ICSI Institute for Clinical Systems Improvement

Health Care Guideline

Heart Failure in Adults

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ICSI
Institute for Clinical Systems Improvement

Health Care Guideline:
Heart Failure in Adults

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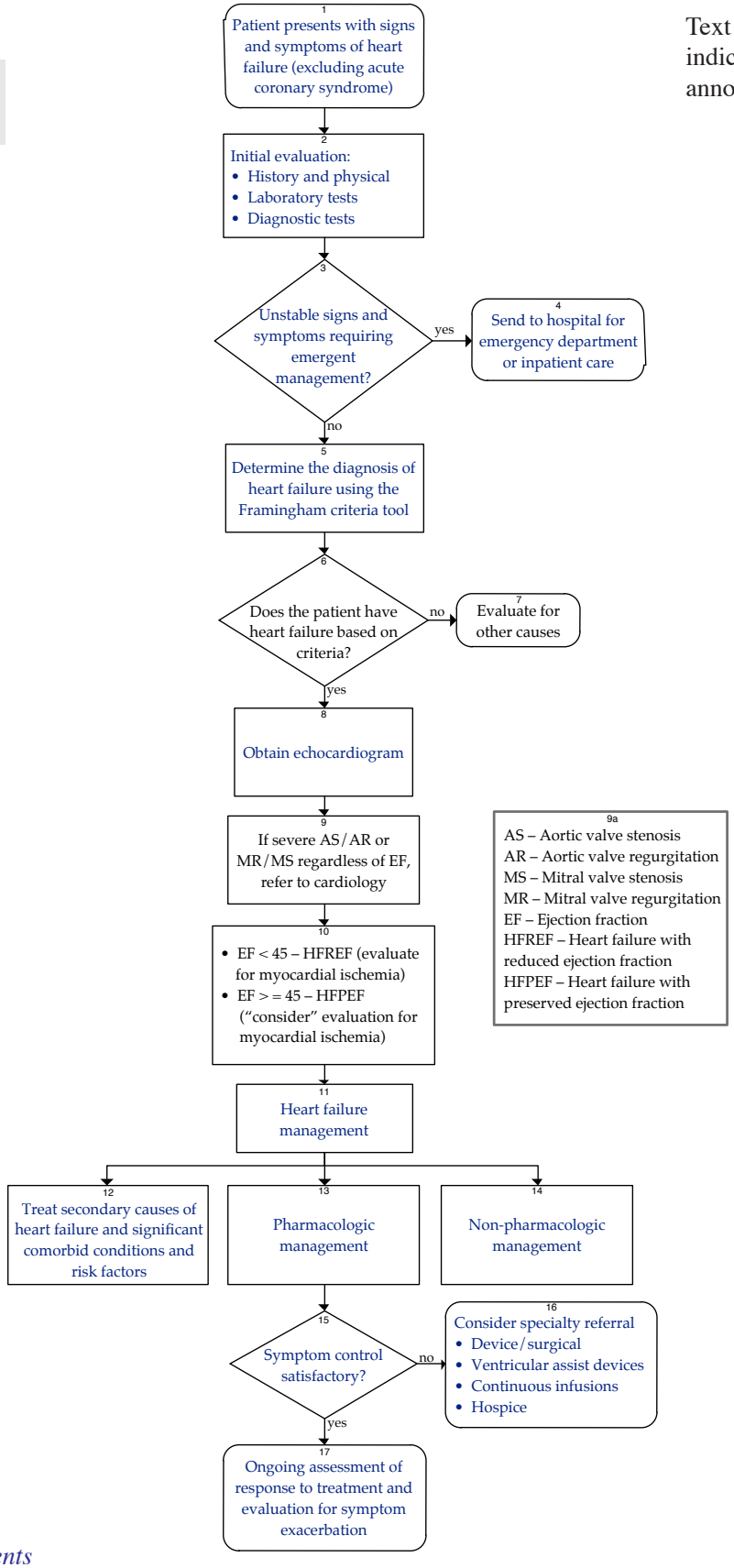
Text in blue in this algorithm indicates a linked corresponding annotation.



Return to Table of Contents

Table of Contents

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Evidence Grading System

Literature Search

A consistent and defined process is used for literature search and review for the development and revision of ICSI guidelines. The literature search was divided into two stages to identify systematic reviews, (stage I) and randomized controlled trials, meta-analysis and other literature (stage II). Literature search terms used for this revision include brain natriuretic peptide, Framingham criteria for heart failure, preventable hospital readmission in heart failure, spironolactone, observation units for heart failure patients and Society of Chest Pain and the American College of Cardiologists guidelines from January 2011 through January 2013.

GRADE Methodology

Following a review of several evidence rating and recommendation writing systems, ICSI has made a decision to transition to the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system.

GRADE has advantages over other systems including the current system used by ICSI. Advantages include:

- developed by a widely representative group of international guideline developers;
- explicit and comprehensive criteria for downgrading and upgrading quality of evidence ratings;
- clear separation between quality of evidence and strength of recommendations that includes a transparent process of moving from evidence evaluation to recommendations;
- clear, pragmatic interpretations of strong versus weak recommendations for clinicians, patients and policy-makers;
- explicit acknowledgement of values and preferences; and
- explicit evaluation of the importance of outcomes of alternative management strategies.

This document is in transition to the GRADE methodology

Transition steps incorporating GRADE methodology for this document include the following:

- Priority placed upon available Systematic Reviews in literature searches.
- All existing Class A (RCTs) studies have been considered as high quality evidence unless specified differently by a work group member.
- All existing Class B, C and D studies have been considered as low quality evidence unless specified differently by a work group member.
- All existing Class M and R studies are identified by study design versus assigning a quality of evidence. Refer to Crosswalk between ICSI Evidence Grading System and GRADE.
- All new literature considered by the work group for this revision has been assessed using GRADE methodology.

Return to Table of Contents

Crosswalk between ICSI Evidence Grading System and GRADE

Design of Study Current ICSI System		ICSI GRADE System
Class A:	Randomized, controlled trial	**High**, if no limitation **Moderate**, if some limitations **Low**, if serious limitations
Class B:	[observational] Cohort study	 **High,** if well done with large effect **Moderate,** if well done with effect **Low,** most studies
Class C:	[observational] Non-randomized trial with concurrent or historical controls Case-control study Population-based descriptive study Study of sensitivity and specificity of a diagnostic test	 **Low** **Low** **Low***
* Following individual study review, may be elevated to Moderate or High depending upon study design		
Class D:	[observational] Cross-sectional study Case series Case report	 **Low**
Class M:	Meta-analysis Systematic review Decision analysis Cost-effectiveness analysis	**Meta-analysis** **Systematic Review** **Decision Analysis** **Cost-effectiveness Analysis**
Class R:	Consensus statement Consensus report Narrative review Guideline	**Low** **Low** **Low** **Guideline**
Class X:	Medical opinion	**Low**
Class Not Assignable		**Class Not Assignable**

Evidence Definitions:

High Quality Evidence = Further research is very unlikely to change our confidence in the estimate of effect.

Moderate Quality Evidence = Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.

Low Quality Evidence = Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate or any estimate of effect is very uncertain.

Return to Table of Contents

Foreword

Introduction

Heart failure is the term to describe the condition of the heart's failure to meet the body's metabolic demands with the symptomatic result of dyspnea, fatigue and cough. It is key to understand that the symptoms may be due to systolic dysfunction emanating from the right or left side of the heart, or may occur with preserved systolic function with symptoms due to abnormal diastolic function of the heart. This guideline delineates how to establish the etiology of heart failure and begin treatment.

Heart failure is a major health problem in the United States, and the incidence of the disease is increasing. This is primarily due to the older population; in age 20-39, the incidence of heart failure is 0.3% of the population in men and 0.2% of the population in women. In the ages 40s and 50s, the incidence is 2% in men and 1.5% in women. In the 60-79 age group, the incidence is 7.2% in men and 5.2% in women. However, once reaching age 80, the incidence of heart failure is higher in women, with 11.6% of men and 12.4% of women. (National Health and Nutrition Examination Survey 1999 to 2004 [NHANES] Data 1999-2004 from the NHLBI on the Web at http://www.cdc.gov/nchs/nhanes.htm).

The overall estimated 2004 prevalence of heart failure in adults age 20 and older in the United States was 5.2 million, with it being equally distributed among men and women. Seventy-five percent of heart failure cases have antecedent hypertension in that the lifetime risk for heart failure doubles for people with blood pressure greater than 160/90 versus those with blood pressure less than 140/90. A community-based cohort study conducted in Olmsted County, Minnesota, showed that the incidence of heart failure (ICD9-428) has not declined during the past two decades, but survival after onset has increased overall, with less improvement among women and elderly persons *(Roger, 2004 [Low Quality Evidence])*.

The outpatient treatment for heart failure has improved dramatically with the advent of neurohormonal and device approaches, patient education, and care or disease management strategies, in addition to traditional diuretic, digoxin and vasodilator therapy. Inpatient treatment has improved in many respects due to the aforementioned medications, improved imaging and physiologic monitoring, and early intervention for ischemic etiologies.

Although heart failure is generally regarded as a hemodynamic disorder, there is a poor correlation between measures of cardiac performance and the symptoms produced by the disease. Patients with a very low ejection fraction (EF) may be asymptomatic, whereas patients with preserved left ventricular ejection fraction (LVEF) may have severe disability. The apparent discordance between EF and the degree of functional impairment is not well understood but may be explained in part by alterations in ventricular distensability, peripheral vascular resistance, valvular regurgitation, pericardial restraint, cardiac rhythm, conduction abnormalities and right ventricular function. As patients with reduced EF may have diastolic dysfunction in addition to systolic dysfunction, and patients with preserved EF may have both systolic and diastolic dysfunction, the American College of Cardiology/American Heart Association (ACC/AHA) prefer the terminology heart failure with preserved ejection fraction (HFPEF) and heart failure with reduced ejection fraction (HFREF).

