New Therapeutic Choices for Heart Failure: Keeping Up With the Latest Recommendations

Learning Objectives

- Recognize the clinical syndrome of HF and who is at risk
- Incorporate diagnostic testing essential for diagnosis and management of HF in patients at risk
- Describe appropriate pharmacologic and nonpharmacologic strategies for patients across the spectrum of HF, including new treatment strategies when conventional therapies have been exhausted

Definition of HF

- Complex syndrome in which the heart cannot pump blood at a rate commensurate with metabolic needs of the tissues, or can do so only with high pressures
- Results from structural or functional impairment of ventricular filling (diastolic HF, or HFpEF) or ejection (systolic HF, or HFrEF) of blood
- The term “heart failure” preferred over “congestive heart failure”; some patients present without signs or symptoms of volume overload
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Epidemiology of HF

- >5.7 million people affected in United States; >8 million by 2030
- >670,000 new cases/year; 1 in 9 deaths due to HF
- Before age 75, rates markedly higher in blacks than in whites
- Mortality ~50% within 5 years after diagnosis

Hospitalizations for HF

About 1 million hospitalizations per year, usually in patients previously hospitalized for HF

Pathophysiology of HF

Cardiac injury
- Increased load
- Activation of RAA system, SNS, and cytokines
- Reduced systemic perfusion
- Altered gene expression
- Growth and remodeling
- Ischemia and energy depletion
- Direct toxicity
- Apoptosis
- Necrosis
- Cell death

RAA = renin-angiotensin aldosterone; SNS = sympathetic nervous system.
Adapted from Eichhorn EJ, Bristow MR. Circulation. 1996;94:2285-2296.

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### HFrEF and HfP EF

- **HFrEF (systolic HF):** EF ≤ 40%
  - Decreased pumping function of the heart
  - Result: fluid backup in the lungs and HF
- **HfP EF (diastolic HF):**
  - Thickened and stiff heart muscle
  - Result: heart does not fill with blood properly, causing fluid backup in the lungs and HF
- HfP EF more common, but medical therapies proven effective only for patients with HFrEF

### Risk Factors and Comorbidities

<table>
<thead>
<tr>
<th>Risk factors</th>
<th>CAD, hypertension, valvular heart disease, diabetes, cigarette smoking, high/low hematocrit</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac comorbidities</td>
<td>ACS, tachy- or bradyarrhythmia, hypertension, myocarditis, cardiomyopathy, acute PE, acute valvular regurgitation, acute aortic dissection, cardiac tamponade</td>
</tr>
<tr>
<td>Noncardiac comorbidities</td>
<td>Renal dysfunction, respiratory diseases, anemia, arthritis, cognitive dysfunction, depression, COPD, thyroid disorder, polypharmacy, infection, inflammatory markers, sleep apnea</td>
</tr>
<tr>
<td>Patient-related factors</td>
<td>Aging, nonadherence, high salt or fluid intake, alcohol use</td>
</tr>
</tbody>
</table>


### The Continuum of HF

LVH = left ventricular hypertrophy; MI = myocardial infarction; QoL = quality of life. Adapted from Dzau V, Braunwald E. Am Heart J. 1991;121:1244-1263.
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**Powerful Role of Risk Factor Management**

“Aggressive implementation of evidence-based management of risk factors for coronary heart disease should be at the core of HF prevention strategies.”

- Early diagnosis essential to address risk factors, prevent progression to structural, symptomatic HF
- Increased primary care focus on screening for risk factors in at-risk patients
  - Risk factor management: antihypertensive, antiplatelet, lipid-lowering proven to prevent HF
  - Even modest weight loss reduces hypertension, dyslipidemia, and glycemic control
- Control of hypertension could significantly reduce incidence of HF
- Opportunity in primary care to dramatically reduce incidence of HF through guideline-based risk factor control


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**Case Study: Ginny**

- Ginny, a 63-year-old musician, visits her primary care clinician with a 3-week history of fatigue, increasing shortness of breath
- Dyspnea worse with ordinary activity and lying flat; Ginny is comfortable sitting
- Her boots are “pinching” intermittently
- No chest pain
- Frequent abdominal discomfort
- History of hypertension, type 2 diabetes, dyslipidemia, MI (5 years ago; received stent)
- Nondrinker; quit smoking 7 months ago

