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Diagnosis and Management of Heart Failure in Older Adults

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HEART FAILURE: A GERIATRIC SYNDROME

Heart failure (HF) is a geriatric syndrome. A disease generally has a known etiology, a known pathogenesis, and a known but variable presentation. A syndrome, on the other hand, is a set of symptoms and signs for which either the etiologic factor or the pathogenesis or both maybe unknown.[1] A geriatric syndrome, like a medical syndrome, is also characterized by a defined set of symptoms and signs but often the underlying etiologic factors are multiple and the pathogenesis may involve multiple interacting pathways (Fig. 1).[1]

CLINICAL PRESENTATION OF HEART FAILURE IN OLDER ADULTS

Most heart failure (HF) patients are older adults, who also suffer from multiple comorbidities and polypharmacy.[2] The management of a 78-year-old woman with HF and left ventricular ejection fraction (LVEF) greater than 55%, with hypertension, atrial fibrillation, diabetes, arthritis, chronic kidney disease, and depression, and who is taking multiple medications is more difficult than that of a 40-year-old young man with ejection fraction (EF) 25%, ischemic heart disease, and no other comorbidity. Yet, older HF patients have often been excluded from major HF trials. Thus, there is little evidence to guide management of HF in older adults.[3,4] That challenge is further compounded by the difficulty in the diagnosis and assessment of HF in older adults.[5] Older adults in general are a heterogeneous group, and as such, geriatric HF is characterized by a wide range of phenotypic heterogeneity, as illustrated in the case scenarios presented (Table 1). An initial

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assessment of HF in older adults involves a clinical diagnosis of HF, an evaluation of potential etiologic factors, an estimation of jugular venous pressure (JVP); and an evaluation of the left ventricular ejection fraction (LVEF) to guide therapy. These steps may be memorized with the mnemonic DEFEAT-HF: diagnosis, etiology, fluid volume, EF, and therapy for heart failure (Table 2).

DEFINITION OF HEART FAILURE

HF is a clinical syndrome that is characterized by symptoms that are manifestations of structural or functional impairment of ventricular filling or ejection of blood (see Table 1).[6] The cardinal manifestations of HF are dyspnea and fatigue, which may limit exercise tolerance, and fluid retention, which may lead to peripheral and pulmonary edema. Because HF is a not a primary disease, a full definition of HF needs to accompany a statement of underlying etiologic factors.

STAGES OF HEART FAILURE

Because HF is a progressive condition, clinical manifestations of HF vary depending on the stages in its natural history. Patients in stage A and stage B are asymptomatic and truly do not have clinical HF. Patients with stage A have risk factors for HF such as hypertension and coronary artery disease (CAD) but no structural myocardial disorder and no clinical HF. When stage A patients develop structural myocardial damage, such as left ventricular hypertrophy (LVH) or asymptomatic left ventricular systolic dysfunction but no clinical HF, they are referred to as stage B. Most clinical HF patients belong to stage C, with current or past symptoms. Stage D represents endstage and refractory HF and patients may require special therapy, such as durable circulatory support, cardiac transplants, or palliative care.

DESCRIPTIVE CLASSIFICATIONS OF HEART FAILURE

The most clinically relevant descriptive classification of clinical HF is based on LVEF: HF with reduced EF (HFrEF; previously known as systolic HF) and HF with preserved EF (HFpEF; previously known as diastolic HF). This classification has prognostic and therapeutic implications (see later discussion). Determination of LVEF is the first crucial step in the assessment of patients diagnosed with HF and is considered a measure of quality of care.[7,8] Other descriptive classifications of HF (see later discussion) are less clinically relevant.

Left-Sided Versus Right-Sided Heart Failure

Left-sided HF occurs when HF predominantly affects the left ventricle. Most early clinical HF patients have left-sided HF. Pure left-sided HF may result in either pulmonary congestion or systemic hypoperfusion, or both, and have associated symptoms, such as dyspnea, cough, wheezing, fatigue, hypotension, tachycardia, confusion, syncope, delirium, and oliguria, pulmonary rales, and left-sided third heart sound (S3). As HF progresses, left-sided HF eventually leads to right-sided HF.[9] The presence of low right ventricular EF is associated with poor outcomes in patients with HFrEF.[10,11]

Right-sided HF occurs when HF predominantly affects the right ventricle. Common symptoms and signs of right-sided HF are often due to systemic congestion, resulting in dyspnea, fatigue, leg swelling, nausea, vomiting, epigastric and upper abdominal pain, elevated JVP, hepatojugular reflux (HJR), hepatomegaly, right-sided S3, prominent pulmonic component of the second heart sound (S2), and dependent edema. Little is known about the parameters of right ventricular function and dysfunction, and the cellular and molecular basis of right-sided HF.[12] Common causes of right HF include pulmonary conditions (cor pulmonale) and left HF.

Thus, most advanced HF patients have both left-sided and right-sided HF, as noted in case 2. However, symptoms and signs related to biventricular HF may also been seen in most patients with early HF who often present with dyspnea on exertion (DOE) and leg edema (case 1).

Backward Versus Forward Heart Failure

The concept of backward failure was first proposed by James Hope in 1832. He suggested that congestion in HF was due to backward pressure in the venous and capillary system as a result of the failing heart's inability to pump forward.[13] This notion is supported in that most left HF are associated with some degree of right HF.[9,14] However, it was later demonstrated that congestion in HF often precedes increase in pressure in the venous and capillary systems and is due to decreased renal blood flow directly associated with forward failure.[15,16] Diminished renal blood flow has later been shown to be associated with the activation of the renin-angiotensin-aldosterone system in HF.[17] Most HF patients have clinical manifestations of both backward and forward HF (cases 1–4). HF may not be clinically distinguished as forward or backward, and such distinctions probably have no diagnostic, therapeutic or prognostic implications.

