CLINICAL GUIDELINE:

EVALUATION AND MANAGEMENT OF HEART FAILURE
In the Ambulatory Setting

Physician Clinical Integration Network, LLC

Scope

An estimated 6.2 million people were diagnosed with heart failure (HF) from 2013 - 2016. Prevalence continues to rise with 3,994 Americans on the waiting list for heart transplant in 2018 [1]. HF is considered a global pandemic as it affects approximately 26 million people worldwide and is expected to rise due to an aging and overweight population. More people are surviving heart attacks, resulting in higher heart failure risk [2].

Guidelines published in 2013 by the American College of Cardiology Foundation (ACCF) and the American Heart Association (AHA) recommended the expansion of evidence-based pharmacotherapies, resulting in a 44% reduction of deaths in patients with HF [9].

Population Included

Patients diagnosed with heart failure in the ambulatory care setting

Exclusions

None

Guidance

The PCIN Quality Committee and its designees reviewed the available information in the medical literature and societal guidelines on the evaluation and management of heart failure, as well as information derived from their clinical practice to devise these guidelines.

The recommendations for this guideline are based on research conducted by the American College of Cardiology (ACC), AHA Task Force on Clinical Practice Guidelines and the Heart Failure Society of America, updated in 2017.

Recommendations

Diagnosis

- ✓ A thorough history and physical examination should be performed in patients presenting with HF to identify cardiac and noncardiac disorders or behaviors that might cause or accelerate the development or progression of HF (Figure 3) [3].
- ✓ Initial laboratory evaluation of patients presenting with HF should include:
  - Complete blood count
  - Urinalysis
  - Serum electrolytes (including calcium and magnesium)
  - Blood urea nitrogen
  - Serum Creatinine
  - Glucose
  - Fasting lipid profile
  - Liver function tests
  - Thyroid-stimulating hormone
  - B-type natriuretic peptide (BNP) (See recommendations under "Biomarkers") [3]
- ✓ A 12-lead electrocardiogram (ECG) should be performed initially on all patients presenting with HF [3].
- ✓ A chest x-ray should be performed on patients with suspected or new-onset HF or with acute decompensated HF to assess heart size and pulmonary congestion and to detect alternative cardiac, pulmonary, and other diseases that may cause or contribute to the patient's symptoms [3].
- ✓ A two-dimensional echocardiogram with Doppler should be performed during initial evaluation of patients presenting with HF to assess ventricular function, size wall thickness, wall motion, and valve function [3].

Biomarkers

- ✓ Prevention
  - For patients at risk of developing HF, natriuretic peptide (NP) biomarker-based screening followed by team-based care, including a cardiovascular specialist optimizing guideline-directed medical therapy (GDMT), can be useful to prevent the development of left ventricular dysfunction (systolic or diastolic) or new onset HF.
- ✓ Diagnosis
  - In patients presenting with dyspnea, measurement of NP biomarkers is useful to support a diagnosis or exclusion of HF.
- ✓ Establishing prognosis or added risk stratification in chronic HF
  - Measurement of B-type natriuretic peptide (BNP) or N-terminal-pro hormone (NT-pro) BNP
  - Measurement of baseline levels of NP biomarkers and/or cardiac troponin on admission to the hospital
  - Measurement of other clinically available tests, such as biomarkers of myocardial injury or fibrosis, may be considered for additive risk stratification (Figure 1) [3].

Treatment

Stage A: High risk of heart failure, but no structural heart disease or symptoms of heart failure

- ✓ Hypertension and lipid disorders should be controlled in accordance with contemporary guidelines to lower the risk of HF.
- ✓ Other conditions (as listed below) that may lead or contribute to HF should be controlled or avoided:
  - Obesity
  - Diabetes mellitus
  - Tobacco use
  - Known cardiotoxic agents
- ✓ Pharmacologic Recommendations:
  - Diuretic-based antihypertensive therapy
  - Angiotensin-converting enzyme (ACE) inhibitors
  - Angiotensin II receptor blockers (ARBs)
  - Beta blockers

- Statins for hyperlipidemia [15]

