBs or beta-blockers reduces all-cause mortality in diastolic HF.^{46–51} However, candesartan and digoxin

have been shown to reduce HF hospitalization, and may be considered in symptomatic diastolic HF patients with frequent hospitalizations as a result of worsening HF.^{38,50,52}

Device-based and surgical therapy for older adults with HF needs to be individualized based on comorbidities and functional status. CRT has been shown to reduce mortality and hospitalization in symptomatic systolic (LVEF <30%) HF patients with a wide QRS complex (>120 ms).⁵³ The advantage of CRT devices in combination with defibrillator function versus pacemaker function in older adults with HF remains unclear. Considering the risk of sudden cardiac death in HF, many prefer an implantable cardioverter with defibrillator (ICD) function over one with pacemaker function. However, the effectiveness of ICD's in older adults with multiple morbidities has not been well established.^{54–56}

Finally, older adults with end-stage HF should be considered for hospice and palliative care referral. Because most acutely decompensated HF patients become asymptomatic when euvolemic and many apparently stable HF patients may die of sudden cardiac death, predicting 6-month life expectancy may be difficult in older adults with end-stage HF. However, clinicians familiar with HF patients may use a subjective criterion of asking themselves if they would be surprised if their HF patients died within the next 6 months. If the answer is no, such patients could be considered for possible hospice and palliative care referrals. The issue of deactivation of ICD and other cardiac devices should be discussed with patients and family members of older adults with HF being considered for hospice and end-of-life care.⁵⁷

SUMMARY

Most of the HF patients are older adults, and HF is the leading cause of hospitalization among older adults. The assessment and management of HF in older adults can be challenging. However, it can be structured and simplified by using the mnemonic DEFEAT-HF: Diagnosis (clinical, and must not be ruled out based on normal LVEF), Etiology (CAD and hypertension being the most common), Fluid volume (must be assessed by estimating JVP using IJV or EJV), Ejection fraction (LVEF has both prognostic and therapeutic implications), And Treatment of Heart Failure (symptom-relieving therapy using diuretic and digoxin for systolic and diastolic HF, and life-prolonging therapy using neurohormonal blockade in systolic HF). Device and surgical therapy must be individualized, and hospice and palliative care should be considered for those with terminal HF.

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Figure 1.

The jugular venous pressure (JVP) is the vertical height of the top of the jugular venous pulsation from the right atrium (RA), expressed in centimeters of water. To estimate JVP, one needs to use the sternal angle (SA) as a landmark. After first estimating the "SA to the top of the jugular venous pulsation" distance (7 cm in this case), one needs to add this number to the "RA to SA" distance (10 cm in this case). Of note, the top of the jugular venous pulsation would be lower than the SA when the JVP is low, in which case the "SA to the top of the jugular pulsation" distance needs to be subtracted from the "RA to SA" distances (see Fig. 2), which would vary depending on the body position. It is helpful to remember 3 "RA to SA" distances: about 5 cm in supine position, about 8 cm at a 30 degree incline, and about 10 cm at 45 degree or higher inclines.

Figure 2.

To estimate the jugular venous pressure (JVP), the jugular venous pulsation must be displayed in the middle of the neck. This requires positioning patients at various inclines, depending on the JVP level. Also, the "right atrium to sternal angle" distance varies, depending on the body position. Of note, when JVP is low (left panel), the "sternal angle to top of jugular pulsation" distance is "negative" and must be *subtracted* from the "right atrium to sternal angle" distance to obtain the estimated JVP in cm of water.

Adopted from Ahmed A, Jones L, Hays CI. DEFEAT heart failure: assessment and management of heart failure in nursing homes made easy. *J Am Med Dir Assoc.* 2008;9:383-389 (page 386).