Heart failure occurs at any level of left ventricular ejection fraction (LVEF). Most intervention trials have shown significant benefits in trials with HFREF but have also shown an unexplained resistance to therapy (especially to renin-angiotensin-aldosterone system inhibition) in HFPEF. Depending on the criteria used to delineate heart failure and the accepted threshold for defining preserved LVEF, it is estimated that as many as 20% to 60% of patients with heart failure have a relatively (or near) normal LVEF and, in the absence of valvular disease, are believed to have reduced ventricular compliance as a major contributor to the clinical syndrome.

Return to Table of Contents

In the Framingham Heart Study, patients presenting with heart failure were studied *(Lee, 2009 [Low Quality Evidence])*. They were divided into two groups with a cutoff LVEF of < 45%. The survival data did not differ between the two groups. Large registries have established that the distribution of LVEF in heart failure is unimodal. LVEF is a powerful prognostic parameter – when decreased – and useful in daily clinical practice to obtain a first impression of global ventricular pump performance during disease staging. Until further data is available, heart failure will continue to be separated into two groups; heart failure with preserved ejection fraction (HFPEF) or heart failure with reduced ejection fraction (HFREF). There is robust evidence to support current management recommendations for HFREF.

(Solomon, 2005 [Moderate Quality Evidence]; Hogg, 2004 [Low Quality Evidence]; Hunt, 2005 [Guideline])

Other related guidelines for heart failure include the ACC/AHA Congestive Heart Failure guidelines, Pacemaker guidelines, and the Heart Failure Society of America guidelines.

Return to Table of Contents

Scope and Target Population

The scope and target population of this document focuses on the adult patient age 18 years and older with suspected heart failure. This includes the diagnosis and outpatient management of the patient. Consideration will also be made to reducing all-cause readmission rates to the hospital for patients who had been previously hospitalized with an exacerbation of heart failure.

Return to Table of Contents

Aims

1. Decrease the readmission rate for patients 18 years and older with heart failure diagnosis, within 30 days of discharge following hospitalization for heart failure. *(Annotation #14)*
2. Increase the rate of heart failure patients 18 years and older who receive optimum evidence-based pharmacologic treatment with heart failure. *(Annotation #13)*
3. Improve the use of diagnostic testing in order to identify and then appropriately treat adult patients with heart failure. *(Annotation #2)*
4. Increase the rate of heart failure patients age 18 years or older who have comprehensive patient education and follow-up care. *(Annotation #14)*

Return to Table of Contents

Clinical Highlights

- Evaluate patients presenting with heart failure for exacerbating and underlying causes, including coronary artery disease, hypertension, valvular disease and other cardiac and non-cardiac causes. *(Annotation #2; Aim #3)*
- Studies show that the distinction between systolic dysfunction and preserved systolic function is important, because the choice of therapy may be quite different and some therapies for systolic dysfunction may be detrimental if used to treat preserved systolic function. *(Annotation #2; Aim #3)*
- Daily weights are critical for managing heart failure and early detection of increases in fluid retention. Patients should call their clinician about a two-pound or greater weight gain overnight or a five-pound or greater weight gain in a week. Patients can expect the clinician to assess symptoms, adjust diuretics

Return to Table of Contents

if appropriate, discuss dietary sodium compliance/restriction, review treatment plan, and recommend appropriate level of care (office visit, ED, etc.) *(Annotation #14; Aim #4)*

- Unless specific contraindications exist, treat all patients with beta-blockers, starting with a low dose and titrating upward. *(Annotation #13; Aim #2)*
- Treat all patients with left ventricular systolic dysfunction with ACE inhibitors (or ARBs if intolerant) unless specific contraindications exist. *(Annotation #13; Aim #2)*
- Consider early specialty referral for patients with ischemia or those who are refractory despite optimal medical therapy. *(Annotation #10; Aim #3)*
- Brain natriuretic peptide (BNP) and N-terminal pro-B-type natriuretic peptide (NTproBNP) are useful in the diagnosis and prognosis of heart failure in patients with dyspnea of unknown etiology. *(Annotation #2; Aim #3)*
- For patients self-described as African Americans who have moderate-to-severe symptoms on optimal therapy with ACE inhibitors, beta-blockers and diuretics, the combination of hydralazine and nitrates is recommended because the combination has resulted in significant benefit to the group in randomized controlled trials. *(Annotation #13; Aim #2)*

Return to Table of Contents

Implementation Recommendation Highlights

The following system changes were identified by the guideline work group as key strategies for health care systems to incorporate in support of the implementation of this guideline.

- Develop a process that will allow primary care clinicians to identity patients who have been admitted or readmitted to the hospital with a diagnosis of heart failure.
- Emphasize patient self-management strategies. These may include heart failure education and other actions designed to sustain engagement of patients with their heart failure care.
- Develop a process to provide education to the patient and/or caregiver in the area of:
  - diet
  - weight monitoring (to include: clinician should be contacted about a two-pound or greater weight gain overnight or a five-pound or greater weight gain during the week)
  - activity level
  - medications
  - the importance of follow-up appointments
  - what to do if symptoms worsen
- Develop a process for timely, early specialty referral for patients with ischemia or those who are refractory despite optimal medical therapy.

Return to Table of Contents

Related ICSI Scientific Documents

Guidelines

- Antithrombotic Therapy Supplement
- Diagnosis and Treatment of Chest Pain and Acute Coronary Syndrome (ACS)
- Hypertension Diagnosis and Management
- Lipid Management in Adults
- Major Depression in Adults in Primary Care
- Palliative Care
- Stable Coronary Artery Disease
- Venous Thromboembolism Diagnosis and Treatment
- Venous Thromboembolism Prophylaxis

Return to Table of Contents

Definition

Clinician – All health care professionals whose practice is based on interaction with and/or treatment of a patient.

Return to Table of Contents

Algorithm Annotations

1. Patient Presents with Signs and Symptoms of Heart Failure (Excluding Acute Coronary Syndrome)

Signs and Symptoms of Congestion:

- Dyspnea
- Orthopnea
- Paroxysmal nocturnal dyspnea (PND)
- Cough (recumbent or exertional)
- Abdominal or epigastric discomfort
- Abdominal bloating (ascites)
- Early satiety
- Hemoptysis, frothy or pink-tinged sputum
- Pedal/leg swelling
- Weight gain (rapid)
- Sleep disturbances (anxiety or air hunger)
- Chest tightness or discomfort
- Unexplained confusion, altered mental status, or fatigue
- Nausea or anorexia
- Dependent edema

Signs and Symptoms of Poor Perfusion/Low Cardiac Output:

- Easy fatigability
- Poor energy level or endurance
- Decreased exercise tolerance
- Cachexia
- Muscle wasting or weakness
- Nausea or anorexia
- Early satiety
- Weight loss, unexplained
- Malaise
- Impaired concentration or memory
- Sleep disturbance
- Altered mentation (somnolence, confusion)
- Resting tachycardia
- Daytime oliguria with recumbent nocturia
- Cool or vasoconstricted extremities
- Cheyne-Stokes respiration (with or without apnea)

See Appendix A for the New York Heart Association Classification and ACC/AHA Staging System Comparison.

Return to Algorithm *Return to Table of Contents*

2. Initial Evaluation/History and Physical/Laboratory Tests/Diagnostic Tests

Recommendations:

- Clinicians must perform an initial evaluation, to confirm a diagnosis of heart failure (HF) and identify etiology/precipitating factor(s). The diagnosis of heart failure should not be a single diagnosis.
- Consider consultation with cardiology during the initial evaluation and any time that it is felt appropriate in the ongoing management of heart failure patients.

Return to Algorithm *Return to Table of Contents*

Questions to Determine Severity:

A. History

Presenting symptoms

- Dyspnea/PND/orthopnea
- Recent weight gain
- Chest pain
- Palpitations
- Blood loss/causes of anemia
- Recent fevers/viral infection
- Cough/sputum production
- Claudication
- Exercise tolerance
- Fatigue
- Edema/ascites
- Color changes

Past medical history

- History of congestive heart failure (HF)
- History of myocardial infarction (MI)
- Hypertension/smoking/diabetes/hyperlipidemia
- History/risk factors for thromboembolic disease
- History of thyroid dysfunction
- Recently postpartum (within the first month)
- History of snoring/sleep apnea
- Blunt chest injury
- Rheumatic fever
- HIV
- Bacterial endocarditis
- Claudication
- Screen for depression
- Foreign travel

Family history

- Screen for family history of ischemic heart disease, HF, congenital heart disease, risk factors for athero-sclerotic cardiovascular disease (ASCVD) and HF

Social history

- Smoking
- Alcohol use/abuse screen
- Drug abuse

Dietary history

- Salt and daily fluid intake
- Balanced diet

B. Physical Exam

- Vital signs, including weight and height
- Diaphoresis
- Diminished peripheral pulse or bruit
- Skin color: cyanosis, pallor, jaundice
- Lower extremity edema in the absence of venous insufficiency

Return to Algorithm *Return to Table of Contents*

- Elevated jugular venous pressure, positive hepato-jugular reflux
- Heart rate: tachycardia, bradycardia/arrhythmias
- Left lateral displacement of point of maximal impulse (PMI)
- Heart sounds: S3, S4 or murmur
- Lungs: labored breathing, rales above the lower 25% of the lung that do not clear with cough
- Abdomen: large, pulsatile, or tender liver or ascites

(Jessup, 2009 [Guideline]; Hunt, 2005 [Guideline])

C. Initial Laboratory Evaluation

- **Initial**
  - Complete blood count
  - Electrolytes (Na^+, K^+) and Cl^-, bicarb, $Ca^{++,}$ MG^{++} (if on diuretics)
  - Renal function (BUN, Cr)
  - Liver function (AST, ALT, Alk phos, Bili, T Prot, Alb)
  - Urinalysis
  - Sensitive TSH (sTSH)
  - PT/INR
  - NT-proBNP or BNP
  - Test for myocardial injury: troponin
- **Other Laboratory Evaluation**
  - Ferritin/iron/TIBC/macrocytic anemias
  - Lipid profile
  - Blood culture (if endocarditis suspected)
  - Lymes serology (if suspect bradycardia/heart block)
  - Connective tissue disease workup
  - HIV

(Hunt, 2005 [Guideline])

Role of Brain Natriuretic Peptide (BNP)/NTproBNP in the Diagnosis and Management of Heart Failure

Brain natriuretic peptide (BNP) and NTproBNP assays have been found useful in the correct diagnosis of patients with dyspnea, especially when the patient has a history of pulmonary disease and/or cardiac disease. Since BNP and NTproBNP concentrations correlate positively with cardiac filling pressures, measurement of a low concentration make it unlikely that dyspnea is due to cardiac dysfunction.