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**Case Study: Ginny (cont’d)**

- **Physical examination**
  - Height: 5 ft 6 in; weight: 175 lb (body mass index: 28.2 kg/m²)
  - BP (treated): 160/92 mm Hg
  - HR 116 beats/min; respiratory rate 22; temperature 98.4°F
  - 1+ pitting edema ankles; S3 heart sound; bilateral rales lung bases
- **Laboratory findings**
  - Random blood glucose: 139 mg/dL
  - Lipids (treated): total cholesterol 200 mg/dL, low-density lipoprotein cholesterol 135 mg/dL
- **Medications**
  - Furosemide 20 mg/d; metformin 500 mg/d; aspirin 81 mg/d;
  - lisinopril 20 mg/d; atorvastatin 20 mg/d; carvedilol 6.25 mg twice daily
- No arrhythmias; mild cardiomegaly

BP = blood pressure; HR = heart rate.
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Clinical Evaluation of HF: ACCF/AHA Guidelines

- Thorough history and physical examination essential to identify cardiac/noncardiac disorders or behaviors that may cause/accelerate development or progression
- In patients with idiopathic dilated cardiomyopathy, 3-generational family history needed to evaluate presence of familial dilated cardiomyopathy
- Assess volume status/vital signs at each visit
  - Serial assessment of weight, estimates of JVP and presence of peripheral edema/orthopnea

ACCF = American College of Cardiology Foundation; AHA = American Heart Association; JVP = jugular venous pressure.


Diagnostic Challenges in HF

- HF is a syndrome, not a disease; no single test is diagnostic
- Early detection hindered by nonspecific symptoms, presence of comorbidities
- Presentations may change from no symptoms to significant symptoms
- Rate of initial misdiagnosis up to 50% in primary care (clinical vs objective criteria)
- Misdiagnosis high for elderly patients with HFpEF (symptoms mild, absent, attributed to other causes)


History

- Dyspnea (focus of Boston criteria, other diagnostic models?)
  - High sensitivity (>95%); if absent, HF unlikely
  - Progression: dyspnea on exertion → paroxysmal nocturnal dyspnea → orthopnea → dyspnea at rest
  - All symptoms except dyspnea have low sensitivity
- Common: fatigue, weakness, exercise intolerance, edema, nocturia, cough, weight gain
- Less common: cognitive impairment, delirium, nausea
- With progression: gastrointestinal symptoms, but can be misleading; better assessed through history than physical examination
- Evaluation of cardiac/noncardiac risk factors
  - CAD responsible for ≤50% of HF with ↓ LV function

LV = left ventricular.

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Physical Examination

- General appearance (resting dyspnea, cyanosis, cachexia)
- BP and HR (compensation, arrhythmia)
- JVP elevation (very high specificity for LV dysfunction)
- Point of maximal impact displacement (very high specificity)
- Heart sounds (S3 very high specificity)
- Pulmonary examination (wheezing, crackles)
- Lower extremity edema (limited diagnostic value)
- Hepatomegaly (very high specificity)
- Patients with compensated HF often do not have congestion

Differential Diagnosis of HF: Signs and Symptoms

- Myocardial ischemia
- Pulmonary disease
- Sleep-disordered breathing
- Obesity
- Deconditioning
- Malnutrition
- Anemia
- Hepatic failure
- Chronic kidney disease
- Hypoalbuminemia
- Venous stasis
- Depression
- Anxiety, hyperventilation syndromes
- Hyper- or hypothyroidism