Table 1

DEFEAT-HF: A simple 5-step protocol for the assessment and management of chronic heart failure in older adults

D	Diagnosis	HF is a clinical diagnosis and cannot be confirmed or ruled out by any single test or procedure. Therefore, a clinical diagnosis of HF must be made. LVEF should not be used to diagnose HF as over half of all older adults with HF have diastolic HF or HF with normal or near-normal LVEF
E	Etiology	HF is a clinical syndrome, and not a disease, thus an underlying cause must be sought and identified. If no underlying cause can be identified, patients should be referred to a cardiologist or HF expert for further evaluation. HF patients with ongoing etiologic insults such as myocardial ischemia should also be referred for appropriate management as it may adversely affect disease progression and prognosis
F	Fluid volume	Nearly all newly diagnosed HF patients present with fluid retention, which is also a recurring problem for those with chronic HF, at times despite proper management. Careful estimation of JVP using IJV and EJVP is the most reliable means to assess fluid volume status. A determination of "no fluid retention" is not enough as many HF patients may suffer from hypovolemia, often due to over-diuresis. Therefore, JVP should be estimated in cm of water
E	Ejection fraction	LVEF, estimated preferably by an echocardiogram, is the single most important test after a clinical diagnosis of HF has been made. It has both prognostic and therapeutic implications. Patients with systolic HF or clinical HF with low LVEF often have poorer prognosis than those with diastolic HF who in turn have poorer prognosis than those without HF. While therapy with neurohormonal blockade has been shown to improve the poor outcomes in systolic HF, their effectiveness in diastolic HF has not been well established
A	And	
T	Therapy	HF therapy can be broadly divided into those directed at relieving symptoms and those directed at improving outcomes. Because HF symptoms are indistinguishable between systolic and diastolic HF, symptom-relieving therapy is also similar for all HF patients. However, life-prolonging therapy is often restricted to those with systolic HF. Although most guideline recommendations for the management of systolic HF are based on evidence from younger systolic HF patients, they can be reasonably individualized and applied to the care of older systolic HF patients.

Adopted from Ahmed A "DEFEAT Heart Failure: Clinical Manifestations, Diagnostic Assessment, and Etiology of Geriatric Heart Failure". Heart Failure Clin 2007;4:389–402 (page 390); with permission

Table 2

Diagnosis of heart failure in older adults

	Cases					Comments
	1	2	3	4	5	
Dyspnea on exertion	Yes	Yes	Yes	Yes	No	DOE, although nearly always present, is a very non-specific symptom, and by itself, without other associated symptoms and signs, is not often very helpful in making a diagnosis of HF.
Orthopnea	No	Yes	Yes	Yes	No	HF patients often sleep on multiple pillows or in a recliner, or use blocks to raise the head of their bed to avoid orthopnea, and may report negatively when asked.
Paroxysmal nocturnal dyspnea	No	Yes	No	No	No	PND is uncommon as chronic HF patients often sleep with their head elevated to avoid orthopnea, which also reduces the occurrence of PND.
Dyspnea at rest	No	No	No	No	No	Constitutes NYHA Class IV or most severe symptom. Rare.
Fatigue on exertion	Yes	Yes	Yes	Yes	No	Exertional fatigue often is more common in older adults with HF than exertional dyspnea. As opposed to exertional dyspnea, which tends to occur when things are done in a hurry, like rushing to pick up the ringing phone at the end of the room, exertional fatigue often occurs when a patient does things, like walking two city blocks, at his/her "own pace". For example, a HF patient may report fatigue without shortness of breath after walking a block at his/her own pace.
Chest pain	No	No	No	No	No	Not a common presenting or associated symptom in geriatric HF patients.
Cough	No	No	No	Yes	No	Rare. In some older adults with HF, cough may precede dyspnea, while in others it may accompany or follow dyspnea.
Swelling of foot/leg	Yes	Yes	Yes	Yes	No	Extremely common. It is more useful in the diagnosis process if the leg swelling is bilateral and the onset of leg swelling and dyspnea on exertion are contemporary. However, leg swelling often may not correlate with dyspnea as venous insufficiency is common in older adults, which may be unilateral.
Gastrointestinal symptoms	No	Yes	No	No	No	Usually uncommon, except in those with prolonged fluid retention leading to congestive hepatopathy. A more prolonged and severe fluid retention may also result in congestive gastropathy, which may cause nausea, vomiting, anorexia, and poor absorption and response to drugs, such as diuretics.
Chronic obstructive pulmonary disease (COPD)	No	No	No	Yes ?	No	The diagnosis of HF may be difficult in the presence of COPD as dyspnea is rather similar in both HF and COPD. Patients with COPD may also report PND, in which cough may precede dyspnea. Often HF and COPD may coexist, which may further complicate the diagnosis process. An elevated JVP is probably the most important physical finding in establishing a diagnosis of HF in this context. A normal BNP, especially in the presence of a normal JVP or when JVP cannot be estimated, would likely rule out HF.
Deconditioning	No	Yes	No	No	No	Exertional dyspnea and fatigue associated with deconditioning may be distinguished by the lack of associated orthopnea, PND, leg edema, or other evidence of fluid retention.
Depression	No	Yes	No	No	Yes	Somatization is common in older adults with depression and symptoms are often not physiologically plausible. For example, dyspnea or chest pain at rest that is not worsened by physical activities. Also, lack of associated symptoms and signs of fluid retention may help distinguish somatization from HF.
HF risk factors present	Yes	Yes	Yes	Yes	Yes	When traditional risk factors such as hypertension and coronary artery disease are absent, referral to cardiologists should be considered for confirmation of diagnosis and identification of an underlying etiologic risk factor.
Jugular venous pressure elevated	No	Yes	No	Yes	No	Almost always present during initial presentation and acute exacerbation of HF, but chronic stable patients may have normal JVP, and may even be low in over-diuresed patients.