Stage B: Structural heart disease, but no symptoms of heart failure

- ✓ For patients with a recent or remote history of myocardial infarction (MI) or acute coronary syndrome (ACS) and reduced ejection fraction (EF):
  - ACE inhibitors should be used to prevent symptomatic HF and reduce mortality
  - ARBs should be used in patients intolerant of ACE inhibitors
  - Evidence-based beta blockers to reduce mortality
- ✓ For patients with a recent or remote history of MI or ACS:
  - Statins should be used
- ✓ For patients with structural cardiac abnormalities, including left ventricular (LV) hypertrophy, in the absence of a history of MI or ACS:
  - Blood pressure should be controlled in accordance with clinical practice guidelines for hypertension to prevent symptomatic HF.
- ✓ Patients with a reduced EF (even without a history of MI):
  - ACE inhibitors to prevent symptomatic HF
  - Beta blockers to prevent symptomatic HF [15]

Stage C: ***Structural heart disease and symptoms of heart failure***

- ✓ Patients with HF should receive specific education to facilitate HF self-care [15].
- ✓ Sodium restriction is reasonable for patients with symptomatic HF to reduce congestive symptoms [15].
- ✓ Exercise training (or regular physical activity) is recommended as safe and effective for patients with HF who are able to participate to improve functional status [15].
- ✓ Patients with chronic HF with reduced ejection fraction (HF*r*EF) (Figure 2)
  - Renin-angiotensin system inhibition for patients with chronic HF*r*EF
    - ACE inhibitors; or
    - ARBs; or
    - Angiotensin receptor-neprilysin inhibitor (ARNI) in conjunction with evidence-based beta blockers, and aldosterone antagonists in selected patients;
    - ARBs in patients intolerant to ACE inhibitors because of cough or angioedema.
  - Chronic symptomatic HF*r*EF
    - ACE inhibitors
    - ARBs for patients intolerant of ACE inhibitors because of cough or angioedema
    - New York Heart Association (NYHA) class II or III who tolerate an ACE inhibitor or ARB, replacement by an ARNI is recommended to further reduce morbidity and mortality
  - Other
    - ARNI should not be administered concomitantly with ACE inhibitors or within 36 hours of the last dose of an ACE inhibitor
    - ARNI should not be administered to patients with a history of angioedema
  - Ivabradine
    - Can be beneficial to reduce HF hospitalization for patients with symptomatic (NYHA class II-III) stable chronic HF*r*EF (left ventricular ejection fraction [LVEF] ≤35%) who are receiving guideline directed evaluation and management (GDEM), including a beta blocker at maximum tolerated dose, and who are in sinus rhythm with a heart rate of 70 bpm or greater at rest [3].
- ✓ Patients with Heart Failure with Preserved Ejection Fraction (HF*p*EF):
  - Systolic and diastolic blood pressure should be controlled in accordance with published clinical practice guidelines to prevent morbidity. The optimal blood pressure for those with hypertension should be less than 130/80 mm Hg.
  - Diuretics should be used for relief of symptoms due to volume overload.
  - Coronary revascularization is reasonable in patients with CAD in whom symptoms (angina) or demonstrable myocardial ischemia is judged to be having an adverse effect on symptomatic HFpEF despite GDMT.
  - The use of beta-blocking agents, ACE inhibitors, and ARBs in patients with hypertension is reasonable to control blood pressure.

- Patients with EF ≥45%; elevated BNP levels or HF admission within one year; estimated glomerular filtration rate >30mL/min; creatinine <2.5 mg/dL; potassium <5.0 mEq/L:
  - Aldosterone receptor antagonists might be considered to decrease hospitalizations.
- The use of ARBs might be considered to decrease hospitalizations [3].

Stage D: Refractory heart failure requiring specialized interventions

✓ Fluid restriction (1.5 to 2 L/d) is reasonable, especially in patients with hyponatremia, to reduce congestive symptoms [3].