Noninvasive Imaging: ACCF/AHA Guidelines

<table>
<thead>
<tr>
<th>Recommendation</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute, new-onset HF: chest radiograph</td>
<td>C</td>
</tr>
<tr>
<td>2-D echo with Doppler for initial evaluation</td>
<td>C</td>
</tr>
<tr>
<td>Repeat EF measurement in patients with HF with significant change in clinical status or who have had treatment that might affect cardiac function or for consideration of device therapy (ICD or biventricular pacemaker)</td>
<td>C</td>
</tr>
<tr>
<td>Noninvasive imaging to detect myocardial ischemia and viability is reasonable in HF and CAD</td>
<td>C</td>
</tr>
<tr>
<td>Viability assessment is reasonable before revascularization in HF patients with CAD</td>
<td>B</td>
</tr>
<tr>
<td>Radionuclide ventriculography or MRI useful for assessing LV EF and volume</td>
<td>C</td>
</tr>
<tr>
<td>MRI reasonable when assessing myocardial infiltration or scar</td>
<td>B</td>
</tr>
<tr>
<td>Do not routinely reassess LV function</td>
<td>B</td>
</tr>
</tbody>
</table>

ICD = implantable cardioverter-defibrillator; LOE = level of evidence; LV EF = left ventricular ejection fraction.

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Chest Radiograph in HF

- May show evidence of HF (eg, cardiomegaly) but not a good independent predictor
- Most useful for detecting alternatives for symptoms (eg, lung disease)
- During treatment, x-ray changes may lag behind clinical improvement

Cardiomegaly and small pleural effusions


Echocardiography

- Essential screening tool if HF suspected
- 2-D echo with Doppler diagnostic standard for HF-EF and HFpEF
- Assesses EF, filling pressures, wall thickness and motion, valve function
- Low rates of false-positives and false-negatives

2-D echo shows normal LV size with hypertrophy


Recommendations for Biomarkers

<table>
<thead>
<tr>
<th>Biomarker/Application</th>
<th>Setting</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natriuretic peptides (BNP, NT-proBNP)</td>
<td>Acute, ambulatory</td>
<td>A</td>
</tr>
<tr>
<td>Diagnosis or exclusion of HF</td>
<td>Ambulatory, acute</td>
<td>A</td>
</tr>
<tr>
<td>Prognosis of HF</td>
<td>Ambulatory, acute</td>
<td>A</td>
</tr>
<tr>
<td>Achieve GDMT</td>
<td>Ambulatory</td>
<td>B</td>
</tr>
<tr>
<td>Guidance of acutely decompensated HF therapy</td>
<td>Acute</td>
<td>C</td>
</tr>
<tr>
<td>Biomarkers of myocardial injury (troponins, creatine phosphokinase)</td>
<td>Acute, ambulatory</td>
<td>A</td>
</tr>
<tr>
<td>Additive risk stratification</td>
<td>Ambulatory</td>
<td>B</td>
</tr>
<tr>
<td>Biomarkers of myocardial fibrosis (ST2, Galectin-3)</td>
<td>Acute</td>
<td>A</td>
</tr>
</tbody>
</table>

BNP = brain natriuretic peptide; GDMT = guideline-directed medical therapy; NT-proBNP = N-terminal prohormone BNP.

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**Laboratory Testing: BNP**

- BNP and NT-proBNP increase when cardiac myocytes are under strain
- Normal BNP values rule out HF
- BNP important measure when cost/access limits use of echo
- May reveal HF with/without systolic dysfunction but does not distinguish HFrEF from HFpEF
- Increases with severity of HF
- Obesity, insulin resistance may lower BNP
- High values lack specificity; clinical correlates required
- Only ~40% of primary care practices use BNP for HF diagnosis


- **Causes of Elevated Natriuretic Peptide Levels**
  - **Cardiac**
    - HF, including RV syndromes
    - ACS
    - Heart muscle disease, including LVH
    - Valvular heart disease
    - Pericardial disease
    - Atrial fibrillation
    - Myocarditis
    - Cardiac surgery
    - Cardioversion
  - **Noncardiac**
    - Advancing age
    - Anemia
    - Renal failure
    - Pulmonary causes: obstructive sleep apnea, severe pneumonia, pulmonary hypertension
    - Critical illness
    - Bacterial sepsis
    - Severe burns
    - Toxic-metabolic insults, including cancer chemotherapy and envenomation


- **Classification of HF**

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Goals of HF Management

- Improve symptoms and QoL
- Prolong life by slowing disease progression
- Relieve circulatory congestion
- Increase tissue perfusion
- Reduce vasoconstriction
- Inhibit activation of the RAA system and SNS
- Inhibit progressive ventricular enlargement or remodeling
- Optimize patient education, adherence, and referral
- Recognize and refer patients who will benefit from specialized care, including ventricular assist device and heart transplant