	Cases					Comments
	1	2	3	4	5	
Jugular venous waveforms	?	?	?	?	?	Not needed to estimate JVP or make a diagnosis of HF. However, familiarity with the double undulation of the JVP pulsation may help distinguish it from carotid pulsation.
Third heart sound	No	Yes	No	Yes	No	Often present in acute HF but its presence is not necessary to establish a diagnosis of HF. An S3 can also be present in a chronic HF patient who is euvolemic.
Pulmonary crackles and wheezing	No	No	No	Yes	No	Rare in chronic HF due to the hyper-efficient pulmonary lymphatic drainage system. However, a more rapid fluid buildup may overwhelm the system and result in pulmonary edema.
Abdominal tenderness	No	Yes	No	No	No	Right upper quadrant tenderness may result from congestive hepatopathy secondary to prolonged fluid retention.
Hepatjugular reflux	No	Yes	No	No	No	It also helps in identifying the position and patency of external jugular veins in neck. When present in those with normal JVP, it may indicate early fluid retention.
Lower extremity edema	Yes	Yes	No	Yes	No	Pitting edema in those with normal JVP may indicate chronic venous insufficiency, often unilateral and may be accompanied by skin discoloration and stasis dermatitis. Prolonged venous insufficiency may lead to secondary lymphedema and non-pitting edema.
Cardiomegaly	No	Yes	Yes	Yes	No	Cardiomegaly by chest x-ray may help support a clinical diagnosis of HF.
Pulmonary venous congestion	No	Yes	No	Yes	No	Uncommon in chronic HF, but when present in x-ray may support diagnosis.
Pulmonary edema	No	Yes	No	No	No	Rare in chronic HF. X-ray evidence of pulmonary edema suggests acute fluid buildup or severe HF.
Pleural effusion	No	No	No	No	No	Not uncommon, usually bilateral, often more severe on right side, rarely massive.
B-type natriuretic peptides (BNP)	Not done	Not done	400	<50	No	Elevated BNP and the N-terminal portion of BNP(NT-pro-BNP) levels may support the diagnosis of HF. HF is unlikely if BNP <100 pg/mL and NT pro-BNP <300 pg/mL. However, their role in older adults with HF has not been well studied.
Left ventricular ejection fraction	35%	55%	25%	55%	55%	A low LVEF may support a diagnosis of HF when clinical presentation is atypical or insufficient. However, a normal LVEF <i>should not</i> be used to rule out a diagnosis of HF.
Response to diuretics	N/A	Yes	Yes	Yes	N/A	A therapeutic response to diuretic may help confirm the diagnosis of HF when clinical presentation is atypical or insufficient.