Rationale

Diagnosis

Diagnosis of HF is based on:
- Clinical history (e.g. CAD, arterial hypertension, diuretic use)
- Presenting symptoms (orthopnea)
- Physical examination (bilateral edema, increased jugular venous pressure, displaced apical beat)
- Resting ECG
- Laboratory tests (Figure 5) [10]

If one or more of these elements is abnormal, a BNP is indicated. If the NP level is above the exclusion threshold, or if circulating NP levels cannot be assessed, an ECG should be performed (Figure 3).

The ECG is an integral part of the cardiovascular evaluation of a patient with HF as a baseline for comparison to any earlier ECG's and with new or worsening congestion or low cardiac output syndrome [3]. Patients with an EF of at least 50% and present with clinical symptoms consistent with heart failure meet the diagnosis criteria for HF*p*EF [5].

Imaging studies may be indicated when alternative causes are being considered:
- A chest x-ray is of limited value for diagnosing HF and should only be performed to identify suspected pulmonary malignancy or interstitial pulmonary disease [4,8].
- Computerized tomography (CT) scans for accurate assessment of cardiac structure and function, including the coronary arteries [4].
- Transthoracic echocardiography (TTE) is used for the assessment of myocardial systolic and diastolic function of the left and right ventricles [4,8].
- Stress echocardiography is helpful in the assessment of inducible ischemia and/or myocardium viability and in patients with mitral valve disease.
- Cardiac magnetic resonance imaging (MRI) is considered the gold standard for the measurements of volumes, mass and EF of both the right and left ventricles [8]. It also provides valuable information about myocardial perfusion, viability and fibrosis to identify HF etiology and assess prognosis [4].
- Coronary angiography is recommended in patients with HF who have angina pectoris, with a history of symptomatic ventricular arrhythmia or aborted cardiac arrest [8].

The diagnosis of HF*p*EF is made based on the presence of symptoms and/or signs of HF a preserved EF (defined as LVEF ≥50% or 40-49% for HF*mr*EF); elevated levels of NPs (BNP >35 pg/mL and/or NT-proBNP >125 pg/mL); objective evidence of other cardiac functional and structural alterations underlying HF; and in the case of uncertainty, a stress test or invasively measured elevated LV filling pressure may be needed to confirm the diagnosis [8].

Biomarkers

Biomarkers provide valuable information regarding the pathophysiology of HF, enabling clinicians to identify possible underlying causes, confirm the presence or absence of the HF syndrome, and estimate the severity and disease progression [4]. New data (2019) supports the use of natriuretic peptide biomarker screening with early intervention to prevent and support clinical judgment when the etiology of dyspnea is unclear [3].

Management of Heart Failure

HF is classified according to time of onset (acute or chronic) and divided into two major types based on the functional status of the heart (HF*p*EF and HF*r*EF).

ACC/AHA staging system is defined as:

- Stage A: High risk of heart failure, but no structural heart disease or symptoms of heart failure
- Stage B: Structural heart disease, but no symptoms of heart failure
- Stage C: Structural heart disease and symptoms of heart failure
- Stage D: Refractory heart failure requiring specialized interventions

Therapeutic recommendations for each stage build upon the previous stage; therefore, recommendations for Stage A patients should be followed for Stage B, C, and D patients (Figure 4).

The goals for treatment of HF are to improve prognosis, reduce mortality, alleviate symptoms, and reduce morbidity by reversing or slowing the cardiac and peripheral dysfunction [4]. ACE inhibitors, ARBs, and beta blockers have been shown to reduce mortality and hospital admissions due to HF [12].

Stage A

The goals of therapy for Stage A HF is to practice a heart healthy lifestyle, prevent vascular and/or coronary disease, and prevent LV structural abnormalities [7].

Stage B

The goals of therapy for Stage B HF is to prevent HF symptoms and further cardiac remodeling [7].

Stage C

Management is dependent on whether the HF patient presents with preserved EF or if HF is caused by left ventricular systolic dysfunction (LVSD) [13].