High-Output Versus Low-Output Heart Failure

Most older HF patients have low-output HF (cases 1–4). High-output HF, characterized by high cardiac output, although rare in older adults, may be associated with hyperdynamic conditions such as severe anemia, thyrotoxicosis, and arteriovenous fistula, including those used for hemodialysis.[18–20] Although anemia may cause exacerbation of HF symptoms, it rarely causes high-output HF in the absence of other cardiac disease and/or severe anemia (hemoglobin <5 gm/dl).[14,21] Thyrotoxicosis alone also rarely causes high-output HF. Clinical features of high-output and low-output HF may be indistinguishable. However, high-output HF patients may have warm extremities. Assessment of all new HF patients should include laboratory tests for anemia, kidney function, and thyroid function.[21–23]

Acute Versus Chronic Heart Failure

The concept of acute and chronic HF is used in 2 different contexts: severity (mild to moderate vs severe) and onset (sudden vs gradual) of symptoms. Most chronic HF patients undergo acute exacerbations of symptoms from time to time (cases 2 and 4). More than two-thirds of all hospitalized HF patients have known chronic HF.[24] Decompensation of chronic HF may be due to noncompliance with drugs, salt or fluid, acute myocardial ischemia, severe hypertension, or natural disease progression. In many of these patients, symptoms are

severe and require ED visit or hospitalization for acute management. HF is associated with over 1 million hospitalizations in the United States and is the main reason for hospitalization for persons 65 years and older.[25,26] This is despite that fact that older HF patients often attribute their HF symptoms to aging and thus delay care, even when symptoms are severe (case 2). Clinical manifestations of ambulatory chronic HF and hospitalized acute HF are generally similar but may be more severe in the latter group.

Clinical manifestations of HF may be sudden, as after a large acute myocardial infarction with or without valve damage, typically leading to HFrEF, and in severe systolic hypertension with flash pulmonary edema, often leading to HFpEF (case 3).[27,28] HF may also present acutely with syncope (case 4). Clinical manifestations of HF may be gradual in the presence of chronic myocardial ischemia and less severe hypertension. Acute exacerbations due to noncompliance are also generally gradual in onset. Both sudden and gradual onset of symptoms may occur in the setting of incident or prevalent HF.

DIAGNOSTIC ASSESSMENT OF HEART FAILURE IN OLDER ADULTS

The diagnostic assessment of HF in older adults should begin with a clinical diagnosis of HF, followed by the establishment of potential underlying causes of HF. Evaluation of JVP is an essential part of the diagnostic process. JVP should also be evaluated during subsequent visits to assess HF control status. Once a clinical diagnosis is made, EF should be measured using an echocardiogram. These key steps may be memorized by the mnemonic DEFEAT: diagnosis, etiology, fluid volume, EF, and treatment.

CLINICAL DIAGNOSIS OF HEART FAILURE

HF is a clinical diagnosis and a diagnosis of HF should be established preferably before an echocardiogram is ordered. This is especially important in patients with limited (case 1) or atypical (case 4) clinical manifestations of HF. In these patients, a normal LVEF may bias diagnostic assessment. For example, case 1 only had DOE and edema, both nonspecific symptoms, and case 4 had syncopal episode, which is an atypical HF symptom (Table 3). If a diagnosis of HF is not already made, a normal LVEF may increase the risk of a false-negative diagnosis, leading to worsening of symptoms, possible emergency room visits or hospitalizations, and delays in diagnosis and therapy. However, when a classic constellation of symptoms and signs of HF is present, and a clinical diagnosis of HF can be established without difficulty (cases 2 and 3), a normal LVEF does not make the clinician question the diagnosis but instead helps classify HF as HFpEF.

Functional, physiologic, and psychological heterogeneity of older adults should be taken into consideration when evaluating clinical manifestations in the diagnostic assessment of HF. DOE is a nonspecific symptom. However, that was the basis of a presumptive clinical diagnosis of HF for case 1. He was a very active man who refused to restrict his activities due to his DOE, which could not be explained otherwise by another illness. In addition, his DOE was accompanied by new-onset leg edema and he had several risk factors for HF. All these pointed toward a clinical diagnosis of HF. This would be difficult if he was obese, deconditioned, or had chronic obstructive pulmonary disease (COPD) (see Table 3). Case 5

had dyspnea at rest but no DOE, which is almost always due to nonorganic causes. Further questioning also revealed that about a year ago she lost her husband of many years and moved to a new home close to her children. She was diagnosed with depression and therapy with antidepressant completely resolved her symptoms (see Table 3).

In case 4, a diagnosis of HF was almost missed due to his atypical presentation (syncope) and would probably have been delayed if he had normal LVEF. Brain natriuretic peptide (BNP) may be useful when a clinical diagnosis of HF is uncertain, especially when other competing causes of dyspnea, such as COPD or obesity, exist. However, too much reliance on BNP should be discouraged (see Table 3).[29] As illustrated in case 4, after his furosemide was increased, he became euvolemic and asymptomatic. However, his BNP remained relatively high and he was continued on the same dose of furosemide. Subsequent overdiuresis led to hypovolemia, hypoperfusion, and recurrence of symptoms. After his furosemide dose was decreased, he gained about 10 pounds over the next week, and his symptoms improved. Biomarkers maybe useful in the diagnosis of HF when the clinical presentation is complicated or less clear. N-terminal probrain natriuretic peptide (NT-pro-BNP) is elevated in most patients with HF. However, the cutoff values may differ among older HF patients and tend to be higher with age and renal dysfunction, which is common in the older HF patient. Biomarkers may be used at the time of diagnosis for determination of prognosis and to guide therapy. However, biomarker-guided therapy for HF has not been shown to be associated with better clinical outcomes or quality of life when compared with therapy guided by clinical symptoms and signs of HF (Table 4).[30]

DETERMINATION OF ETIOLOGIC FACTORS OF HEART FAILURE

HF is multifactorial in older patients. Aging results in biochemical and structural changes in the myocardium and predisposes to systolic, as well as diastolic, impairment in the older patient. An etiologic factor for HF must be established for all HF patients. HF in older adults may be associated with multiple etiologic factors (Fig. 1). Often, the historic role of an etiologic factor may not be ascertained but the presence of a risk factor as comorbidity suggests a potential etiologic role. When no etiologic factors can be found, primary care physicians should consider referring new geriatric HF patients to cardiologists for evaluation of underlying etiologic factors, such as myocardial ischemia and familial and/or infiltrative cardiomyopathy, which if present may cause continued myocardial damage. For example, case 1 underwent a nuclear stress test that showed mild myocardial ischemia, which possibly played an etiologic role, along with hypertension, in his HF, and was referred for cardiology consultation. Case 4 had a long history of hypertension and diabetes, 2 known predictors of HF in older adults (see Table 3).[31] He had a normal coronary angiogram. However, microvascular dysfunction, in the absence of epicardial CAD, has also been shown to cause HF.[32,33]

Hypertension and CAD are the 2 most common causes of HF in all ages, including older adults.[31,34] In the Cardiovascular Health Study, 5888 community-dwelling older adults (65 years and older, mean age 73 years) without HF at baseline were followed for a median of 5.5 years and 597 developed new-onset HF. Individuals with CAD or hypertension had 87% and 36%, respectively, higher risk of incident HF. However, due to high prevalence

of hypertension (41% vs 17% for CAD), the population-attributable risk for CAD and hypertension were similar (both 13%). Thus, if CAD and hypertension were removed as risk factors, each would prevent HF in about 13% of the population. Relative risks of other risk factors were: diabetes (78%), serum creatinine 1.4 mg/dL or more (81%), LVH by electrocardiogram (129%), low LVEF (180%), and atrial fibrillation (106%).[31] However, because the prevalence of these conditions was low, their population-attributable risks were also low (8% for diabetes and 2% for atrial fibrillation, and others in between).