Barriers to Management Goals

- Gaps in outpatient and hospital care perpetuate cycle of HF exacerbations
- Patients at highest risk of death least likely to receive recommended therapies
- Treatment, patient education gaps evident even in cardiology practices

Clinical Course of HF With Type and Intensity of Management

- Transition to advanced HF
- Oral therapies failing
- A time for many major decisions
- Consider MCS and/or transplantation, if eligible
- Consider transition of care plan to one dominated by palliative approach, which may involve formal hospice
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Treatment of Stage A HF

- Treat hypertension and dyslipidemia using contemporary guidelines
- Other conditions/agents that may lead to or contribute to HF (eg, obesity, diabetes, tobacco use, and known cardiotoxic agents) should be controlled or avoided

Therapies for HF: Stages B-D/NYHA Classes I-IV

<table>
<thead>
<tr>
<th>Severity of HF</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage B, Class I</td>
<td>ACE inhibitor or ARB</td>
</tr>
<tr>
<td>Stage C, Class IVA</td>
<td>ACE inhibitor and/or ARB (I-IV)</td>
</tr>
<tr>
<td>Stage C, Class IVA</td>
<td>Diuretic (II-IV)</td>
</tr>
<tr>
<td>Stage C, Class IVA</td>
<td>ivabradine (III)</td>
</tr>
<tr>
<td>Stage C, Class IVA</td>
<td>Aldosterone antagonist (III)</td>
</tr>
<tr>
<td>Stage C, Class IVA</td>
<td>Isosorbide dinitrate/hydralazine (II-IV)</td>
</tr>
<tr>
<td>Stage C, Class IVA</td>
<td>Digoxin (II-IV)</td>
</tr>
<tr>
<td>Stage C, Class IVA</td>
<td>CRT, ICD (II-IV)</td>
</tr>
<tr>
<td>Stage D, Class IV</td>
<td>All Stage C treatments</td>
</tr>
<tr>
<td>Stage D, Class IV</td>
<td>Inotropic agents, vasodilators</td>
</tr>
<tr>
<td>Stage D, Class IV</td>
<td>Experimental drugs/surgery</td>
</tr>
<tr>
<td>Stage D, Class IV</td>
<td>ICD deactivation</td>
</tr>
<tr>
<td>Stage D, Class IV</td>
<td>Transplant</td>
</tr>
<tr>
<td>Stage D, Class IV</td>
<td>Palliative care, hospice</td>
</tr>
</tbody>
</table>

ARNI = angiotensin receptor neprilysin inhibitor; CRT = cardiac resynchronization therapy

Oral Drug Therapies for HF

- ACE inhibitors – captopril, lisinopril, ramipril, enalapril
- ARBs – losartan, valsartan, candesartan
- Aldosterone antagonists – spironolactone
- β-blockers – bisoprolol, carvedilol, metoprolol
- Digitalis – digoxin (HF/EF)
- Diuretics – furosemide, hydrochlorothiazide
- Vasodilators – isosorbide dinitrate/hydralazine
- Statins in patients with recent/remote history of CAD
- ARNI (sacubitril/valsartan) – in place of ACE or ARB
- Ivabradine – with maximally tolerated β-blocker
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### Incremental Benefit of HF Therapies

<table>
<thead>
<tr>
<th>HF Therapies</th>
<th>Change in Odds of 24-Month Mortality (%)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>β-blocker</td>
<td>-39% (−28% to −49%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-blocker + ACEI/ARB</td>
<td>-63% (−54% to −71%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-blocker + ACEI/ARB + ICD</td>
<td>-76% (−68% to −81%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-blocker + ACEI/ARB + ICD + HF Education</td>
<td>-81% (−75% to −86%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-blocker + ACEI/ARB + ICD + HF Education + Anticoagulation for AF</td>
<td>-81% (−72% to −87%)</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>β-blocker + ACEI/ARB + ICD + HF Education + Anticoagulation for AF + CRT</td>
<td>-83% (−77% to −88%)</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

- ACEI = ACE inhibitor; ARB = angiotensin receptor blocker; CRT = cardiac resynchronization therapy; ICD = implantable cardioverter-defibrillator.