The number of patients with HF and a normal EF continues to increase due to common risk factors such as older age, female sex, hypertension, metabolic syndrome, renal dysfunction and obesity [5]. The goals of therapy for Stage C HF*p*EF is to control symptoms, improve health-related quality of life (HRQOL), prevent hospitalization, and prevent mortality, focusing on managing comorbid conditions (e.g., hypertension, ischemic heart disease, diabetes mellitus) [13].

The goals for Stage C HF*r*EF is to control symptoms, patient education, prevent hospitalizations and mortality [7]. An ACE inhibitor and a beta blocker is recommended for patients with LVSD [14]. Research indicates patients with stable systolic HF, already on maximum medication therapy, may benefit from the addition of ivabradine. While it may reduce hospitalizations, it has not been proven to reduce cardiovascular mortality. Patients should be monitored for the development of atrial fibrillation and bradycardia associated with the use of ivabradine [11]. High sodium intake has been linked to the development of cardiovascular disorders; however, based on current evidence, care should be taken in reducing sodium consumption to less than 2,300 mg per day. Sodium restricted diets with the use of diuretics require close monitoring of serum sodium levels and creatinine [14].

All stable patients should be offered supervised exercise-based rehabilitation, shown to reduce hospitalizations and increase quality of life, particularly for patients with LVSD [13].

Stage D

ACC and AHA define advanced HF as a pattern of clinical characteristics that include repeated ($\geq$2) hospitalizations or Emergency Department visits for HF in the past year; progressive deterioration in renal function; weight loss without other causes; intolerance of ACE inhibitors due to hypotension and/or worsening renal function; intolerance of beta blockers due to worsening HF or hypotension; frequent systolic blood pressure <90 mm Hg; persistent dyspnea with dressing or bathing requiring rest; inability to walk one block on level ground due to dyspnea or fatigue; recent need to escalate diuretics to maintain volume status, often reaching daily furosemide equivalent dose >160 mg and/or use of supplemental metolazone therapy; progressive decline in serum sodium, usually to <133 mEq/L; and/or frequent implantable cardioverter-defibrillator (ICD) shocks [6].

The goals of therapy for Stage D HF is to control symptoms, improve HRQOL, reduce hospital admissions, and establish patient's end-of-life goals [7].