Presence of these risk factors may often be determined from history and other tests. It is important to identify the presence of these comorbidities, whether they are causally associated with HF or not. The presence of most of these comorbidities is associated with poor outcomes in HF and thus should be managed according to established guidelines. With the exception of CAD, primary care physicians should be able to identify and manage most of these comorbidities. However, because many older adults might have silent ischemia, which may cause further myocardial damage and disease progression, all HF patients with CAD should be referred to cardiologists for appropriate assessment and treatment of ischemic heart disease.

DETERMINATION OF FLUID VOLUME STATUS

JVP is expected to be elevated in patients presenting with HF for the first time and a normal JVP rules out clinical HF. JVP may be low in a patient with chronic HF and may be a marker of volume depletion and overdiuresis. Thus, fluid volume needs to be routinely assessed in all HF patients to achieve an euvolemic state to reduce symptoms and hospital admission and readmission. Careful estimation of JVP may allow accurate assessment of fluid balance in almost all older patients with HF (Figs. 2 and 3, Table 5). Ideally, JVP should be estimated using the internal jugular vein (IJV). The IJV, which lies deep in the neck behind the sternocleidomastoid muscle, is not visible in the neck and its pulsation is transmitted to the surface through the neck muscles. Thus, the transmitted IJV pulsation may be confused with the transmitted carotid pulsation. Generally speaking, unlike carotid pulsation, the low-pressure jugular pulsation cannot be palpated. In addition, jugular pulsation is distinguished from the carotid pulsation by its double waveforms and its response to respiration and HJR maneuver. IJV pulsation is better seen in the upper medial part of the neck when the head is turned to the opposite side. However, IJV pulsation may not be easily visible in patients with chronic HF. The external jugular vein (EJV) is superficial and its contour is visible like that of the veins on the dorsum of the hand. EJV pulsation is more visible than the IJV pulsation, and maybe reliably used to estimate JVP.[35] EJV pulsation is best seen with the head in the neutral position because when head is turned to the other side the taut neck skin may obliterate the superficial EJV, making the pulsation disappear. Because EJV is superficial, it may be blocked by internally (thrombosed valves) or externally (subcutaneous scarring). Thus, a distended EJV without a visible pulsation is useless for the estimation of JVP. When both IJV and EJV pulsations are visible, IJV should be used to estimate JVP. When EJV on both sides of the neck have different heights, the lower should be used.

Proper estimation of JVP requires both training and practice. The textbook rule that the head of the bed or examination table must be at 30° or 45° of incline has no relevance to the proper estimation of JVP. The key in JVP estimation is to make the top of the EJV or IJV pulsation visible in the middle of the neck so that its vertical distance from right atrium (RA) can be estimated. Because the RA to top of jugular pulsation vertical distance cannot be directly measured, the sternal angle (SA) is used as a landmark to measure SA to top of jugular pulsation vertical distance (see Fig. 2). The SA to top of jugular pulsation distance is then added to the RA to SA position-specific distance to obtain the estimated JVP (Fig. 2). When the JVP is low, the top of the jugular pulsation in neck is at a lower level than SA, and as such the SA to top of jugular pulsation distance is negative (ie, less than the RA to SA distance), and should be subtracted from the RA to SA position-specific distance to obtain the estimated JVP.

The process of estimating the vertical distance from the SA to top of IJV or EJV pulsation is rather simple and requires careful adjustment of the incline of examination table or bed so that the top of the pulsation is visible in the middle of the neck (see Fig. 3). For example, the top of the jugular pulsation for a patient with low JVP is only visible in a supine position (0° incline; see Fig. 3). In contrast, the top of the jugular pulsation for a patient with very high JVP is only visible in a sitting position (90° incline; see Fig. 3 and Table 6). It is important to remember that the SA to top of IJV or EJV pulsation distance is vertical. Thus, in patients with low JVP when the patient is in a supine position, the SA to top of IJV or EJV pulsation distance is generally negative because the anterior chest wall is at a higher level than the neck. As such, the top of IJV or EJV pulsation is vertically below the imaginary horizontal line passing through the SA. This is important because when the JVP is low, the negative SA to top of jugular pulsation distance should be subtracted from the RA to SA distance to obtain the estimated JVP. The textbook assumption of a 5 cm RA to SA distance in all positions may underestimate the JVP. Estimates based on computerized tomography scans of chest suggest that the RA to SA distance is about 5 cm in supine position, 8 cm at 30°, and 10 cm at 45° or higher inclines.[36] Thus, the RA to SA to be used to estimate JVP needs to be position-specific (see Fig. 3).

Recent weight may also be used to assess fluid volume status. Weight gain or loss in the range of 2 to 3 pounds in 2 to 3 days is almost always due to fluid overload or diuresis. If educated and encouraged to do so, most patients who have HF will monitor and report daily weight. Assessment, achievement, and maintenance of euvolemia ensure proper physical functioning, which is particularly important in older adults because they are likely to decondition quickly. Physical activity and exercise reduce deconditioning, improve quality of life, and also may reduce mortality in HF.[37–39] If not properly assessed by clinicians, however, older patients who have HF may not report DOE and other exertional symptoms. Instead, they might restrict their activities in response to those symptoms and become deconditioned.

DETERMINATION OF LEFT VENTRICULAR EJECTION FRACTION

Once a diagnosis of HF is established, every patient who has HF should have an echocardiographic determination of LVEF. In patients who have HFrEF, there is no need to

repeat the procedure unless indicated by major changes in clinical conditions. LVEF should be checked periodically in patients who have HFpEF because these patients may eventually develop HFrEF.[40,41]

HFrEF is defined as clinical HF with reduced EF. Varying EF cutoffs ranging between 35% and 45% have been used to define HFrEF.[42–48] Recent American College of Cardiology/American Heart Association (ACC/AHA) guidelines define systolic HF as LVEF 40% or less.[6] HFrEF is typically characterized by a large thin-walled ventricle that is weak and unable to eject enough blood to produce a normal cardiac output (Table 6). Over the past 2 decades, most randomized clinical trials in HF were restricted to HFrEF.[42–50] It is also the predominant type of HF among younger adults.[51] However, epidemiologic data from the past several decades suggest that as many as 50% of all HF patients may have HFpEF.[52–57] HFpEF is defined as clinical HF with preserved EF, generally 45% to 55% or greater. HFpEF is characterized by a ventricle that may be small or stiff, and does not have enough blood to pump to produce a normal cardiac output (see Table 6). Even though several HF signs, namely elevated JVP and S3, may be more frequent in systolic HF, there is no evidence that the overall clinical manifestations of HF varies by LVEF.[21,22,58–60] Case 2 presented with classic textbook symptoms and signs of HF, and had normal LVEF.