In general, a BNP less than 100 pg/mL helps exclude a cardiac cause of dyspnea. A BNP greater than 500 pg/mL is highly specific and prognostic for short-term increased mortality risk. For those patients

between 100 pg/mL and 500 pg/mL, which was about 26% of subjects in a BNP trial, two-thirds had heart failure and one-third did not. The normal ranges of BNP and NTproBNP are age and sex dependent. In patients with chronic renal insufficiency, BNP levels may not be indicative of heart failure. In acute renal dysfunction, BNP measurement is not diagnostic.

The ICON study established the sensitivity and specificity for the use of NT-proBNP in the diagnosis of acute heart failure. Using NT-proBNP values above 450 pg/ml for ages less than 50, above 900 pg/ml for ages 50-70, and above 1,800 pg/ml for those patients above age 75 yielded a sensitivity of 90% and a specificity of 84%. In addition the study found that having a NT-proBNP less than 300 pg/ml had a negative predictive value of 98% in adult patients of all ages *(Januzzi, 2006 [Low Quality Evidence])*.

The use of BNP/NTproBNP as a risk stratification technique has shown to reduce the length of hospitalization *(Mueller, 2004 [Moderate Quality Evidence])*. In patients with and without heart failure, BNP levels are inversely related to BMI *(McCord, 2004 [Low Quality Evidence])*. Also, the use of BNP or NTproBNP in conjunction with troponin has been shown to have significant incremental predictability on in-hospital mortality *(Fonarow, 2008 [Low Quality Evidence])*.

Persistent elevation of plasma BNP and NTproBNP despite optimum medical therapy also has prognostic significance. The ICON study found that those patients with a severely elevated NT-proBNP had a higher risk of death at 76 days *(Januzzi, 2006 [Low Quality Evidence])*. In hospitalized patients, persistent elevation of BNP/NTproBNP prior to discharge from the hospital is predictive of risk of death or readmission *(Hartmann, 2004 [Low Quality Evidence]; Logeart, 2004 [Low Quality Evidence])*

Cardiac Troponins in Heart Failure

Detectable circulating levels of troponins in the general population is rare. Multiple studies have studied the prevalence of elevation of cardiac troponins in patients with heart failure, both in the compensated and decompensated states. The prevalence appears to vary depending on the assay method and the heart failure population studied. In general, cardiac troponin levels are higher in patients with more advanced disease as in patients with decompensated heart failure.

Newer methods of cardiac troponin (cTn) assay are more sensitive, and detectable levels of cTn are found in most patients with chronic heart failure. Multiple mechanisms, in addition to cardiac ischemia, have been proposed for the elevation of cTn in this population. The end result of the different mechanisms results in worsening cardiac dysfunction and progression of heart failure.

Regardless of the heart failure population studied or the assay method, multiple studies have demonstrated the consistent association between cTn elevation and worse outcomes. In patients with acute heart failure, the ADHERE study *(Fonarow, 2008 [Low Quality Evidence])* demonstrated a marked increase in in-hospital mortality (8.0% vs. 2.7%, p_0.001). In the EFFECT (Enhanced Feedback for Effective Cardiac Treatment) study, elevated cardiac troponin levels were associated with worse mortality. In the Val-HEFT study, ambulatory patients with heart failure, higher levels of cTn was associated with worse outcomes, with incrementally increased risk of death. There appears to be some correlation with the cTn levels and the level of risk for mortality.

Elevated cTn levels, therefore, identify a cohort of patients in the heart failure population who are at higher risk. Some of these patients may have ischemia as a factor and may be candidates for revascularization. Circulating cTn levels may also provide insight into the transition from a chronic compensated state to acute decompensated heart failure.

We recommend an initial determination of cTn for patients presenting to the hospital with acute heart failure. Troponin assessment can be used for immediate risk stratification and may also suggest ACS as the underlying etiology, depending on other presenting features. In patients with initially elevated cTn levels, a repeat cTn measurement within 6 to 12 hours can help determine whether or not the kinetics of cTn change are more

consistent with either ACS or acute decompensated heart failure. In ambulatory patients with heart failure, cTn measurement is a reasonable prognostic indicator. Persistently elevated cTn values in chronic heart failure patients should lead to consideration of more intensive medical therapy, as well as an evaluation for ischemic heart disease (if not already performed).

(Kociol, 2010 [Low Quality Evidence])

D. Diagnostic Tests

- Electrocardiogram
- Chest radiograph
- Assessment of ventricular function (echocardiogram, radionuclide ventriculography)
  - It is reasonable to reassess ejection fraction if patient is clinically decompensated or after patient has been titrated up to target doses of beta-blockers and ACE inhibitors.
- Ischemia evaluation in patients with CAD risk factors (stress test, angiography). Refer to the ICSI Diagnosis and Treatment of Chest Pain and Acute Coronary Syndrome (ACS) guideline.

An electrocardiogram and a chest radiograph are fundamental parts of the initial evaluation for heart failure. In addition, the objective evaluation of ventricular performance is also a critical part for patients with suspected or known heart failure. Objective evaluation of left ventricular (LV) function is necessary because CXR, ECG and history and physical often fail to distinguish normal from low EF in patients with heart failure *(Grossman, 1991 [Low Quality Evidence]; Aguirre, 1989 [Low Quality Evidence]; Soufer, 1985 [Low Quality Evidence])*.

E. Assess for Causative and Precipitating Factors

Causes of heart failure can be classified as cardiac and non-cardiac. Refer to Table 1, "Cardiac-Related Causes" and Table 2, "Non-Cardiac-Related Causes," for the salient features of the more common causes.

Return to Algorithm *Return to Table of Contents*

Table 1: Cardiac-Related Causes

Etiology	History and Physical	Main Treatment
*__Coronary artery disease__ • Most common cause of heart failure 1) Stable with or without ischemic cardiomyopathy 2) New ischemia	Hx: Of stable CAD, or ischemic cardiomyopathy, chest pain, pulmonary edema, diabetes or other ischemic symptoms Px: Tachycardia, diaphoresis, hypoxia Labs: Normal troponin, abnormal ECG or imaging (stress) test, Na^+, K^+	- ASA, beta-blocker, ACE inhibitor, statin, antiplatelets - Other treatment for ischemic cardiomyopathy - Evaluation for revascularization - Investigate for new ischemia and treat the new ischemia.
*__Hypertension__ • One of the most common causes of heart failure	Hx: Family history of hypertension Px: Elevated BP, S4 Labs: ECG, chest x-ray, urinalysis, lipid panel, electrolytes, renal function, studies for secondary hypertension and end organ damage	- For stable (and outpatient) patient, pharmacologic treatment needs individualization to fit heart failure patient. Heart failure medication will lower BP. - Non-pharmacologic treatment is also important (lifestyle management).
*__Valvular heart disease__	Hx: Dyspnea on exertion Px: Pulmonary edema, murmurs Labs: Echocardiogram and cardiac catheterization are essential	- Advanced valvular heart disease may need surgery; systolic dysfunction from aortic stenosis usually improves after surgery. - Afterload reduction treatment is important in mitral regurgitation.
__Arrhythmia__ 1) Tachycardia-induced cardiomyopathy 2) Bradycardia/Heart block	Hx: Tachycardia, palpitations, often A Fib Px: Tachycardia Labs: ECG and rhythm monitor, electrolytes Hx: Vertigo, syncope Px: Sinus node dysfunction, AV block, bradycardia Labs: Lyme serology, electrolytes, ECG, rhythm monitor	- Control of the tachycardia should lead to improvement of ejection fraction. - Short-term treatment is atropine and temporary pacing. - Long-term treatment is pacemaker or biventricular pacing for patients with low LV ejection fraction.
__Myocarditis__	Hx: Prior valvular disease, shortness of breath, fever Px: Exclude other specific cardiomyopathies, anemia, hematuria, murmur Labs: ECHO, ECG, MRI, Troponin, Chemistry Panel, blood cultures, specific viral or microorganism study, endomyocardial biopsy may be helpful but only to few types, e.g., giant cell myocarditis	- Fulminating myocarditis will need urgent and aggressive treatment. - Steroid and immunosuppressive therapy may be helpful.
__Postpartum cardiomyopathy__	Hx: About 1 month before and 6 months after delivering, mostly early postpartum, shortness of breath, dyspnea on exertion Px: Jugular venous distention, rales, S3, hepatomegaly, peripheral edema, murmurs Labs: ECG, chest x-ray, cardiac catheterization, myocardial biopsy, iron/IBC, iron saturation test, TSH	- About 50% completely recover. - Subsequent pregnancy 1. Decreased LV function – deteriorate further 2. Normal LV function – better prognosis
*__Idiopathic cardiomyopathy Viral or autoimmune__	Exclude other specific cardiomyopathies. Always consider alcohol intake as possible etiology.	- Acute or unstable - Stable condition
__Acute, reversible, stress-induced cardiomyopathy/Takotsubo cardiomyopathy__	Hx: Severe physical/emotional stress, chest pain Px: Pulmonary edema, cardiac failure/collapse Labs: Troponins, BNP, other labs as for cardiogenic shock, ECG, echo, coronary angiogram, cardiac MRI	- As in acute coronary syndrome: negative inotropes, vasopressors, ASA, nitrates, heparin, IABC (Intra-aortic balloon counter pulsation) - There is rapid reversal of left ventricular function and survival without long-term sequelae. IABC is not the only modality to improve condition.