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Appendix

Figure 1: Biomarkers Indications for Use

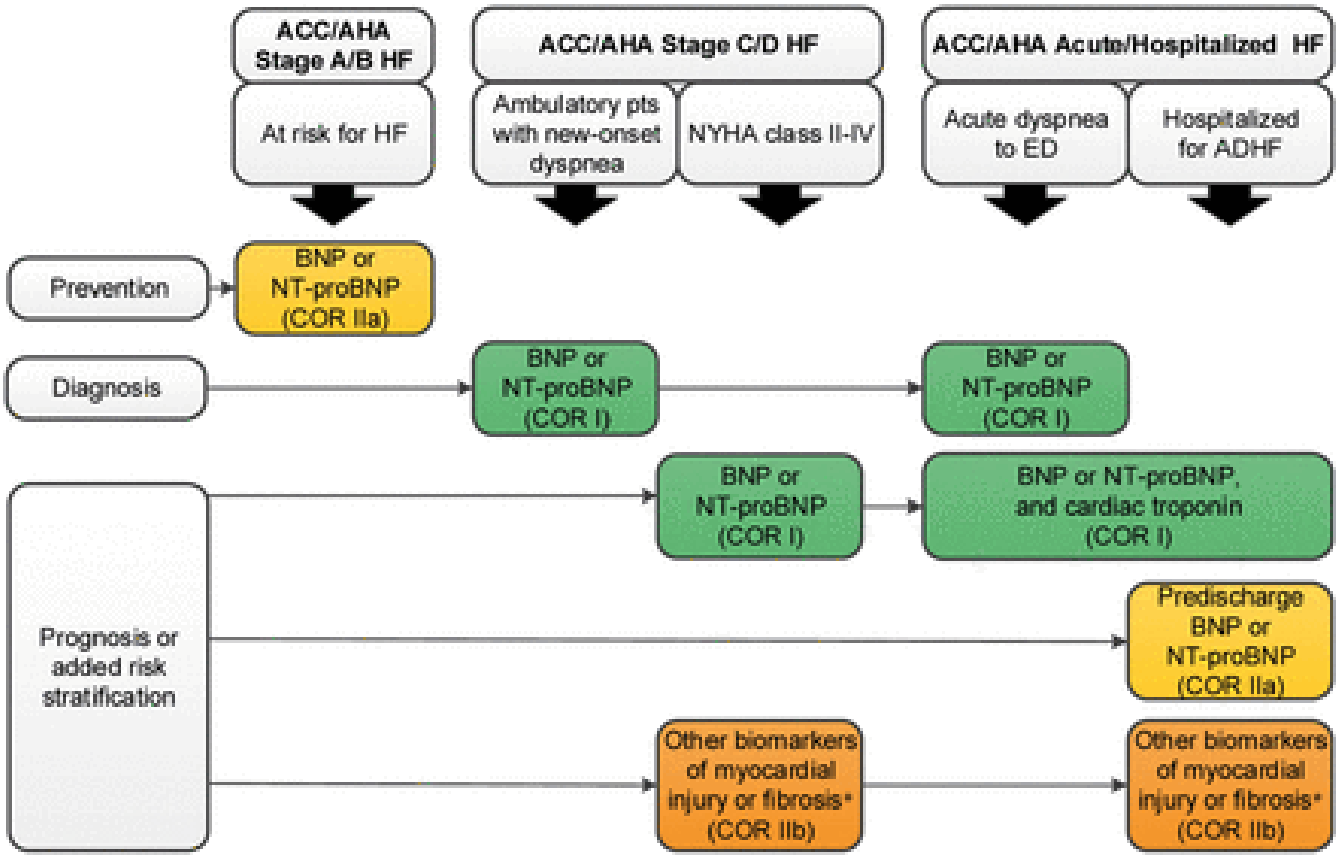

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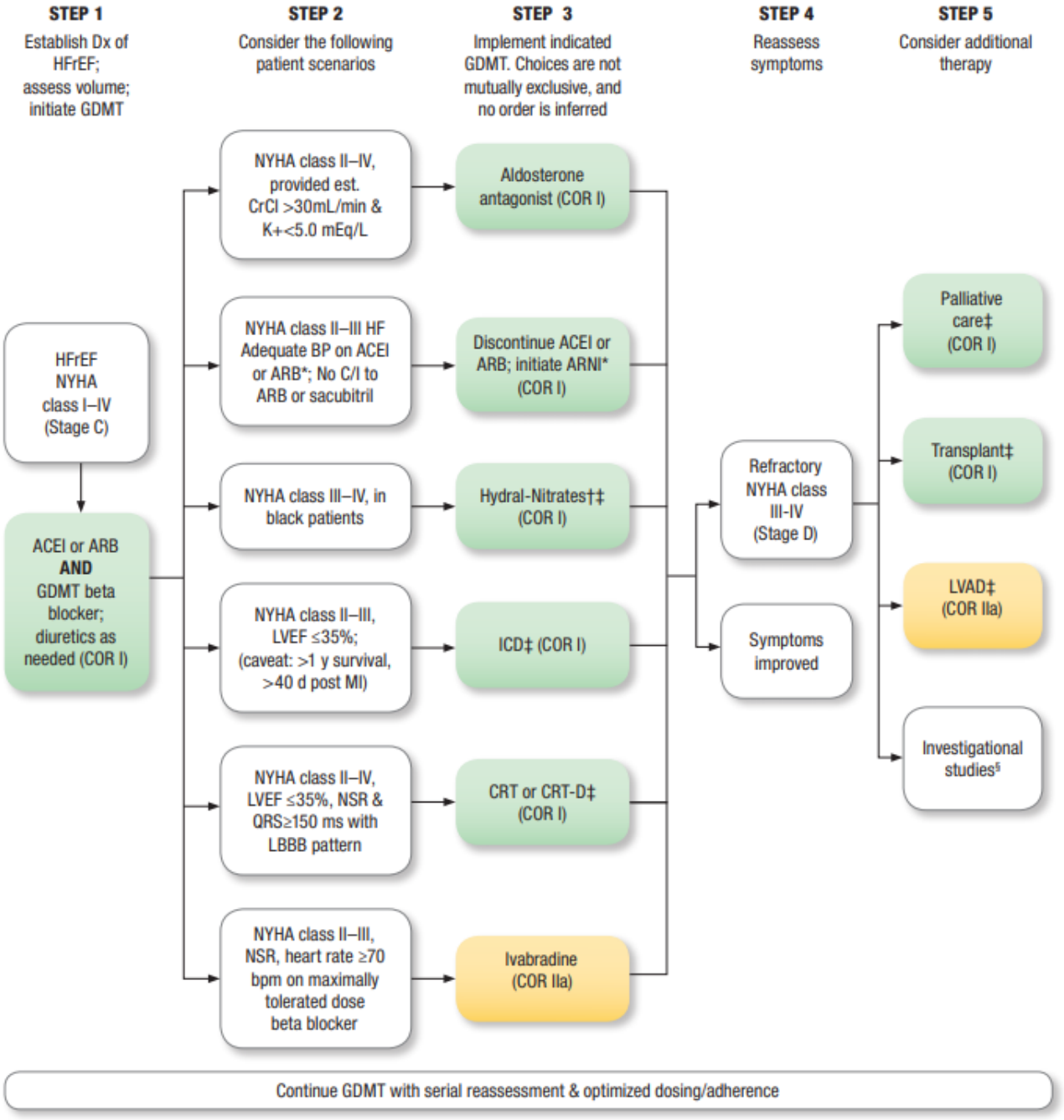
Figure 2: Treatment of HF*r*EF Stage C and D