Case 1: Had no clinical evidence of HF, and the diagnosis was facilitated by his low LVEF

Case 2: Had a classic text book presentation of HF and the diagnosis could not be confounded by her normal LVEF

Case 3: Although he was over-diuresed and hypovolemic in his initial presentation, he had historical evidence of clinical HF and a low LVEF

Case 4: Despite clear evidence to clinical HF, a diagnosis of HF may have been previously confounded by his normal LVEF and BNP

Case 5: Dyspnea at rest but not during exertion suggested somatization. Therapy with antidepressant completely resolved her symptoms

Table 3

Etiology of heart failure in older adults

	Cases					Comments
	1	2	3	4	5	
Coronary artery disease	Yes	No	No	Yes	No	CAD is a rather strong risk factor for HF, and the risk is further increased for those with a prior AMI. The presence or absence of CAD can often be established during history and physical examination.
Hypertension	Yes	Yes	Yes	Yes	Yes	Hypertension is probably the most common risk factor for HF among older adults, especially for diastolic HF. The presence or absence can be established during history and physical examination. Persons with hypertension have a lower relative risk for HF than those with CAD, but hypertension contributes to more cases of new HF as hypertension is more common in older adults.
Diabetes mellitus	No	No	Yes	No	No	The risk of HF among those with diabetes is nearly similar to that among those with CAD.
Valvular heart disease	No	No	No	No	No	May coexist with other risk factors. Diagnosed by echocardiography.
Cardiomyopathy	No	No	No	No	No	While dilated and hypertrophic cardiomyopathies are less common in older adults, restrictive cardiomyopathies may be more common in older adults.
Chronic kidney disease	No	Yes	No	Yes	No	Has long been known as a risk factor, the independent risk may not increase until the chronic kidney disease is advanced.
Smoking	No	No	Yes	No	No	Heavy smokers may have increased risk despite many years of cessation.
Atrial fibrillation	No	Yes	No	Yes	No	Uncontrolled ventricular rate is probably more important than the presence of atrial fibrillation itself.
Hyperthyroidism	No	No	No	No	No	Rare.
Peripheral arterial disease	No	No	No	No	No	Probably as a marker of overall atherosclerosis.
Anemia	No	No	No	No	No	Rarely a risk factor, and often not until anemia is very severe.
Left ventricular hypertrophy	No	Yes	No	No	No	A very strong risk factor.
Asymptomatic left ventricular systolic dysfunction	No	No	No	No	No	Also, a very strong risk factor. The low LVEF and slowly progressive symptoms of Case 1 suggest that he may have had asymptomatic left ventricular systolic dysfunction.