LVEF can be measured using multiple techniques.[61] However, transthoracic 2-dimensional echocardiography with Doppler imaging is usually preferred. This is widely available, safe, and noninvasive with little or no patient discomfort, and provides excellent images of not only the heart but also the great vessels and paracardiac structures.[62] Assessment of LVEF is important because it is of crucial therapeutic and prognostic significance.[21,22,58,60] However, LVEF should not be assessed until after a clinical diagnosis has been made or clinical suspicion exists.

Most diastolic HF patients have significant abnormalities in active and passive relaxation.[55,63] However, it is not essential to determine diastolic abnormalities to make a clinical diagnosis of HFpEF.[56] Doppler studies of velocity of transmitral blood flow can determine ventricular filling patterns. In HFpEF, the peak transmitral E velocity (represents early filling during active ventricular relaxation) is decreased, whereas there is a relative increase in the peak A velocity (due to a compensatory increase in atrial contraction in late diastole). Thus, the E/A ratio is decreased, and even reversed to less than 1 in diastolic HF.[23,55,64] However, E/A ratio may also be decreased with normal aging[65] and may be normalized with progressive diastolic HF (pseudonormalization).[66,67] However, mitral annular tissue Doppler imaging can distinguish normal from pseudonormal filling patterns and, if combined with transmitral flow Doppler imaging and clinical history, can accurately determine severity of diastolic abnormalities.[55,66,67] There is evidence that severity of diastolic dysfunction may be associated with increased mortality.[55]

OTHER DIAGNOSTIC ASSESSMENTS

Initial evaluation for HF should also include a 12-lead electrocardiogram, a chest radiograph, and a laboratory workup, including serum electrolytes, renal function, liver function, thyroid function, lipid profile, complete cell count, fasting blood glucose,

and hemoglobin A1C. All newly diagnosed patients who have HF should be screened for depression using a 15-item Geriatric Depression Scale. Functional status should be ascertained using New York Heart Association (NYHA) functional class. Higher NYHA class is associated with poor outcomes.[68,69]

TREATMENT OF HEART FAILURE

Therapy for older HF patients is similar to that for younger adults but needs to be initiated and titrated with caution following the principle of geriatric pharmacotherapy: start low, go slow! Evidence-based therapy is primarily guided by LVEF and is restricted for patients with HFrEF. These patients should be treated with an angiotensin-converting enzyme (ACE) inhibitor or an angiotensin receptor blocker unless contraindicated.[70] Chronic kidney disease is common in HF and should not be considered a contraindication to these drugs.[71,72] Older HFrEF patients should be treated with approved beta-blockers.[70] There is no need to wait to initiate beta-blockers until a target dose of ACE inhibitor has been achieved. Low-dose digoxin should be used for patients with HFrEF who are symptomatic despite therapy with an ACE inhibitor and a beta-blocker.[73] Aldosterone antagonists should be used in advanced symptomatic HF patients with normal potassium and normal renal function. There is little evidence that these drugs improve outcomes in HFpEF.[74–82] Diuretics are essential for the management of fluid overload in both HFrEF and HFpEF. However, there are little or no data on the long-term safety and these drugs should be in the lowest dose needed to keep patients euvolemic and asymptomatic.[83] Hypokalemia should be avoided and corrected.[84] Decisions to use device-based therapies should be individualized based on patients' functional status and preference.

SUMMARY

Clinical manifestation of HF in older adults may be atypical and diagnostic assessment might be delayed. Assessment of HF in older adults may be made simple by following a simple 5-step process, DEFEAT-HF: diagnosis, etiology, fluid status, EF, and treatment of HF. A thorough history and a careful physical examination should allow proper clinical diagnosis of HF in most cases. An effort to identify an underlying cause for HF must be made. All patients who have HF should be assessed carefully for volume status to achieve euvolemia. LVEF must be determined in all patients with a clinical diagnosis of HF to assess prognosis and guide therapy. All HF patients should be treated with evidence-based therapies according to the recommendations of a major national HF guideline. Older HF patients offer unique challenges in diagnosis and management. A careful consideration and evaluation of comorbidities may afford tailored approaches for management of these patients and contributes toward improving quality of care and outcomes.

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KEY POINTS

- Heart failure is a clinical diagnosis. A diagnosis of heart failure needs to be made based on medical history and clinical examination. There is no single test or procedure to rule in or rule out a diagnosis of heart failure.
- Heart failure is also a clinical syndrome. As such, after a clinical diagnosis of heart failure has been made, underlying etiologic factors for heart failure must be sought because ongoing insults from an etiologic factor such as myocardial ischemia may adversely affect prognosis.
- Heart failure symptoms are often due to fluid retention. Fluid volume status should be carefully assessed by estimating jugular venous pressure by examining both internal and external jugular veins.
- Heart failure prognosis and therapy vary by left ventricular ejection fraction, which should be measured in all heart failure patients, preferably after a clinical diagnosis has been made.
- Heart failure therapy is generally of 2 types: symptom-relieving and outcome-improving. The former applies to all heart failure patients and the latter mostly applies to heart failure and reduced ejection fraction. Recommendations from major national heart failure guidelines should be consulted and treatment of older heart failure patients must be individualized.