### Treatment of Stage B HF

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>History of MI and ↓EF: ACE inhibitors or ARBs</td>
<td>A</td>
</tr>
<tr>
<td>History of MI and ↓EF: β-blockers</td>
<td>B</td>
</tr>
<tr>
<td>Post-MI: statins</td>
<td>A</td>
</tr>
<tr>
<td>Hypertension: GDMT to prevent symptomatic HF</td>
<td>A</td>
</tr>
<tr>
<td>All patients with ↓EF: ACE inhibitors</td>
<td>A</td>
</tr>
<tr>
<td>All patients with ↓EF: β-blockers</td>
<td>C</td>
</tr>
<tr>
<td>ICD reasonable with asymptomatic ischemic cardiomyopathy ≥40 days post-MI, LVEF ≤30%, with GDMT</td>
<td>B</td>
</tr>
</tbody>
</table>

**Caution:** nondihydropyridine calcium blocker may be harmful in patients with low LVEF.


### Treatment of Stage C HFpEF

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control hypertension according to published clinical practice guidelines</td>
<td>B</td>
</tr>
<tr>
<td>Diuretics to relieve symptoms of volume overload</td>
<td>C</td>
</tr>
<tr>
<td>Coronary revascularization for patients with CAD and angina or myocardial ischemia despite GDMT</td>
<td>C</td>
</tr>
<tr>
<td>Management of AF according to clinical practice guidelines to improve symptomatic HF</td>
<td>C</td>
</tr>
<tr>
<td>β-blockers, ACE inhibitors, and ARBs for hypertension</td>
<td>B</td>
</tr>
<tr>
<td>ARBs may be considered to decrease risk of hospitalization</td>
<td>C</td>
</tr>
<tr>
<td>Nutritional supplementation not recommended</td>
<td>C</td>
</tr>
</tbody>
</table>

Lacking direct clinical trial evidence to drive HFpEF management, guidelines are largely consensus-based.

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Treatment of Stage C HFrEF

- **HFrEF Stage C NYHA Class II-IV**
  - For all volume overload, diuretics
  - For African Americans with systolic dysfunction (Stage C): Hydralazine-nitrates
  - For patients with NYHA class III-IV, or symptomatic left ventricular ejection fraction ≤30%, aldosterone antagonist

  *Data are limited for use of hydralazine-nitrates with ARNI. Careful monitoring of potassium advised in patients taking an ARNI with aldosterone antagonist.


Case Study Conclusion: Ginny's Uncertain Future

- Ginny was prescribed the following:
  - Increased furosemide dose (40 mg) for diuresis
  - Carvedilol, target dose 25 mg twice daily
  - Increased doses of metformin (1000 mg), atorvastatin (40 mg), lisinopril (40 mg/d)

- At 7 months, Ginny was hospitalized for HF symptoms after a 5-day lapse in adherence (did not refill medications)

- At 24 months, despite adherence, Ginny was hospitalized again for HF symptoms including dyspnea at rest (NYHA IV; Stage C; EF 34%)
  - Received intravenous therapy to counter bowel edema/malabsorption of medications

- Referred to specialist for evaluation and treatment; lisinopril was held for 36 hours and ARNI therapy was initiated

New Medication Options in HF

Both ivabradine and sacubitril/valsartan (ARNI) evaluated in patients with NYHA class II-IV HF and ↓ EF

- **Ivabradine**
  - Inhibits the If (“funny” (pacemaker) current in the sinoatrial node to decrease heart rate but does not impact contractility
  - Approval based on placebo-controlled SHIFT trial

- **Sacubitril/valsartan**
  - Neprilysin (enzyme) inhibitor + ARB; increases peptides (eg, natriuretic peptide) usually degraded by neprilysin, to counter maladaptive mechanisms
  - Approval based on PARADIGM-HF trial vs enalapril

ARNI = angiotensin receptor-neprilysin inhibitor.
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2016 ACC/AHA/HFSA Focused Update: Use of Newer Drugs for HF