Colors correspond to COR in Table 1. For all medical therapies, dosing should be optimized and serial assessment exercised.
*See text for important treatment directions.
†Hydral-Nitrates green box: The combination of ISDN/HYD with ARNI has not been robustly tested. BP response should be carefully monitored.
‡See 2013 HF guideline (9).
§Participation in investigational studies is also appropriate for stage C, NYHA class II and III HF.
ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor-blocker; ARNI, angiotensin receptor-neprilysin inhibitor; BP, blood pressure; bpm, beats per minute; C/I, contraindication; COR, Class of Recommendation; CrCl, creatinine clearance; CRT-D, cardiac resynchronization therapy–device; Dx, diagnosis; GDMT, guideline-directed management and therapy; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; ICD, implantable cardioverter-defibrillator; ISDN/HYD, isosorbide dinitrate hydral-nitrates; K+, potassium; LBBB, left bundle-branch block; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSR, normal sinus rhythm; and NYHA, New York Heart Association.

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Yancy CW, Jessup M, Bozkurt B, Butler J, Casey Jr DE, Colvin MM, Drazner MH, Filippatos GS, Fonarow GC, Givertz MM, Hollenberg SM, Lindenfeld J, Masoudi FA, McBride PE, Peterson PN, Stevenson LW, Westlake C, 2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure, Journal of the American College of Cardiology (2017), doi: 10.1016/j.jacc.2017.04.025