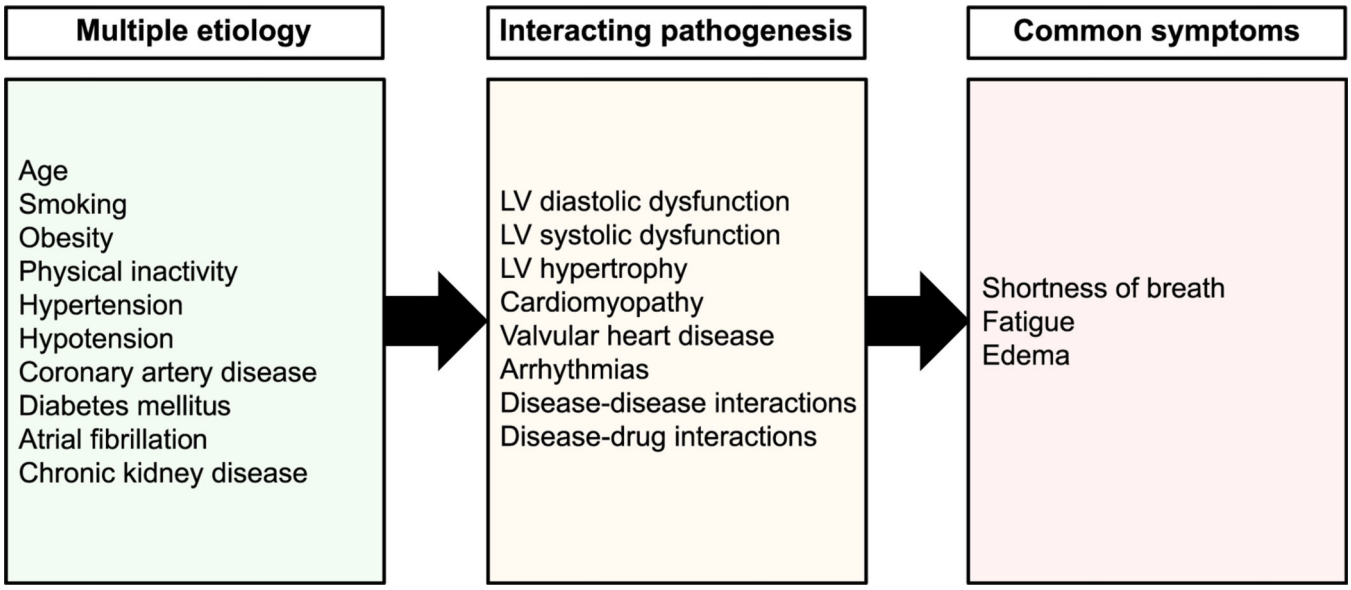

Fig. 1.

Geriatric syndrome model of heart failure in older adults. LV, left ventricular. (*Adapted from* Inouye SK, Studenski S, Tinetti ME, et al. Geriatric syndromes: clinical, research, and policy implications of a core geriatric concept. J Am Geriatr Soc 2007;55:781; with permission.)

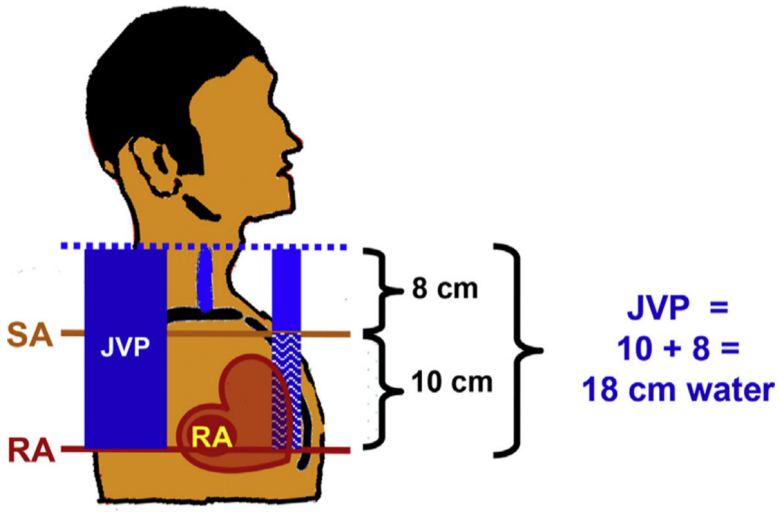

Fig. 2.

Estimation of jugular venous pressure (JVP) in centimeters (cm) of water. First, estimate the distance between the sternal angle (SA) and the top of the jugular venous pulsation (marked by the horizontal dotted blue line) in cm (8 cm in the example above). Then, estimate the distances between right atrium (RA) and SA (10 cm in the example above, but would vary depending on the body position as shown in Fig. 3). Finally, add these two numbers to get the estimated JVP (18 cm water in the example above). (*Adapted from* Ahmed A. Chronic heart failure in older adults. Med Clin N Am 2011;95:439–61; with permission.)

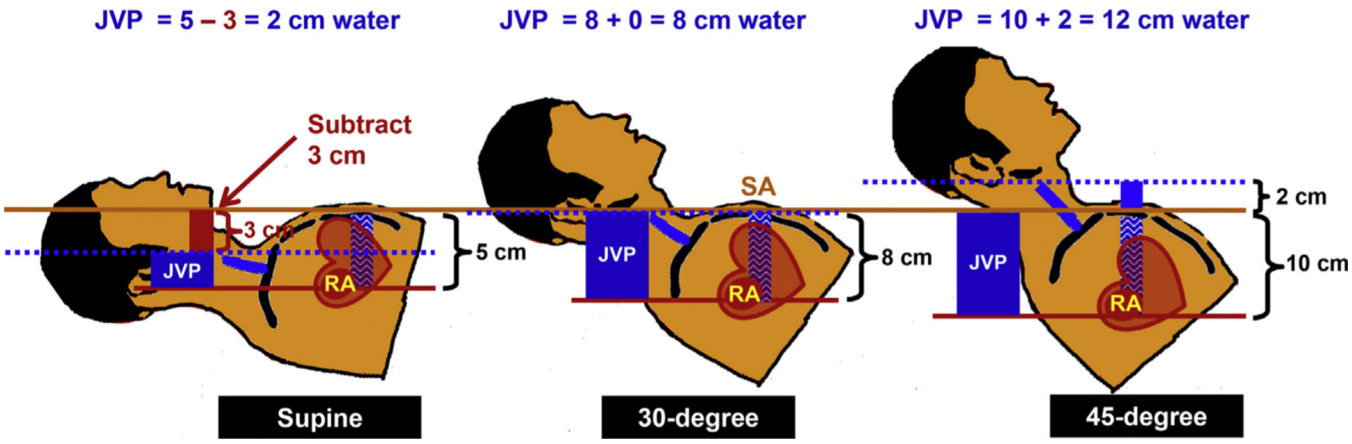

Fig. 3.

Estimation of jugular venous pressure (JVP, marked by the vertical blue bars) in centimeters (cm) of water. First, identify the top of the external jugular venous pulsation in the middle of the neck (the blue line in neck) by adjusting the patient's position. Second, estimate the distance between the sternal angle (SA, marked by the dark brown line), and the top of venous pulsation (marked by the horizontal dotted blue lines). Third, estimate the distance from right atrium (RA, marked by the red line) to SA (note that this distance would vary with the body position). Finally, add these two numbers, the "SA to the top of the jugular venous pulsation distance" and the "RA to SA distance", to obtain JVP in cm water. Please note that when JVP is low as it in the left panel, the top of jugular pulsation is only visible in the middle of the neck when the patient is in the supine position. Because in this position, the top of the jugular pulsation is below the SA, the JVP is estimated by subtracting the "SA to the top of the jugular venous pulsation distance" from the "RA to SA distance. (*Adapted from* Ahmed A. Chronic Heart Failure in Older Adults. Med Clin N Am 2011;95:439–61; with permission.)