<table>
<thead>
<tr>
<th>Sacubitril-Valsalan (ARNI)</th>
<th>Ivabradine</th>
</tr>
</thead>
<tbody>
<tr>
<td>• In patients with chronic symptomatic HFrEF NYHA class II-III who tolerate ACE inhibitor or ARB, replacement by ARNI is recommended to further reduce morbidity and mortality</td>
<td>Can reduce HF hospitalization in patients with NYHA class II-III stable chronic HFrEF (LVEF ≤35%) who are receiving GDMT, including maximally tolerated β-blocker, and who are in sinus rhythm with heart rate ≥70 bpm</td>
</tr>
</tbody>
</table>

ESC = European Society of Heart Failure; HFSA = Heart Failure Society of America


SHIFT: Cardiovascular Death or HF Hospitalization With Ivabradine

Patients achieving HR <60 bpm or with >10 bpm reduction have best prognosis

<table>
<thead>
<tr>
<th>Placebo (n = 3290)</th>
<th>Ivabradine (n = 3268)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR = 0.82 (0.75-0.90)</td>
<td>P &lt; .0001</td>
</tr>
</tbody>
</table>


SHIFT: Adverse Events

• Symptomatic/asymptomatic bradycardia more common in ivabradine group (11%) than in placebo group (2%) (both P < .0001); led to withdrawal from study in ≤1% in either group
• Visual symptoms (phosphenes) in 3% of patients taking ivabradine, 1% taking placebo (P < .0001); led to withdrawal in <1% in either group
• No relevant differences in laboratory values

Ivabradine: Precautions

- Fetal toxicity: women should use effective contraception
- Monitor patients for atrial fibrillation
- Monitor heart rate decreases and bradycardia symptoms during treatment
- Not recommended for patients with second-degree atrioventricular block


PARADIGM-HF: Cardiovascular Death or HF Hospitalization With Sacubitril/Valsartan

PARADIGM-HF: Adverse Events

- Fewer patients in sacubitril/valsartan group than in enalapril group stopped study medication due to an adverse event (10.7% vs 12.3%, P = .03) or because of renal impairment (0.7% vs 1.4%, P = .002)
- Symptomatic hypotension more common with sacubitril/valsartan (14%) than with enalapril (9.2%) (P <.001)
- No significant difference in rate of angioedema

**Sacubitril/Valsartan: Precautions**

- Do not use in patients with angioedema
  - Patients with a history of angioedema and use of an ACE inhibitor should be treated with an ARB
- Discontinue ACE inhibitor for $\geq 36$ hours before initiating therapy with sacubitril/valsartan

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**Strategies for Optimal Management of HF in Primary Care: Nonpharmacologic Interventions**

- Patient and family education on self-care
- Regular, suitable physical activity
- Sodium restriction if congestive symptoms
- Cardiac rehabilitation for clinically stable patients
- Smoking cessation, weight loss
- Enhanced patient education—referral to HF management program (60 minutes, qualified educator or AHA interactive workbook)
  - [https://www.heart.org/idc/groups/heart-public/@wcm/@hcm/@gwtg/documents/downloadable/ucm_467882.pdf](https://www.heart.org/idc/groups/heart-public/@wcm/@hcm/@gwtg/documents/downloadable/ucm_467882.pdf)

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**Strategies for Optimal Management of HF in Primary Care**

- Early re-evaluation crucial, especially for higher-risk patients; opportunity for:
  - Reassessment, including laboratory
  - Medication review, titration, assessment of adherence, absorption of drug
- Up-titrate drugs in small increments, with monitoring of vital signs before and after
  - Provide reassurance on transient symptoms
  - Discourage discontinuation without consult
- Carefully review doses of medications for risk factor control and need for dose increases
Perform screening and guideline-based risk factor control to prevent progression to HF in at-risk patients
Identify potential causes of progression in patients presenting with HF through thorough history, physical examination, and follow-up
Use 2-D echo with Doppler to diagnose systolic and diastolic dysfunction
Use guideline-based treatment for all patients with chronic heart failure
Identify candidates who could benefit from therapy with sacubitril-valsartan or ivabradine
Refer patients to a comprehensive HF management program to improve outcomes