Table 4

Fluid volume status evaluation in older adults with heart failure

	Cases					Comments
	1	2	3	4	5	
Internal jugular vein	No	No	No	Yes	No	IJV lies behind the sternocleidomastoid muscle for most of its course in the neck and thus not visible. The IJV pulsation is also often not seen over this area. However, the pulsation can be seen in the triangular area at the base of the neck in between the two heads of the muscle where the IJV is subcutaneous, and in the upper part of the neck where the IJV emerges from under the sternocleidomastoid muscles (as the muscle ascends laterally to its insertion on the mastoid). In some chronic HF patients, the IJV pulsation may not be seen.
External jugular vein	Yes	Yes	Yes	Yes	Yes	The contour of the EJV can be seen in the neck like the veins on the dorsum of the hand. If the vein is patent, the pulsation is also clearly visible. When EJV is visible on either side of the neck and have different heights, the one with the higher height should be used. When both IJV and EJV pulsations are visible, the JVP based on the IJV should be used. At times, the top of EJV pulsation may be lower than the top of the IJV pulsation, likely due to narrow lumen of the former.
Hepatjugular reflux	No	Yes	No	Yes	No	Often present in patients with elevated JVP, but not necessary for diagnosis.
Degrees of incline of the head of the bed at which top of the jugular pulsation visible in neck	30	80	10	45	30	The incline of the head of the bed or examination table is irrelevant as the goal is to make the top of the jugular venous pulsation visible in the middle of the neck. The top of jugular pulsation for a patient with low JVP may only be visible in a supine position and that for a patient with high JVP in a sitting position. However, it is important to make a note of the degree of the head elevation so that the appropriate "right atrium to the sternal angle" distance can be added to the "sternal angle-to-top of the jugular pulsation" distance.
Estimated distance in centimeter from sternal angle to top of jugular pulsation	-2	8	-2	6	-2	When the top of the jugular pulsation is below the sternal angle, then one need to subtract the "sternal angle-to-top of the jugular pulsation" (a negative value) from the "right atrium to sternal angle" distance to obtain the JVP. Of note, the "sternal angle-to-top of the jugular pulsation" distance is the vertical distance, not horizontal. This is especially important to remember in patients with low JVP who often would need to be in a supine or near-supine position.
Estimated distance in centimeter from right atrium to sternal angle	8	10	5	10	8	Unlike what is taught in many old text books, the distance is not 5 cm regardless of body position. Findings based on CT scan images of chest suggest that it varies by the body position (head elevation) and that it is 5 cm only in supine position. At a 45 degrees incline it is 10 cm and at about 30 degrees, it is 8 cm. The distance may be higher for individuals with large chest. For example, at a 45 degrees incline, the "right atrium to sternal angle" distance in a male patient with an average anteroposterior chest diameter of 25 cm (about 10 inches) would be 10 cm. However, for a patient with an anteroposterior chest diameter of 35 cm (about 14 inches), it would be about 12 cm (an increase by 2 cm at a rate of 0.2 cm per every 1 cm increase in anteroposterior chest diameter).
Estimated jugular venous pressure	6	18	3	16	6	A JVP of 6-8 cm of water may be considered normal in HF. Some HF patients may tolerate a JVP of 8-10 cm of water. Most HF patients with JVP above 10 cm of water may be symptomatic and achieving euvolemia in these patients would improve breathing. However, a JVP of 4-5 cm of water in a symptomatic patient may indicate over-diuresis and hypovolemia, and should be avoided.
How jugular venous pressure was estimated	8-2 = 6	10+8 = 18	5-2 = 3	10+6 = 16	8-2 = 6	Two key points to remember: (1) the "sternal angle-to-top of the jugular pulsation" distance is vertical and varies with the position of the patient, (2) the "sternal angle-to-top of the jugular pulsation" distance must be subtracted from the "right atrium to the sternal angle" distance, when the JVP is low and the top of jugular pulsation is below the sternal angle.

Table 5

A simplified illustration demonstrating how patients with diastolic heart failure may have similar symptoms as those with systolic heart failure, despite normal left ventricular ejection fraction

	End-diastolic volume (ml)	ventricular ejection fraction (%)	ventricular stroke volume (ml)	Heart rate (beats per minute)	Cardiac output (liters per minute)	Symptoms
Normal	150	55%	75	72	5.4	None
Systolic heart failure	200	25%	50	72	3.6	Dyspnea, fatigue, edema
Diastolic heart failure	100	55%	50	72	3.6	Dyspnea, fatigue, edema

Adopted from Ahmed A "DEFEAT Heart Failure: Clinical Manifestations, Diagnostic Assessment, and Etiology of Geriatric Heart Failure". Heart Failure Clin. 2007; 4: 389-402

Table 6

Pharmacotherapy in older adults with heart failure

Symptom-relieving therapy (applicable to both systolic and diastolic HF)	
Diuretics	After euvoolemia has been achieved, lower doses of diuretics should be tried to maintain euvoolemia. Serum potassium should be periodically monitored to keep between 4 and 5 mEq/L
Digoxin	Applicable to both systolic and diastolic HF. The main benefit of digoxin is in the reduction of hospitalization due to worsening HF. However, when used in low doses (0.125 mg or less per day), digoxin may also lower mortality, which is especially important for older adults with systolic HF who cannot afford or tolerate ACE inhibitors or beta-blockers
Life prolonging therapy (applicable to systolic HF only) *	
ACE inhibitors or ARBs	ACE inhibitors are the drugs of first choice. ARBs should only be used in those who cannot tolerate ACE inhibitors, often due to cough.
Beta-blockers	Unlike ACE inhibitors, there is no class effect, and one of the three approved beta-blockers (carvedilol, metoprolol succinate and bisoprolol) should be used
Aldosterone antagonist	Limited indication. Avoid use in patients with renal insufficiency and monitor serum potassium to avoid hyperkalemia

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker.

* Although many life-prolonging drugs also reduce the risk of hospitalization, most HF patients require diuretics to remain euvolemic.