Table 1

Examples of clinical presentation of heart failure in older adults

Case 1	A 79-year-old man with a history of hypertension and prior acute myocardial infarction (AMI) presented at an outpatient clinic with a 6-month-old history of progressive dyspnea on exertion (DOE) and leg swelling. He had no history of dyspnea at rest, orthopnea, paroxysmal nocturnal dyspnea (PND), cough, wheezing, or chest pain. He had no emergency department (ED) visits or hospitalizations due to dyspnea. His physical examination was remarkable for mild pitting edema around his ankles and lower legs. His JVP estimated using external jugular vein (EJV) at a 30° incline was 5 cm water. He had no HJR. He had no S3 or pulmonary rales. His electrocardiogram and chest radiograph were normal. An echocardiogram showed a LVEF of 35%. He had no evidence of fluid retention and no diuretic was prescribed. He was prescribed a low-dose angiotensin-converting enzyme (ACE) inhibitor and, over the next several weeks, his symptoms, including his leg edema, improved.
Case 2	An 86-year-old woman with a history of HF with unknown EF and hypertension presented at an outpatient clinic with a 4-wk history of dyspnea and fatigue on minimal exertion (eg, turning over in bed) and orthopnea but no PND (she slept in a recliner) or chest pain. She also complained of weakness, right upper quadrant pain, nausea, loss of appetite, and severe leg swelling. She did not seek ED care, nor was she hospitalized for her symptoms. Her physical examination was remarkable for a JVP of 20 cm water (estimated using EJV in a near-seated position), a positive HJR, a right-sided S3, occasional pulmonary rales but no 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 lower legs. An accentuated S2 at left fourth intercostal space suggested pulmonary hypertension, with an estimated pulmonary artery systolic pressure of 40–45 mm Hg. She had a normal electrocardiogram. A chest radiograph revealed marked cardiomegaly and pulmonary congestion. An echocardiogram later showed an EF >55%. She was on a thiazide diuretic for her hypertension, which was replaced with a loop diuretic. A week later, her symptoms were much improved.
Case 3	An 85-year-old woman with a history of hypertension and atrial fibrillation had progressive dyspnea and fatigue on exertion over the past 6 mo. She presented at the local ED with worsening dyspnea on exertion and a 2-pillow orthopnea. She reported no dyspnea at rest, PND, palpitation, or chest pain. For several days, she had ran out of her atenolol that she took for hypertension for several days. At the ED, her systolic blood pressure (BP) was 220 mm Hg. She had a few bibasilar pulmonary rales and lower extremity edema up to mid-leg. She had no S3. Her JVP was 15 cm water estimated at 50° incline using EJV with a positive HJR. An electrocardiogram was remarkable for atrial fibrillation with a ventricular rate of 170 beats/min but no evidence of ischemia. Her cardiac enzymes were normal. A chest radiograph demonstrated cardiomegaly with mild pulmonary edema. An echocardiogram later showed an EF of 55%. A diagnosis of flash pulmonary edema was made and she was treated with a loop diuretic and a beta-blocker. Her symptoms improved and she was discharged home. Despite controlled BP and heart rate, she needed long-term therapy with a low-dose loop diuretic to avoid HF symptoms.
Case 4	An 84-year-old man, with a history of hypertension and diabetes was recently hospitalized with syncope. He was physically very active and had noted mild DOE before hospitalization. He had no dyspnea at rest, orthopnea, PND, or chest pain. He had 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, which was later increased to 160 mg/d, due to his progressive DOE and fatigue. Over the next 2 wk, he lost over 20 pounds and his symptoms improved. He had trace leg edema. His serum creatinine was 1.5 mg/dL, and his blood urea nitrogen to creatinine ratio was normal at 15. His brain natriuretic peptide (BNP) level was elevated at 400 (normal <100) pg/ml. He was thought to have residual fluid retention and was maintained on furosemide 160 mg/d. Several weeks later, his fatigue and DOE returned. An outpatient examination was remarkable for systolic BP of 95 mm Hg and a JVP of 3 cm water. His furosemide dose was reduced to 80 mg/d. A week 2later, he gained 10 pounds and his HF symptoms improved.
Case 5	An 82-year-old woman with a history of hypertension and CAD presented with a 1-year history of dyspnea at rest, chest tightness, and dizziness presented to the local ED and was hospitalized each time. During these hospitalizations, she underwent comprehensive investigations, including an echocardiogram, a cardiac catheterization, and an MRI of brain, none of which revealed any pathologic condition. She had no DOE, orthopnea, PND, or leg swelling. Her physical examination was unremarkable with no signs of HF. Her JVP was normal at 5 cm water. However, she continued to remain symptomatic. An outpatient evaluation revealed significant loss and stress in personal and social life, and she scored positively on a geriatric depression scale. She denied sad or depressed mood. A diagnosis of somatization associated with geriatric depression was made, and she was started on a low-dose selective serotonin reuptake inhibitor (SSRI). Within several weeks, her symptoms completely resolved.

Adapted from Ahmed A. DEFEAT heart failure: clinical manifestations, diagnostic assessment, and etiology of geriatric heart failure. Heart Failure Clin 2007;3(4):389–402; with permission.

Table 2

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 needs to be made based on routine history and clinical examination. Symptoms and signs of HF are similar in patients with HF and reduced EF (HFrEF) and HF with preserved EF (HFpEF). There is no laboratory test to confirm or rule out a diagnosis of HF. EF should not be used to diagnose HF because more than half of all older adults with HF have HFpEF.
E	Etiology	HF is a syndrome, and as such, a diagnosis is incomplete without the identification of underlying causes. Untreated underlying etiologic factors, such as ongoing myocardial ischemia, may adversely affect prognosis.
F	Fluid volume	Fluid retention is a hallmark sign of HF and is present in nearly all newly diagnosed HF patients. Fluid retention is also a recurring problem for most patients with chronic HF. JVP in centimeters of water needs to be estimated by careful examination of both internal and external jugular venous pulsations in the middle of the neck to properly assess fluid volume status. A description of “jugular venous distension” should be avoided. Also, a description of “no fluid retention” should be avoided because some HF patients may suffer from hypovolemia, especially if overdiuresed.
E	EF	LVEF should be determined using an echocardiogram and used to assess prognosis and guide therapy.
A	and	
T	Treatment	HF therapy can be broadly categorized as symptom-relieving and outcome-improving. Because HF symptoms are indistinguishable between HFrEF and HFpEF, therapy directed to relieve HF symptoms is generally similar for both HFrEF and HFpEF. Evidence-based therapy to improve outcomes, on the other hand, is often restricted to HFrEF. Recommendations of major HF guidelines are mostly based on clinical trials in younger patients with HFrEF but most can reasonably individualized for older patients with HFrEF with the principle of start low, go slow.