*2005 ACC/AHA Practice Guidelines

Return to Algorithm *Return to Table of Contents*

Table 2: Non-Cardiac-Related Causes

Etiology	History and physical	Work-up
Alcoholic cardiomyopathy	Moderate intake of alcohol (alcohol-related social or medical problems need not be present)	Labs: Elevated MCV, gamma glutamyl transferase (GGT), serum uric acid, triglycerides
Sarcoidosis	Hx: Fever, weight loss, anorexia, fatigue, isolated neurological complaints Px: Chest x-ray positive for bilateral hilar adenopathy, skin lesions	Chest x-ray, ECG (heart block), pulmonary function tests Labs: Calcium, liver function tests
Amyloidosis	Hx: Diarrhea, gastrointestinal upset, DOE, check for associations (carpal tunnel, rheumatic diseases) Px: Skin lesions	ECG (heart block, decreased forces), fat aspirate or rectal biopsy Labs: BNP/NTproBNP, cTN
Hemochromatosis	Hx: New-onset diabetes, weight loss, lassitude, weakness, abdominal pain Px: Hepatomegaly, splenomegaly, skin pigmentation	Labs: Serum ferritin, iron binding and saturation, serum calcium Liver biopsy, CT or MRI of liver
Low oxygen-carrying capacity (anemia)	Hx of blood loss/anticoagulation Px: Pallor	Labs: CBC Subsequent workup based on type of anemia
Fluid overload (dietary, lifestyle, medication, etc.)	Hx: Missed medication, dietary indiscretion, over-the-counter meds, NSAIDs, renal failure Px: Edema, weight increase	Labs: Creatinine, BUN, serum albumin
Renal failure/Nephrotic syndrome/Glomerulonephritis		Labs: Urinalysis (proteinuria) Urinalysis (red blood cells or cellular casts)
Thyroid disorders	Px: Myxedema – pale, cool skin	Labs: Thyroid function tests, thyroid antibodies; sensitive TSH with further testing as needed
Systemic infection	Hx: Fever, cough, dysuria	CXR, CT scan Labs: Culture, hematology, serology

(continued on next page)

Return to Algorithm *Return to Table of Contents*

Table 2: Non-Cardiac-Related Causes (cont.)

Etiology	History and physical	Work-up
Pulmonary causes • Cor pulmonale • Pulmonary hypertension • Pulmonary embolism	Hx: SOB, prolonged immobilization Px: Tachycardia, hypoxia, RV overload and/or failure	CXR, CT pulmonary angiogram, ABGs, ECHO
Cardiac toxins • Alcohol • Chemotherapy • Stimulants (methamphetamine, ephedra, cocaine) • Tricyclic antidepressants, vascular (renal) toxins • COX-1 and COX-2 inhibitors • Licorice • Glitazones • Glucocorticoids, androgens, estrogens	Hx: Cardiotoxic agent exposure	• ECHO • Discontinue or seek alternatives to exacerbating drugs
Sleep apnea	Hx: Snoring, nocturnal awakening, A Fib, sudden cardiac arrest	Overnight polysomnography

Return to Algorithm *Return to Table of Contents*

3. Unstable Signs and Symptoms Requiring Emergent Management?

Recommendation:

- Early triage should be performed to determine whether emergent or inpatient care is needed.

Unstable symptoms may include:

- Dyspnea: at rest/orthopnea (change from baseline), sudden onset of shortness of breath (SOB), worsening SOB, exertional dyspnea, gasping
- SaO_2 less than 90%
- Coughing up pink/frothy sputum
- Dizziness or syncope
- Chest pain
- Systolic BP less than 80-90 mmHg and symptomatic
- Evidence of hypoperfusion (cyanosis, decreased level of consciousness, etc.)

Return to Algorithm *Return to Table of Contents*

4. Send to Hospital for Emergency Department or Inpatient Care

Consider hospitalization in the presence or suspicion of heart failure with any of the following findings:

- Clinical, laboratory or electrocardiographic evidence of acute myocardial ischemia or infarction
- Severe symptoms of heart failure refractory to outpatient therapy
- Pulmonary edema or severe respiratory distress
- Thromboembolic complications requiring interventions
- Severe complicating medical illness (e.g., pneumonia, renal failure)
- Management of clinically significant arrhythmias (hemodynamic effects)
- Anasarca (generalized edema)
- Inadequate social support for safe outpatient management
- Symptomatic hypotension or syncope
- Hyperkalemia

By definition, these patients are Stage C and D, NYHA Class III or IV. (See Appendix A, "Heart Failure Classification Comparison," for the New York Heart Association classification and ACC/AHA Staging System comparison.) Heart failure should not be the final, stand-alone diagnosis. There should always be an associated etiology and/or contributing factor. The etiology of heart failure and the presence of exacerbating factors or other diseases that may have an important influence on management should be carefully considered in all cases.

Return to Algorithm *Return to Table of Contents*

5. Determine the Diagnosis of Heart Failure Using the Framingham Criteria Tool

Diagnosis of heart failure requires the simultaneous presence of at least two major criteria or one major criterion in conjunction with two minor criteria.

Major criteria:

- Paroxysmal nocturnal dyspnea
- Neck vein distention
- Rales
- Radiographic cardiomegaly (increasing heart size on chest radiography)
- Acute pulmonary edema
- S3 gallop
- Increased central venous pressure (greater than 16 cm H_2O at right atrium)
- Hepatojugular reflux
- Weight loss greater than 4.5 kg in five days in response to treatment

Return to Algorithm *Return to Table of Contents*

Minor criteria:

- Bilateral ankle edema
- Nocturnal cough
- Dyspnea on ordinary exertion
- Hepatomegaly
- Pleural effusion
- Decrease in vital capacity by one-third from maximum recorded
- Tachycardia (heart rate greater than 120 beats/minute)

Minor criteria are acceptable only if they cannot be attributed to another medical condition (such as pulmonary hypertension, chronic lung disease, cirrhosis, ascites, or the nephrotic syndrome).

The Framingham Heart Study criteria *(McKee, 1971 [Low Quality Evidence])* are 100% sensitive and 78% specific for identifying persons with definite heart failure.

Return to Algorithm *Return to Table of Contents*

8. Obtain Echocardiogram

Recommendation:

- Clinicians must determine whether ventricular dysfunction is systolic or diastolic, because therapies are different. Some therapies for systolic dysfunction may be harmful if used to treat preserved systolic function.

In patients with heart failure symptoms, it is important to determine if they have left or right ventricular systolic dysfunction or preserved systolic function. One-third of patients have predominantly preserved systolic function, one-third have both systolic and diastolic dysfunction, and one-third have predominantly systolic dysfunction.

The community prevalence of heart failure with preserved EF is high, and among these patients, most have preserved systolic function. Heart failure with preserved ejection fraction (HFPEF) has increased in prevalence over time. Patients with preserved ejection fraction (EF) are older, are more likely to be women, are less likely to be smokers or have a history of MI, and have a lower New York Heart Association class but have similar comorbidities.

Patients with heart failure with preserved ejection fraction (HFPEF) have the same or only slightly better rates of survival than those with systolic heart failure *(Aurigemma, 2006 [Low Quality Evidence]; Bhatia, 2006 [Low Quality Evidence]; Owan, 2006 [Low Quality Evidence])*.

Studies show that the distinction between systolic dysfunction and diastolic dysfunction with preserved systolic function is important, because the choice of therapy may be quite different, and some therapies for systolic dysfunction may be detrimental if used to treat patients with primarily diastolic dysfunction. *(Owan, 2006 [Low Quality Evidence]; Topol, 1985 [Low Quality Evidence])*.

Diastolic dysfunction of mild degree is commonly associated with systolic dysfunction, but isolated diastolic dysfunction may be seen with left ventricular hypertrophy, myocardial ischemia, constrictive pericarditis or cardiac tamponade, or in the case of infiltrative diseases such as amyloidosis or in long-standing hypertension *(Persson, 2007 [Moderate Quality Evidence])*.

Return to Algorithm *Return to Table of Contents*

Interpretation of ventricular function testing

Heart failure is a clinical syndrome that correlates poorly with ejection fraction. Some patients may have symptoms based on systolic dysfunction (heart failure with reduced ejection fraction [HFREF]), while others have heart failure with diastolic dysfunction and preserved systolic function (heart failure with preserved ejection fraction [HFPEF]). Measurement of LV function provides important prognostic information.

Objective assessment of left ventricular (LV) function is necessary because CXR, ECG and history and physical often fail to distinguish normal from low EF in patients with heart failure.

Measurement techniques

Both echocardiography and radionuclide ventriculography may be used to measure left ventricular performance. Both methods are reasonably accurate and reproducible for the assessment of systolic dysfunction, but may be influenced by operator technique and ventricular loading conditions. In general, it is appropriate to think of the EF measurement in an individual patient at a particular point in time as being an estimate with a range of ± 5%. Reproducible and operator independent quantitative assessment of preserved systolic function is more difficult and may be influenced by changes in ventricular preload and afterload at the time of the test.

Measurements may vary with changes in the underlying disease process or with differences in systolic or diastolic ventricular loading conditions. Hence, they may change over time because of progression or regression of the underlying ventricular muscle dysfunction, and/or with changes in therapy, as well as the level of hydration at the time of measurement. It is reasonable to reassess ventricular function after interventions or when symptoms have changed significantly. Changes in ventricular function may imply a change in prognosis and may require changes in therapy.

The quantitative assessment of systolic function does not imply an understanding of the underlying etiology of the ventricular dysfunction. Care must be taken to determine the cause of dysfunction so that specific therapy can be instituted, (e.g., treatment of ischemia, valve disease, hypertension, pericardial disease, hyperthyroidism).

The quantitative assessment of ventricular function is essential for the proper classification of the type of ventricular dysfunction and of the severity of dysfunction. Quantitative measurement is valuable for prognosis, as well as for the serial assessment of the response to therapy.