Figure 3: History and Physical Examination in HF

	Comments
History	
Potential clues suggesting etiology of HF	A careful family history may identify an underlying familial cardiomyopathy in patients with idiopathic DCM.[118] Other etiologies outlined in Section 5 should be considered as well.
Duration of illness	A patient with recent-onset systolic HF may recover over time.[113]
Severity and triggers of dyspnea and fatigue, presence of chest pain, exercise capacity, physical activity, sexual activity	To determine NYHA class; identify potential symptoms of coronary ischemia.
Anorexia and early satiety, weight loss	Gastrointestinal symptoms are common in patients with HF. Cardiac cachexia is associated with adverse prognosis.[191]
Weight gain	Rapid weight gain suggests volume overload.
Palpitations, (pre)syncope, ICD shocks	Palpitations may be indications of paroxysmal AF or ventricular tachycardia. ICD shocks are associated with adverse prognosis.[192]
Symptoms suggesting transient ischemic attack or thromboembolism	Affects consideration of the need for anticoagulation.
Development of peripheral edema or ascites	Suggests volume overload.
Disordered breathing at night, sleep problems	Treatment for sleep apnea may improve cardiac function and decrease pulmonary hypertension.[193]
Recent or frequent prior hospitalizations for HF	Associated with adverse prognosis.[194]
History of discontinuation of medications for HF	Determine whether lack of GDMT in patients with HF*r*EF reflects intolerance, an adverse event, or perceived contraindication to use. Withdrawal of these medications has been associated with adverse prognosis.[195,196]
Medications that may exacerbate HF	Removal of such medications may represent a therapeutic opportunity.
Diet	Awareness and restriction of sodium and fluid intake should be assessed.
Adherence to medical regimen	Access to medications; family support; access to follow-up; cultural sensitivity
Physical Examination	
BMI and evidence of weight loss	Obesity may be a contributing cause of HF; cachexia may correspond with poor prognosis.
Blood pressure (supine and upright)	Assess for hypertension or hypotension. Width of pulse pressure may reflect adequacy of cardiac output. Response of blood pressure to Valsalva maneuver may reflect LV filling pressures.[197]
Pulse	Manual palpation will reveal strength and regularity of pulse rate.
Examination for orthostatic changes in blood pressure and heart rate	Consistent with volume depletion or excess vasodilation from medications.
Jugular venous pressure at rest and following abdominal compression (http://wn.com/jugular_venous_distension_example)	Most useful finding on physical examination to identify congestion.[187–190,198]
Presence of extra heart sounds and murmurs	S_3 is associated with adverse prognosis in HF*r*EF.[188] Murmurs may be suggestive of valvular heart disease.
Size and location of point of maximal impulse	Enlarged and displaced point of maximal impulse suggests ventricular enlargement.
Presence of right ventricular heave	Suggests significant right ventricular dysfunction and/or pulmonary hypertension.
Pulmonary status: respiratory rate, rales, pleural effusion	In advanced chronic HF, rales are often absent despite major pulmonary congestion.
Hepatomegaly and/or ascites	Usually markers of volume overload.
Peripheral edema	Many patients, particularly those who are young, may be not edematous despite intravascular volume overload. In obese patients and elderly patients, edema may reflect peripheral rather than cardiac causes.
Temperature of lower extremities	Cool lower extremities may reflect inadequate cardiac output.

AF indicates atrial fibrillation; BMI, body mass index; DCM, dilated cardiomyopathy; GDMT, guideline-directed medical therapy; HF, heart failure; HF*r*EF, heart failure with reduced ejection fraction; ICD, implantable cardioverter-defibrillator; LV, left ventricular; and NYHA, New York Heart Association.

Yancy CW, Jessup M, Bozkurt B, Butler J, Casey Jr DE, Colvin MM, Drazner MH, Filippatos GS, Fonarow GC, Givertz MM, Hollenberg SM, Lindenfeld J, Masoudi FA, McBride PE, Peterson PN, Stevenson LW, Westlake C, 2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure, *Journal of the American College of Cardiology* (2017), doi: 10.1016/j.jacc.2017.04.025