Adapted from Ahmed A. Chronic heart failure in older adults. Med Clin N Am 2011;95:441; with permission.

Table 3

Diagnosis and etiologic factors of heart failure in older adults

	Cases					
	1	**2**	**3**	**4**	**5**	**Comments**
DOE	**Yes**	**Yes**	**Yes**	**Yes**	No	Case 1 had DOE without other associated symptoms and signs, and a clinical diagnosis of HF could not be made. He had low EF but because of his DOE he could not be diagnosed to have asymptomatic LV systolic dysfunction. So, his low EF in the context of DOE helped make a diagnosis of HF.
Orthopnea	No	**Yes**	**Yes**	No	No	Case 2 slept in in recliner to avoid orthopnea. Use of multiple pillows or sleep in a recliner, or use blocks or bricks to raise the head of bed to avoid orthopnea may be used as markers of orthopnea.
PND	No	**Yes**	No	No	No	Case 2 slept in in recliner to avoid orthopnea and thus may also have avoided PND.
Dyspnea at rest	No	**Yes**	No	No	**Yes**	Case 2 had near-class IV symptoms. Case 5 had dyspnea at rest but not on exertion, unlikely to be HF.
Fatigue	Yes	**Yes**	**Yes**	No	No	Often experienced when patients do things at their own pace.
Dizziness	No	No	No	**Yes**	**Yes**	Not a typical HF symptom. Likely due to hypotension (case 4) or somatization (case 5).
Syncope	No	No	No	**Yes**	No	Atypical. Likely due to hypotension (case 4). A risk factor for sudden cardiac death.
Chest pain	No	No	No	No	No	Uncommon as an HF symptom.
Cough	No	No	No	No	No	Rare. May follow dyspnea or PND but may also precede dyspnea.
Swelling of foot or leg	**Yes**	**Yes**	**Yes**	**Yes**	No	When onset of leg swelling is simultaneous with DOE, as in case 1, it may be more suggestive of HF than having different onsets.
GI symptoms	No	**Yes**	No	No	No	Rare in chronic HF. May suggest congestive hepatopathy. Maybe accompanied by right upper quadrant pain and tenderness, and some mild abnormalities of liver function.
COPD	No	No	No	No	No	Differentiating dyspnea of HF from that of COPD may be difficult. Important to carefully estimate JVP to assess fluid balance.
Deconditioning	No	**Yes**	No	No	No	May manifest as DOE and fatigue but often there is no orthopnea, PND, leg edema.
Depression	No	No	No	No	**Yes**	May manifest as somatization, as in case 4. Physiologically implausible symptoms, such as dyspnea at rest without DOE, should raise suspicion
HF risk factors	**Yes**	**Yes**	**Yes**	**Yes**	**Yes**	Often present as morbidities in older patients with HF.
JVP	5	20	15	3	5	Almost always present during initial presentation and acute exacerbation of HF but chronic stable patients may have normal JVP, and may even be low if overdiuresed.
Jugular waveforms	?	?	?	?	?	Not needed to estimate JVP or make a diagnosis of HF. Familiarity with the double undulation of the jugular venous pulsation may help distinguish it from carotid pulsation.
Third heart sound	No	**Yes**	No	No	No	Often present but not necessary to establish a diagnosis of HF.
Pulmonary rales	No	**Yes**	**Yes**	No	No	Rare in chronic HF due to efficient pulmonary lymphatic system in these patients.
HJR	No	**Yes**	**Yes**	No	No	It helps identify the position and patency of EJV in neck. When present in those with normal JVP it may indicate early fluid retention.

	Cases					
	1	**2**	**3**	**4**	**5**	**Comments**
LE edema	**Yes**	**Yes**	**Yes**	Yes	No	Pitting edema despite normal JVP may indicate chronic venous insufficiency. It is also accompanied by skin discoloration and stasis dermatitis.
Cardiomegaly	No	**Yes**	**Yes**	**Yes**	No	Cardiomegaly by chest radiograph may help support a clinical diagnosis of HF
Pulmonary venous congestion	No	**Yes**	No	No	No	Uncommon in chronic HF but when present in radiograph may support diagnosis
Pulmonary edema	No	No	**Yes**	No	No	Rare in chronic HF. May occur in acute decompensation.
Pleural effusion	No	No	No	No	No	Not uncommon, usually bilateral.
BNP	Not done	Not done	Not done	400	No	Elevated BNP and 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. Role in older adults is less clear. Rarely needed for diagnosis and management. Case 4 had elevated BNP when he was dry and that may have led to overdiuresis and hypovolemia.
LVEF	35%	55%	55%	25%	55%	A low EF may support a diagnosis of HF when clinical presentation is atypical or insufficient as in case 1. A normal EF should not be used to rule out a diagnosis of HF.
Response to diuretics	NA	**Yes**	**Yes**	**Yes**	NA	A therapeutic response to diuretic may help confirm the diagnosis of HF when clinical presentation is atypical or insufficient.
ED visit	No	No	**Yes**	No	**Yes**	Older adults often attribute HF symptoms to aging thus delaying diagnosis and therapy that may increase their risk of ED visit and hospital admission.
Hospitalization	No	No	No	**Yes**	**Yes**	Charts from prior hospital admissions for HF maybe help in the diagnosis, especially in patients whose symptoms and signs are compensated.

Case 1: no clinical evidence of HF and the diagnosis was facilitated by low EF.

Case 2: a classic textbook presentation of HF.

Case 3: flash pulmonary edema, ongoing symptoms, and need for diuretic therapy.

Case 4: HF symptoms, low EF, and diuretic response.

Case 5: no HF, symptoms were likely somatization associated with depression.

Abbreviations: BNP, brain natriuretic peptide; COPD, chronic obstructive pulmonary disease; GI, gastrointestinal; LE, lower extremity; NA, not applicable; NT-pro-BNP, N-terminal probrain natriuretic peptide.