Return to Algorithm *Return to Table of Contents*

Table 3: Measurement Techniques of LV Function

Test	Advantage	Disadvantages
Echocardiogram	• Permits concomitant assessment of valvular disease, left ventricular hypertrophy, and left atrial size • Able to detect pericardial effusion and ventricular thrombus • More generally available • Can be done at time of stress testing • Can assess RV function	• Difficult to perform in patients with lung disease • Usually only semiquantitative estimate of ejection fraction provided • Technically inadequate in approximately 10% of patients under optimal circumstances
Radionuclide ventriculogram	• More precise and reliable measurement of ejection fraction • Can be done at time of stress testing	• Requires venipuncture and radiation exposure and regular R-R intervals • No assessment of valvular heart disease and left ventricular hypertrophy
Left ventriculogram	• Can be done at the time of coronary angiography • Can assess for mitral regurgitation and systolic function • Can measure pressure, particularly in diastolic filling pressure	• Invasive procedure that requires increased dye load with potential renal insufficiency and/or hypotension • Does not assess diastolic function or right ventricular function
MRI	• Most precise method for determining LV function, size and mass	• Requires patient to be in normal sinus rhythm and be able to suspend respiration for a short period of time • Can be difficult to perform in critically ill and unstable patients • Cannot perform on patients with defibrillators
Right heart catheterization (Swan-Ganz)	• Useful when bedside assessment of volume status is unclear	• Invasive procedure and does not assess coronary arteries or valvular heart disease • An indirect measure of LV function
Non-invasive Bioimpedence Hemodynamics	• Non-invasive measure of thoracic fluid	• Poor reliability • An indirect measure of LV function

In patients with CAD and angina, patients with suspected CAD as a cause of heart failure or patients with LV dysfunction but without angina, coronary angiography may be the investigation of choice to determine coronary anatomy and the need for revascularization. In patients in whom coronary artery disease has been

Return to Algorithm *Return to Table of Contents*

excluded previously as the cause of left ventricular dysfunction, repeated invasive or non-invasive assessment for ischemia is generally not indicated.

Magnetic resonance imaging or computed tomography may be useful in evaluating ventricular mass, detecting right ventricular dysplasia or recognizing the presence of pericardial disease.

Return to Algorithm *Return to Table of Contents*

11. Heart Failure Management

Treatment of systolic dysfunction

The cornerstone of treatment is the use of beta-blockers and ACE inhibitors. Certain beta-blocking medications have been shown to improve clinical symptoms and ventricular function in patients with systolic dysfunction.

Beta-blockers decrease hospitalizations and mortality, and have objective beneficial effect on measures of exercise duration. The MERIT HF study of metoprolol succinate compared to placebo showed a mortality reduction at one year in patients with NYHA Class II-IV heart failure and, recently, the COMET trial has shown carvedilol to produce an additional 17% risk reduction in mortality versus metoprolol tartrate *(Poole-Wilson, 2003 [Moderate Quality Evidence]; Packer, 2002 [High Quality Evidence]; Packer, 2001 [High Quality Evidence]; CIBIS-II Investigators and Committees, 1999 [High Quality Evidence]; MERIT-HF Study Group, 1999 [Moderate Quality Evidence]; Bristow, 1996 [High Quality Evidence]; Packer, 1996a [Moderate Quality Evidence]; Andersson, 1994 [Moderate Quality Evidence])*.

ACE inhibitors prolong life in patients with heart failure symptoms and EF less than 35% and reduce symptom development in asymptomatic patients with EF less than 35% *(The SOLVD Investigators, 1992 [High Quality Evidence]; The SOLVD Investigators, 1991 [High Quality Evidence])*.

There is also a mortality benefit in the use of ACE inhibitors in patients with recent myocardial infarction and asymptomatic EF less than 40% *(Pfeffer, 1992 [High Quality Evidence])*.

ACE inhibitors slow disease progression, improve exercise capacity and decrease hospitalizations and mortality *(Packer, 1999 [High Quality Evidence]; Captopril-Digoxin Multicenter Research Group, 1988 [Moderate Quality Evidence]; CONSENSUS Trial Study Group, 1987 [Moderate Quality Evidence])*.

Patients who are intolerant of ACE inhibitors may benefit from the combination of hydralazine and nitrates. This treatment has been shown to improve survival compared to placebo but is less effective than ACE inhibition *(Cohn, 1991 [High Quality Evidence]; Cohn, 1986 [Moderate Quality Evidence])*. ARBs are recommended for patients intolerant of ACE inhibitors *(Hunt, 2009 [Guideline])*.

The combination of hydralazine and nitrates in addition to ACE inhibitors, beta-blockers and duretics is recommended for patients self-described as African Americans who have moderate-to-severe symptoms. This combination has resulted in significant benefit to the group in randomized controlled trials *(Jessup, 2009 [Guideline])*.

Digoxin improves symptoms for patients in sinus rhythm with ventricular dilatation, elevated filling pressures and a third heart sound *(Packer, 1993 [High Quality Evidence]; Lee, 1982 [Moderate Quality Evidence]; Arnold, 1980 [Low Quality Evidence])*.

Digitalis improves symptoms, exercise tolerance and quality of life but neither increases nor decreases mortality *(Digitalis Investigation Group, 1997 [High Quality Evidence])*. Digoxin significantly increased ventricular ejection fraction compared to both placebo and captopril. It also decreased hospitalizations and treatment failure compared to placebo *(Captopril Digoxin Research Group, 1988 [Moderate Quality Evidence])*. The ACC/AHA 2009 Guideline Focused Update lists Digitalis as beneficial in heart failure patients with reduced LVEF to decrease hospitalizations for HF-Class IIa Level B *(Hunt, 2009 [Guideline])*.

Return to Algorithm *Return to Table of Contents*

Finally, diuretics should be used, in the smallest doses necessary, to control fluid retention. Care should be taken to avoid hypokalemia, hypomagnesemia, prerenal azotemia, or orthostatic hypotension. Diuretic doses may need to be reduced in order to introduce or optimize treatment with ACE inhibitors and beta-blockers. Aldosterone antagonists have been shown to reduce mortality. Addition of an aldosterone antagonist is recommended in selected patients with moderately severe to severe symptoms of HF and reduced LVEF who can be carefully monitored for preserved renal function and normal potassium concentration. Creatinine should be 2.5 mg per dL or less and potassium should be less than 5.0 mEq per liter. Under circumstances where monitoring for hyperkalemia or renal dysfunction is not anticipated to be feasible, the risks may outweigh the benefits of aldosterone antagonists. An ARB or isosorbide/hydralazine combination can be considered in patients intolerant to ACE inhibitors *(Hunt, 2009 [Guideline])*.

Treatment of heart failure with preserved ejection fraction (HFPEF)

Recent cross-sectional, population-based echocardiographic studies show that about half of all patients with heart failure have preserved left ventricular ejection fraction. Cohort studies of hospitalized patients show a smaller proportion of heart failure with preserved ejection fraction (HFPEF). Compared to those with reduced systolic function, patients with HFPEF are more often female, older, less likely to have coronary artery disease, and more likely to have hypertension. Patients with HFPEF are less symptomatic and receive different pharmacologic therapy than patients with reduced ejection fraction. Morbidity and mortality rates in patients with HFPEF are high but not quite as high as in patients with reduced systolic function *(Hogg, 2004 [Low Quality Evidence])*.

In the Framingham Heart Study, heart failure was attributed to coronary artery disease in 52%, valvular heart disease in 8%, hypertension in 26% and other causes in 14%. Multivariable predictors of HFPEF versus heart failure with reduced ejection fraction (HFREF) included elevated systolic blood pressure, atrial fibrillation and female sex. Conversely, prior myocardial infarction and left bundle-branch block QRS morphology reduced the odds of HFPEF. Long-term prognosis was grim, with a median survival of 2.1 years (five-year mortality rate, 74%), and was equally poor in men and women with HFREF or HFPEF *(Lee, 2009 [Low Quality Evidence])*.

In contrast to the treatment of heart failure due to reduced LVEF, few clinical trials are available to guide the management of patients with heart failure and relatively preserved LVEF. For the management of patients with heart failure with preserved ejection fraction (HFPEF), it is particularly important to address the underlying etiology. Ischemia and hypertension must be optimally controlled. Pericardial disease must be specifically treated if present. Control of atrial tachyarrhythmias may be of particular importance since these patients need adequate time for diastolic filling, and they poorly tolerate tachycardia. Beta-blockers may be of value to slow the heart rate and allow a longer time interval for diastolic filling.

In general, drugs used to treat systolic dysfunction (ACE, ARBs, diuretics, beta-blockers) are generally used in patients with heart failure with preserved systolic function but indicated to manage comorbidities *(Yusuf, 2003 [Low Quality Evidence])*.

Patients with hypertrophic cardiomyopathy should be identified and may benefit from genetic counseling. Patients with hypertrophic cardiomyopathy may benefit from beta-blockers to slow heart rate. Some may benefit from verapamil or disopyramide if beta-blockers are not effective. In cases of significant intracavitary pressure gradients, dual chamber pacing or septal myectomy surgery may be indicated. Particular attention must be given to the control of atrial tachyarrhythmias. Care should be taken to avoid venodilators and arterial vasodilators.

For patients with predominant heart failure with preserved ejection fraction (HFPEF):

Treat specific contributing causes:

- Hypertension (goal is blood pressure of less than 130/80 mmHg) *(Hunt, 2009 [Guideline])*

Return to Algorithm *Return to Table of Contents*

- Ischemic heart disease
- Hypertrophic cardiomyopathy – consider referral to subspecialist (for verapamil, disopyramide, surgical myectomy, pacemaker)
- Constrictive pericarditis

See Table 4, "New York Heart Association Funtional Classification and Treatment."

Also see Annotation #13, "Pharmacologic Management."