Figure 4: Stages in the Development of HF and Recommended Therapy by Stage

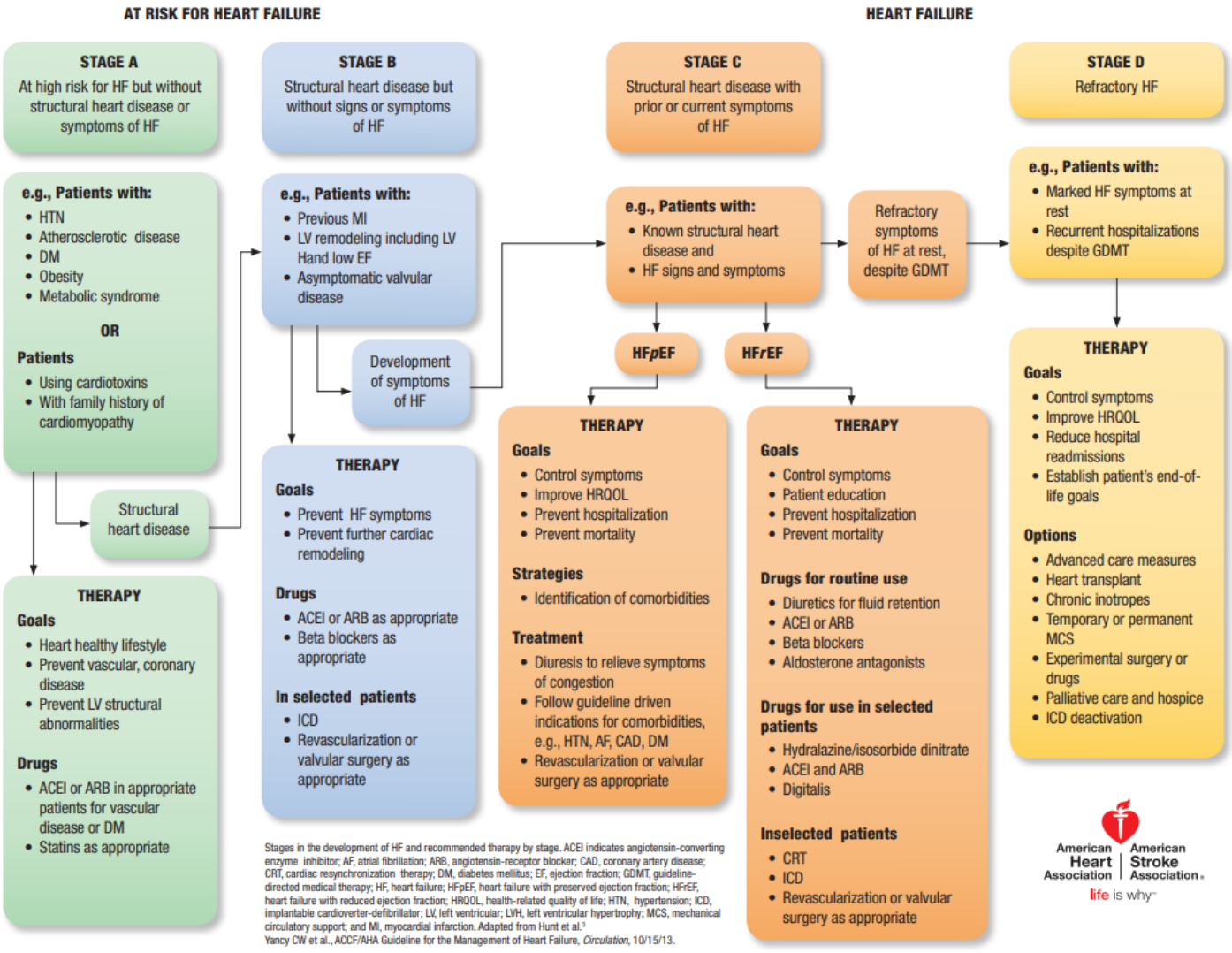

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Figure 5: Laboratory Evaluation for Heart Failure and Selected Alternative Causes

Initial tests

B-type natriuretic peptide level

Calcium and magnesium levels (diuretics, cause of arrhythmia)

Complete blood count (anemia)

Liver function (hepatic congestion, volume overload)

Renal function (renal causes)

Serum electrolyte level (electrolyte imbalance)

Thyroid-stimulating hormone level (thyroid disorders)

Urinalysis (renal causes)

Other tests for alternative causes

Arterial blood gases (hypoxia, pulmonary disease)

Blood cultures (endocarditis, systemic infection)

Human immunodeficiency virus (cardiomyopathy)

Lyme serology (bradycardia/heart block)

Serum ferritin level, transferrin saturation (macrocytic anemia, hemochromatosis)

Thiamine level (deficiency, beriberi, alcoholism)

Troponin and creatine kinase-MB levels (myocardial infarction, myocardial injury)

Tests for comorbid conditions, risk management

A1C level (diabetes mellitus)

Lipid profile (hyperlipidemia)

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