Table 4

Symptoms and signs of heart failure in older adults

Dyspnea	DOE or exertional fatigue, with or without some degree of lower extremity swelling, is generally the most common early symptom of HF (cases 1 and 3). With progression of disease, especially in the absence of appropriate treatment, DOE or fatigue gradually become more severe and appears with decreasing exertion (case 2), and eventually at rest. Older adults often attribute their DOE or fatigue on exertion to aging and respond to their early symptoms by restricting their physical activities, thus delaying clinical manifestations and diagnosis. It is important to take that into consideration while inquiring about DOE from an older adult (see Table 2). When a patient presents with dyspnea at rest, it is important to determine its duration and if it was preceded by DOE. Dyspnea at rest without DOE is almost never organic in cause and may represent somatization in older adults.[85]
Orthopnea	Orthopnea is a relatively specific symptom for HF in older adults (see Table 2).[5,86] Orthopnea is particularly helpful if associated with edema (may help distinguish from rare orthopnea due to pulmonary causes). It is usually occurs soon after lying down and is also relieved promptly by sitting or standing up. However, orthopnea is relatively infrequent symptom in older adults with HF and may not be reported until fluid overload is severe, as in cases 2 and 3.[87,88] Many older adults may sleep in a chair or a recliner toavoid orthopnea and may not voluntarily reportthat unless specifically asked.
PND	PND is a more specific HF symptom.[5,86] Dyspnea in PND may occur 2–3 h after onset of sleep, and causes patients to wake up from sleep with dyspnea, which may be followed by cough and/or wheezing. Relief starts with sitting up but complete relief of symptoms may take about 30 min. Patients sleeping with multiple pillows or on a recliner to avoid orthopnea may not experience PND. When PND is due to COPD, cough maybe the early and predominant symptom. However, PND is relatively infrequent in older adults.[87,88]
LE edema	Edema is a relatively nonspecific symptom. It is estimated that 5 L of extra fluid must accumulate before edema become clinically manifest.[14] Edema is generally dependent and progressive if untreated. Edema associated with HF is always symmetric and pitting. Chronic venous insufficiency is common in older adults and may cause pitting edema. Unilateral edema may be caused by cellulitis, past trauma or surgery, deep vein thrombosis, or arthritis. Chronic severe edema may lead to skin changes, including erythema, brown pigmentation, and induration. Leg edema alone should not be used to assess fluid volume status.
Other symptoms	Other less common and atypical symptoms of HF in older adults include fatigue, syncope, angina, nocturia, oliguria, and changes in mental status. Weight gain almost always accompanies symptomatic HF but is rarely reported as a symptom by older HF patients. However, if educated by their doctors about the importance of daily weight, most older HF patients are likely to monitor and report. This may, however, confounded by loss of appetite and early satiety, which in turn may result in weight loss and failure to thrive.
JVP	An elevated JVP is the most specific sign of fluid overload in HF and is the most important physical examination in the initial and subsequent examinations of an older HF patient.[89] External jugular veins (EJV) maybe more easily seen than internal jugular vein (IJV) in older adults with chronic HF and maybe used judiciously to estimate JVP. Using EJV and the right technique, JVP can be estimated in 90%–95% of all HF patients.
HJR	HJR reflects inability of the right ventricles to respond to increased venous return, which is caused by pressure over an abdomen in which the veins may already be congested with blood.[90] HJR is considered positive if the JVP rises by 2–3 cm and remains elevated for about 10 s when sustained pressure is applied to the mid-abdomen area. In 1 study, a positive HJR predicted right atrial pressure >9 mm Hg with high sensitivity (100%) and specificity (85%).[91] A positive HJR in the presence of high JVP confirms fluid overload.[90,91] A positive HJR in the presence of normal or low JVP may indicate mild residual fluid overload (may be baseline for some HF patients).
Other signs	An fourth heart sound (S4) may be common in older HF patients. Ascites and pleural effusion are nonspecific signs and must be coordinated with other symptoms and signs.

Adapted from Ahmed A. DEFEAT heart failure: clinical manifestations, diagnostic assessment, and etiology of geriatric heart failure. Heart Failure Clin 2007;4:389–402; with permission.

Table 5

Estimation of jugular venous pressure in older adults with heart failure

	Cases					
	1	**2**	**3**	**4**	**5**	**Comments**
IJV used to estimate JVP?	No	No	No	Yes	No	Transmitted IJV pulsation is most ideal for reliable estimation of JVP but not easily visible in older adults with chronic HF.
EJV used to estimate JVP?	Yes	Yes	Yes	Yes	Yes	EJV contour is visible and its pulsation is more easily visible; a reliable source for JVP estimation.
HJR	No	Yes	Yes	No	No	Not needed for JVP estimation; useful to check for patency of EJV.
Incline needed to make top of jugular pulsation visible in neck	0°	90°	30°	0°	0°	The head of the examination table or bed must be adjusted to make the top of the IJV or EJV pulsation visible in the middle of the neck.
Estimated distance from RA to SA (cm)	5	10	8	5	5	The RA to SA distance is rather fixed and is incline-specific (5, 8, and 10 cm at 0, 30, and 45 incline).[36]
Estimated distance from SA to jugular pulsation top (cm)	0	10	7	−2	0	The SA to top of jugular pulsation distance is vertical, not horizontal. This is especially important to remember in patients with low JVP (Case 5) who should be in a supine or near-supine position.
Estimated JVP (cm water)	5	20	15	3	5	5–8 cm water normal for most, and up to 10 for some HF patients. A lower JVP may indicate over-diuresis.
How JVP (cm water) was estimated	5 + 0 = 5	10 + 10 = 20	8 + 7 = 15	5-2 = 3	5 + 0 = 5	The vertical SA to top of jugular pulsation distance is added to the incline-specific RA to SA distance to estimate JVP; however, it must be **subtracted** from the RA to SA distance when JVP is low (Case 5).

Abbreviations: RA, right atrium; SA, sternal angle.

Table 6

A simplified example demonstrating how patients with heart failure with reduced ejection fraction and with heart failure preserved ejection fraction have similar symptoms and signs

	End-Diastolic Volume (mL)	LVEF (%)	Left Ventricular Stroke Volume (mL)	Heart Rate (Beats per min)	Cardiac Output (liters per min)	Symptoms
Normal	150	55	75	72	5.4	None
HFrEF	200	25	50	72	3.6	Dyspnea, fatigue, edema
HFpEF	100	55	50	72	3.6	Dyspnea, fatigue, edema

Adapted from Ahmed A. DEFEAT heart failure: clinical manifestations, diagnostic assessment, and etiology of geriatric heart failure. Heart Failure Clin 2007;4:391; with permission.