Table 4. New York Heart Association Functional Classification and Treatment

NYHA Class 1	NYHA Class 2	NYHA Class 3	NYHA Class 4
No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation or dyspnea (shortness of breath). **ACE inhibitor** and **beta-blocker** (at recommended doses)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation or dyspnea. **ACE inhibitor** and **beta-blocker** (at recommended doses) The combination of **hydralazine and isosorbide dinitrate** is recommended in addition to beta-blockers and ACE inhibitors for **African Americans**. **For others,** may be added for symptom control or as an alternative if ACE/ARB not tolerated. **Aldosterone antagonist** if creatinine < 2.5 mg/dL and K+ < 5.0 mmol/L. **Loop diuretics** convey no survival benefit but reduce symptoms due to fluid overload. Digoxin conveys no survival benefit but reduces symptoms. **ICD placement** considered if LVEF < 35% and mild to moderate symptoms • Ischemic etiology • Non-ischemic etiology	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation or dyspnea. **ACE inhibitor** and **beta-blocker** (at recommended doses) The combination of **hydralazine and isosorbide dinitrate** is recommended in addition to beta-blockers and ACE inhibitors for **African Americans**. **For others,** may be added for symptom control or as an alternative if ACE/ARB not tolerated. **Aldosterone antagonist** if creatinine < 2.5 mg/dL and K+ < 5.0 mmol/L **Loop diuretics** convey no survival benefit but reduce symptoms due to fluid overload. **Digoxin** conveys no survival benefit but reduces symptoms. **ICD placement** considered if LVEF < 35% and mild to moderate symptoms • Ischemic etiology • Non-ischemic etiology **CRT-P** considered if LVEF < 35% and QRS prolongation (QRS width 120 ms) in those who are symptomatic despite optimal medical therapy.	Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased. **ACE inhibitor** and **beta-blocker** (at recommended doses) A combination of **hydralazine and isosorbide dinitrate** is recommended in addition to beta-blockers and ACE inhibitors for **African Americans** with heart failure and reduced LVEF. **For others,** may be added for symptom control or as an alternative if ACE/ARB not tolerated. **Aldosterone antagonist** if creatinine < 2.5 mg/dL and K+ < 5.0 mmol/L **Loop diuretics** convey no survival benefit but reduce symptoms due to fluid overload. **Digoxin** conveys no survival benefit but reduces symptoms. **ICD placement** considered if LVEF < 35% and mild to moderate symptoms. • Ischemic etiology • Non-ischemic etiology **CRT-P** considered if LVEF < 35% and QRS prolongation (QRS width 120 ms) who are symptomatic despite optimal medical therapy. Consider evaluation for **heart transplant** or other surgical intervention. **LVAD placement** may be used as a bridge to transplantation and in the management of patients with acute, severe myocarditis and may be considered for long-term use when no definitive procedure is planned.

Adapted from ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008. *Eur J Heart Failure* 2008:933-89.

12. Treat Secondary Causes of Heart Failure and Significant Comorbid Conditions and Risk Factors

Recommendations:

- Beta-blockers and digoxin should be used either alone or in combination for achieving rate control in atrial fibrillation in heart failure.
- Blood transfusions are not recommended to treat anemia in heart failure.
- Intravenous iron replacement may improve anemia symptoms specifically the six-minute walk test.

Treat as indicated by the particular disease state. Specific treatment modalities for secondary causes of HF are considered outside of the scope of this guideline. See Table 1, "Cardiac-Related Causes" and Table 2, "Non-Cardiac-Related Causes." See also the ICSI Hypertension Diagnosis and Treatment guideline.

Atrial fibrillation in heart failure

Several studies have been done in patients with heart failure and atrial fibrillation that will influence management of this arrhythmia *(Köber, 2008 [Moderate Quality Evidence]; Roy, 2008 [Moderate Quality Evidence])*. Patients with heart failure are at increased risk for atrial fibrillation and constitute an important subgroup of all patients with this arrhythmia. Atrial fibrillation affects 10-30% of patients with chronic heart failure. Atrial fibrillation may be a marker of poor prognosis, in which the primary problem is poor ventricular function, neurohormonal activation, or inflammation, with no independent effect of atrial fibrillation on outcome.

In the multicenter AF-CHF trial on patients with heart failure who had LVEF less than 35% and atrial fibrillation, a strategy of "rhythm control" with drugs and cardioversion was not superior to a strategy of "rate control" *(Roy, 2008 [Moderate Quality Evidence])*. There was no significant difference between the two groups in primary outcome of time to cardiovascular death. Secondary outcomes were similar in the two groups, including death from any cause, stroke, worsening heart failure, and the composite of death from cardiovascular causes, stroke or worsening heart failure.

The control of ventricular rate and the prevention of thromboembolic events are essential elements of treatment of heart failure in patients with an underlying supraventricular arrhythmia. Beta-blockers and digoxin used either alone or in combination are the drugs of choice for achieving rate control. Digoxin is effective in controlling ventricular rate at rest but may not achieve satisfactory rate control with exertion. Amiodarone may be added to beta-blockers and/or digoxin if adequate rate control is not achieved. Of anti-arrhythmics used in patients with heart failure with reduced ejection fraction and AF, only amiodarone and dofetilide do not effect survival adversely *(Roy, 2008 [Moderate Quality Evidence])*.

In a similar patient population that was resistant to drug treatment for both rhythm and rate control, electrophysiological interventions such as pulmonary vein isolation and atrio-ventricular node ablation combined with bi-ventricular pacing have shown promising results *(Khan, 2008 [Moderate Quality Evidence])*. The work group awaits results of ongoing trials.

Cardiorenal syndrome

No interventions based on proposed mechanisms of the development of cardiorenal syndrome (CRS) have shown consistent advantage, and the work group does not have any specific recommendations. However, developing awareness, the ability to identify and define, and physiological understanding will help improve the outcome of these complex patients.

A large proportion of patients with the syndrome of heart failure have concurrent heart and renal dysfunction. The "Cardiorenal syndrome" has been classified *(Ronco, 2008 [Low Quality Evidence]*). The development

and progression of renal failure is a strong independent predictor of long-term adverse outcome in patients with congestive heart failure. The overall understanding of the pathogenesis of Cardiorenal syndrome is limited. Cardiorenal syndrome can be generally defined as a pathophysiologic disorder of the heart and kidneys whereby acute or chronic dysfunction of one organ may induce acute or chronic dysfunction of the other. The characterization and classification of this syndrome may provide ideas for the testing of hypotheses regarding the pathogenesis of this syndrome and help in designing interventions for the management of Cardiorenal syndrome.

Decreased cardiac output, and the resultant hypoperfusion of the renal vascular bed has been implicated as the dominant pathogenetic factor. Venous congestion and elevated intra-abdominal pressure have been proposed as contributing factors. Further studies are needed to test the role of hemodynamic versus nephrotoxic factors in the development and progression of this syndrome.

The Acute Dialysis Quality Initiative (ADQI) Consensus Group has recently released consensus statements on the epidemiology *(Bagshaw, 2010 [Low Quality Evidence])*, prevention *(McCullough, 2010 [Low Quality Evidence])* and management *(Davenport, 2010 [Low Quality Evidence])* of Cardiorenal syndrome based on the classification proposed by Ronco and colleagues *(Ronco, 2008 [Low Quality Evidence])*.

Anemia: Workup and Treatment for Iron Deficiency in Patients with Heart Failure

The prevalence of iron deficiency in congestive heart failure ranges from 5-21% and may be related to malabsorption, long-term aspirin, uremic gastritis, or reduced iron recycled in the reticuloendothelia system *(Silverberg, 2008 [Low Quality Evidence])*. Heart failure guidelines suggest that the correction of anemia has not been established as routine heart failure therapy, and more specifically, that blood transfusions are not recommended to treat the anemia of chronic disease in heart failure *(Stamos, 2010 [Low Quality Evidence]; Dickstein, 2008 [Guideline])*. It is unclear whether anemia is the cause of decreased survival or a marker of more severe disease in heart failure patients. Intravenous iron replacement for iron deficiency in heart failure patients with or without anemia has been shown to improve symptoms of heart failure, specifically an improvement in the six-minute walk test with an increase of 40 meters from baseline at 24 weeks of therapy and improvements in quality-of-life assessments *(Toblli, 2007 [Moderate Quality Evidence])*. The death rate and serious adverse event rate are not significantly improved. Other treatments including oral iron replacements *(Groenveld, 2008 [Systematic Review])*, erythropoiesis stimulating agents (ESAs), and blood transfusions have not shown proven benefit.

The Ferric Iron Sucrose in Heart Failure (FERRIC-HF) trial of 35 heart failure patients with iron deficiency witnessed an improved global assessment score and significantly increased peak oxygen uptake in patients with anemia *(Okonko, 2008 [Moderate Quality Evidence])*.

Ferinject Assessment in Patients with Iron Deficiency and Chronic Heart Failure (FAIR-HF) trial tested intravenous ferric carboxymaltose and found that in patients, with or without anemia, symptoms improved, functional capacity increased and quality of life improved *(Anker, 2009 [Moderate Quality Evidence])*. Patients in the FAIR-HF trial had NYHA class II or III, left ventricular function less than 40%, ferritin level < 100 *u*g per liter or between 100 and 299 *u*g per liter, if the trans-ferrin saturation was < 20%. Patients were treated with intravenous iron one time per week for 4-5 weeks to replenish their iron store and then had maintenance treatments at 8, 12, 24 and 26 weeks. If ferritin level was greater then 800 *u*g or 500 *u*g with trans-ferrin sat > 50%, the IV iron dose was not given. Significant improvements were seen with IV iron in the distance on the six-minute walk test with an increase of 40 meters from baseline in 24 weeks and improvements in quality-of-life assessments. The rates of death, adverse events, and serious adverse events were similar in the two study groups. Overall, IV iron in patients with chronic heart failure and iron deficiency, with or without anemia, improves symptoms, functional capacity and quality of life. The side-effect profile is acceptable.

Return to Algorithm *Return to Table of Contents*

13. Pharmacologic Management

Recommendations:

- Carvedilol, metoprolol succinate (extended release) and bisoprolol have demonstrated reductions in mortality for patients with all classes of heart failure, so use these agents before using other generic beta-blockers.
- ACE inhibitors should be prescribed for all patients with left ventricular systolic dysfunction unless specific contraindications exist. An elevated baseline creatinine is not a specific contraindication.
- In non-African Americans, ACE inhibitors are recommended for decreasing heart failure mortality over the isosorbide dinitrate/hydralazine combination. In contrast, combining hydralazine and nitrates is recommended for patients self-described as African Americans, with moderate-severe symptoms on optimal therapy with ACE inhibitors, beta-blockers and diuretics.
- Angiotensin receptor blockers should be considered primarily for patients who are intolerant of ACE inhibitors or those receiving standard drug therapy (including ACE inhibitors) who continue to show clinical deterioration.
- Routine use of angiotensin receptor blockers with ACE inhibitors and aldosterone antagonists cannot be recommended.
- Diuretics should not be the sole therapy for patients with signs of volume overload, and vasoactive drugs should be considered.
- In severe heart failure use loop diuretics over thiazide diuretics and combination therapy with thiazide (or thiazide-like medication). Loop diuretics are also effective in refractory cases of volume overload.
- In patients with NYHA Class III-IV heart failure on stable doses of digoxin and ACE inhibitors, reduce mortality by administering aldosterone-blocking agents (spironolactone, eplerenone).
- Currently, the work group recommends that nesiritide be reserved for patients with acutely decompensated heart failure who remain volume overloaded despite aggressive treatment with diuretics/vasodilators display tolerance and/or resistance to vasodilators or diuretics, or demonstrate significant side effects to other vasodilators.
- When considering the use of calcium channel blockers (CCB) in heart failure patients, only dihydropyridine CCBs such as amlodipine and felodipine have been shown to be safe. However, non-dihydropyridines such as diltiazem and verapamil can be used in patients with preserved systolic heart failure *(Haney, 2005 [Low Quality Evidence])*.
- Non-Dihydropyridine Calcium Channel Blockers
  - In patients with HFREF (systolic dysfunction), diltiazem and verapamil appear to worsen heart failure and therefore should be avoided *(Goldstein, 1991 [Moderate Quality Evidence])*.

Return to Algorithm *Return to Table of Contents*

- In patients with HFPEF (diastolic dysfunction), diltiazem and verapamil may be safe to use. In this setting, they may improve exercise capacity and reduce heart failure symptoms *(Hung, 2002 [Low Quality Evidence]); Setaro, 1990 [Low Quality Evidence])*.
- Verapamil is not effective in patients with HF after an acute myocardial infarction *(Danish Study Group on Verapamil in Myocardial Infarction, The, 1990 [Moderate Quality Evidence])*.

• Dihydropyridine Calcium Channel Blockers

- Nifedipine increases hospitalizations and worsens symptoms in NYHA class II or III heart failure patients *(Elkayam, 1990 [Moderate Quality Evidence])*.
- Amlodipine and felodipine have a neutral effect on mortality in heart failure. While not effective for heart failure treatment, they may be useful in the treatment of other conditions in this patient population *(Cohn, 1997 [High Quality Evidence]; Packer, 1996 [Moderate Quality Evidence])*.

The following is an overview of the pharmacological approach to drug selection for patients with heart failure. The optimal management of patients with heart failure is complex. Numerous drugs from various classes are needed for each patient, and frequent titration and dosage adjustments may be needed on an ongoing basis. The following section summarizes major conclusions regarding the optimal use of agents prioritized by beta-blockers, vasodilators, diuretics, aldosterone-blocking agents, inotropes, calcium channel blockers, antiarrhythmic agents, and anticoagulants. Some classes of agents are clearly most beneficial to manage symptomatology (diuretics, for example), while others, such as neurohormonal antagonists (ACE inhibitors and beta-blockers), are clearly morbidity and mortality-reducing agents. Although these classifications are arbitrary, they hopefully serve to organize the information presented in this section.

For detailed information about medication comparisons, interactions and dosing, the work group recommends the following sources:

Epocrates: www.epocrates.com
Micromedex: www.micromedex.com
Lexi-Comp: www.lexi.com
UpToDate: www.uptodate.com
PDR.net: www.pdr.net

Return to Algorithm *Return to Table of Contents*

Pharmacologic Management

Aldosterone antagonists (Eplerenone, spironolactone)	**Role in Heart Failure** Aldosterone antagonists reduce mortality and hospitalizations in patients with heart failure. **Clinical Indication(s)** Patients with heart failure symptoms (NYHA class II-IV) and an EF ≤ 35% who are already on standard therapies (ACE-inhibitors, beta-blockers, diuretics and digoxin). Regardless of the underlying mechanism(s) of benefit, and given the results of EMPHASIS-HF (*Zannad, 2011 [High Quality Evidence]),* aldosterone-receptor antagonists should be prescribed to patients with NYHA class II symptoms, LVEF less than 30% (or wide QRS for those with LVEF 30-35%), and cardiovascular hospitalization within six months or evidence of elevated natriuretic peptide levels in the absence of contraindications *(Jacob, 2011 [Low Quality Evidence]).* **Evidence for Use** • A multicenter, randomized clinical trial showed a reduction in mortality among patients with NYHA Class III-IV HF who were treated with spironolactone 25-50 mg per day. These patients were already on stable doses of digoxin and ACE inhibitors *(Pitt, 1999 [High Quality Evidence]).* • Worsening renal function was associated with a negative prognosis, yet the mortality benefit of spironolactone was maintained *(Vardeny, 2012 [Moderate Quality Evidence]).* • In the EPHESUS trial, eplerenone, was studied in subjects who had a myocardial infarction 3 to 14 days prior and had an LVEF less than 40% with evidence of heart failure (in 90%) and/or diabetes mellitus. There was a significant lower rate of all-cause mortality (14.4%) due to reduction in cardiovascular mortality, reduction in sudden cardiac death, decreased mortality and hospitalizations for heart failure. Most patients in this trial (unlike RALES) were on an ACE inhibitor, or angiotensin receptor blocker and a beta-blocker *(Pitt, 2003 [High Quality Evidence]).* **Contraindications/Cautions** Contraindications include: • Renal dysfunction (creatinine > 2.5, GFR < 30 mL/min) and/or significant impairment of renal excretory function. • A recent increase in serum creatinine > 25% • Hyperkalemia (potassium > 5) Cautions include: • Spironolactone has anti-androgen properties and can cause associated gynecomastia. Eplerenone can safely be used in patients who experience this side effect. • Excessive potassium intake may cause hyperkalemia in patients receiving aldosterone antagonists. • Aldosterone antagonists should not be administered concurrently with other potassium-sparing diuretics. **Lab Monitoring** • **Potassium and BUN/creatinine:** - Baseline - Three to seven days after initiation - Every month for the first three months - Quarterly thereafter • The above lab monitoring cycle should be restarted after dosage increases and when initiating medications that interact with spironolactone/eplerenone.

Return to Algorithm *Return to Table of Contents*

Angiotensin Converting Enzyme Inhibitors (ACE-Inhibitors)	**Role in Heart Failure** ACE-inhibitors reduce morbidity, mortality and hospitalizations in patients with heart failure. **Clinical Indication(s)** ACE inhibitors are indicated for patients with all stages of heart failure (NYHA I-IV) when the EF has dropped below 40%. **Evidence for Use** • For patients with asymptomatic or mildly symptomatic decreases in LV systolic performance, use of ACE inhibitors has been shown to decrease mortality, progression of heart failure and need for hospitalization *(Pfeffer, 1992 [High Quality Evidence]; SOLVD Investigators, The, 1992 [High Quality Evidence]; Cohn, 1991 [High Quality Evidence]; Packer, 1991 [High Quality Evidence]; Captopril-Digoxin Multicenter Research Group, The, 1988 [Moderate Quality Evidence]; CONSENSUS Trial Study Group, 1987 [Moderate Quality Evidence])*. • ACE inhibitors should be prescribed for all patients with left ventricular systolic dysfunction unless specific contraindications exist *(Masoudi, 2004 [Low Quality Evidence])*. • In non-African Americans, ACE inhibitors are more effective in decreasing heart failure mortality than the isosorbide dinitrate/hydralazine combination *(Cohn, 1991 [High Quality Evidence])*. • In African-Americans (self-described) with moderate to severe symptoms, hydralazine and nitroglycerin are recommended in addition to ACE inhibitors, beta-blockers and diuretics. Approach to initiating ACE inhibitor therapy: Start at a low dose and titrate upward over several weeks to targeted moderate to high doses and maximum tolerated dose. Consider holding one dose of diuretic before giving the first dose of ACE inhibitors, particularly in patients with low baseline blood pressure. Where possible, heart failure patients should have their ACE inhibitor dose gradually increased to achieve target doses based on the individual's tolerance and side effects with other heart failure medications *(Packer, 1999 [High Quality Evidence])*. • In studies demonstrating decreased mortality in heart failure, relatively high doses of ACE inhibitors were used. - Enalapril 20 mg daily (twice-daily dosing) - Lisinopril 20-40 mg daily. Lower-dose therapy has been shown to be less effective in reducing mortality *(Packer, 1999 [High Quality Evidence])*. - Captopril 100-150 mg daily (three-times-daily dosing). *(Pfeffer, 1992 [High Quality Evidence]; Cohn, 1991 [High Quality Evidence]; Cohn, 1986 [Moderate Quality Evidence])*

Return to Algorithm *Return to Table of Contents*

Angiotensin-Converting Enzyme Inhibitors (ACE-Inhibitors)	**Contraindications/Cautions** In patients who are intolerant to ACE inhibitors, an angiotensin receptor blocker (ARB) should be initiated. Contraindications include: • history of intolerance or adverse reactions to these agents including angioedema, persistent cough and/or rash; • serum potassium greater than 5.5 mEq/L; • symptomatic hypotension (unless due to excessive diuresis); • severe renal artery stenosis; and • pregnancy Cautions include: • **Renal insufficiency:** Creatinine should be monitored regularly in patients on ACE inhibitors, and more frequently during active titration. An increase in serum creatinine of 0.5 mg/dL or more is an indication for reassessment of volume status. There is no absolute level of creatinine to preclude the use of ACE inhibitors • **Hypotension.** Patients should be well hydrated before initiation or increase of ACE inhibitors. If hypotension develops in the absence of hypovolemia, splitting the dose or switching from morning to bedtime dosing (in long-acting agents) may be helpful. If this is ineffective, the dose should be reduced to the highest dose tolerated. **Lab Monitoring** • **Potassium and BUN/creatinine:** - One to two weeks after initiation/dose increases (one week recommended in the elderly) - Three to four weeks after initiation - Thereafter, one to two times per year • The above lab monitoring cycle should be restarted after dosage increases and when initiating other medications which increase creatinine/potassium

Return to Algorithm *Return to Table of Contents*

Angiotensin Receptor Blockers (ARBs)	**Role in Heart Failure** Angiotensin receptor blockers reduce morbidity, mortality and hospitalizations in patients with heart failure. **Clinical Indication(s)** ARBs should be primarily utilized in patients who meet the criteria for ACE-inhibitor use (NYHA class I-IV, EF < 40%) but who cannot tolerate ACE-inhibitor therapy. **Supporting Literature** ARBs are to be used if ACE inhibitors are not tolerated. • Contraindications to ARBs include history of intolerance or adverse reactions to serum potassium greater than 5.5 mEq/L, symptomatic hypotension (unless due to excessive diuresis), severe renal artery stenosis and pregnancy. • Based on the findings of the series of CHARM studies, recommendations to consider adding an angiotensin receptor blocker to standard optimized drug therapy for those with systolic dysfunction may be supported *(Granger, 2003 [Moderate Quality Evidence]; McMurray, 2003 [Moderate Quality Evidence]; Pfeffer, 2003 [High Quality Evidence]; Yusuf, 2003 [Low Quality Evidence])*. An ARB is the preferred alternative to hydralazine/isosorbide dinitrate in most patients because of ease of use except in renal dysfunction, hyperkalemia and possibly in African Americans *(Taylor, 2004 [Moderate Quality Evidence])*. • According to the CHARM-Added trial, *(McMurray, 2003 [Moderate Quality Evidence])*, there is a benefit in terms of composite cardiovascular endpoints when adding ARB to a regimen of an ACE inhibitor and beta-blockers (triple therapy) *(Granger, 2003 [Moderate Quality Evidence]; McMurray, 2003 [Moderate Quality Evidence]; Pfeffer, 2003 [High Quality Evidence]; Yusuf, 2003 [Low Quality Evidence]; Cohn, 2001 [Low Quality Evidence])*. This observation is consistent overall, with the results of the Val-HeFT study *(Cohn, 2001 [Low Quality Evidence])*. In spite of subgroup analysis from Val-HeFT suggesting that the addition of an ARB to the ACE inhibitor and beta-blocker may have resulted in a negative effect on both mortality and morbidity, the group feels that based on the findings from the CHARM-Added study *(McMurray, 2003 [Moderate Quality Evidence])*, the combination of ARBs to and ACE and beta-blocker regimen is more favored than disfavored at this time. • Only Valsartan and Candesartan are FDA approved for use in patients with heart failure. Although Losartan is not approved for heart failure, there appears to be a beneficial class effect *(ELITE I and ELITE II trials)*. • Direct comparison with regards to mortality in patients with heart failure showed no difference between the ARB Losartan and the ACE inhibitor Captopril *(Pitt, 2000 [High Quality Evidence])*. • According to the VALIANT trial, 2003, there is no benefit when adding ARB to ACE inhibitors in early post-MI patients. **Contraindications/Cautions** Contraindications include: • history of intolerance or adverse reactions to these agents including angioedema and/or rash; • serum potassium greater than 5.5 mEq/L; • symptomatic hypotension (unless due to excessive diuresis); • severe renal artery stenosis; and • pregnancy. Cautions include: • **Renal dysfunction.** While there is no absolute creatinine that precludes use of an angiotensin receptor blocker, any significant rise in creatinine while on an ARB may necessitate holding/discontinuing therapy. • **Hypotension.** Patients should be well hydrated before initiation or increase of an ARB. If hypotension develops in the absence of hypovolemia, splitting the dose or switching from morning to bedtime dosing (in long-acting agents) may be helpful. If this is ineffective, the dose should be reduced to the highest dose tolerated. **Lab Monitoring** • **Potassium and BUN/creatinine:** - One to two weeks after initiation/dose increases (one week recommended in the elderly) - Three to four weeks after initiation - Thereafter, one to two times per year • The above lab monitoring cycle should be restarted after dosage increases and when initiating other medications that increase creatinine/potassium.

Return to Algorithm *Return to Table of Contents*

Beta-Blockers	**Role in Heart Failure** Beta-blockers reduce mortality, hospitalizations and improve symptoms in patients with heart failure. **Clinical Indications** Beta-blockers are indicated for stable, symptomatic (NYHA class II and III) patients with an EF < 40%) and heart failure that is of ischemic, hypertensive or cardiomyopathic origin. Most beta-blocker studies have been done in patients already taking an ACE-inhibitor, diuretic and (in some cases) digoxin. **Supporting Literature** • Studies strongly support use of certain beta-blockers that have demonstrated reductions in mortality and hospitalizations (e.g., carvedilol, metoprolol succinate [extended release], bisoprolol) in patients with NYHA Class I-IV HF. Recent data from COMET demonstrated carvedilol to have a 17% risk reduction in mortality over metoprolol tartrate (immediate release) *(Beta-Blocker Evaluation of Survival Trial Investigators, The, 2001 [High Quality Evidence]; CAPRICORN Investigators, The, 2001 [High Quality Evidence]; Freemantle, 1999 [Systematic Review])*. • Beta-blockers have been shown to have an objective beneficial effect on measures of exercise duration *(CIBIS-II Investigators and Committees, 1999 [High Quality Evidence]; MERIT-HF Study Group, 1999 [Moderate Quality Evidence]; Bristow, 1996 [High Quality Evidence])*. • Beta-blockers are used to decrease mortality and reinfarction among patients with compensated heart failure following acute myocardial infarction. • Beta-blockers are used to improve hemodynamics in patients with idiopathic dilated cardiomyopathy *(Australia/New Zealand Heart Failure Research Collaborative Group, 1997 [Moderate Quality Evidence]; Packer, 1996b [High Quality Evidence])*. • When only one drug can be initiated for heart failure, beta-blockers are preferred *(Fonarow, 2007 [Moderate Quality Evidence]; Sliwa, 2004 [Moderate Quality Evidence])*. • Beta-blockers should be started as soon as the patient is stable (without fluid overload or hypotension). • Start cautiously in acute heart failure or during an exacerbation. After appropriate stabilization, they may be safely started in the inpatient setting. • Start at low initial doses and gradually titrate up at rates consistent with those from key studies *(Sliwa, 2004 [Moderate Quality Evidence])*. Titration of beta-blockers to a target heart rate of 65 to 70 bpm has been shown to increase ejection fraction – depending on dose – in patients with heart failure. • Beta-blockers should not be unnecessarily reduced or discontinued. Sudden cessation may cause a reflexive increase in sympathetic output and worsen heart failure. • If significant bradycardia/AV block occurs with use of beta-blockers, the dose may need to be decreased. If hypotension or fluid retention occurs, either the dose of beta-blocker, ACE inhibitor or diuretics should be adjusted as clinically appropriate. • Patients should be informed that positive effects of beta-blockers may not be seen until several months after titration to their highest clinically tolerated dose *(Jessup, 2009 [Guideline])*. • For rate control in tachycardia-induced heart failure, the work group prefers beta-blockers over other agents. If the patient is limited by symptoms of orthostatic hypotension with carvedilol, consider a change to metoprolol succinate to allow titration to goal heart rate.

Return to Algorithm *Return to Table of Contents*

Beta-Blockers (continued)	**Carvedilol** • The COMET trial demonstrated carvedilol to have a 17% risk reduction in mortality over metoprolol tartrate *(Poole-Wilson, 2003 [Moderate Quality Evidence])*. In type 2 diabetes patients, carvedilol did not adversely affect HgbA1C as compared to metoprolol tartrate. Carvedilol has demonstrated the ability to reduce insulin resistance and microalbuminemia, while metoprolol tartrate did not show the same benefits *(Bakris, 2004 [High Quality Evidence])*. • Recommended starting dose for carvedilol is 3.125 mg twice daily for two weeks. Dosage can be doubled every two weeks to highest level tolerated by patient to maximum 25 mg twice daily (less than 85 kg) or 50 mg twice daily (greater than 85 kg). It is suggested that after initiation of each new dose, patients should be observed for signs of dizziness or lightheadedness. Also consider instructing patients to take carvedilol two hours before ACE inhibitors to decrease potentiating effects. Carvedilol should be taken with food to slow the rate of absorption and reduce the risk of postural hypotension. • Carvedilol has been shown to reduce heart failure-related hospitalizations and death *(Poole-Wilson, 2003 [Moderate Quality Evidence]; Packer, 2002 [High Quality Evidence]; Australia/New Zealand Heart Failure Research Collaborative Group, 1997 [Moderate Quality Evidence])*. **Metoprolol Succinate** • In the MERIT HF study of metoprolol succinate compared to placebo, a mortality reduction was shown at one year in patients with NYHA Class II-IV heart failure *(MERIT-HF Study Group, 1999 [Moderate Quality Evidence])*. • There are no head-to-head trials comparing carvedilol and metoprolol succinate (extended release) *(Beta-Blocker Evaluation of Survival Trial Investigators, The, 2001 [High Quality Evidence]; CAPRICORN Investigators, The, 2001 [High Quality Evidence]; Freemantle, 1999 [Systematic Review])*. • Recommended starting dose of metoprolol succinate is 25 mg/once daily. In patients with more severe heart failure (NYHA Class III or IV), recommended starting dose is 12.5 mg/once daily. The dose may then be doubled every two weeks up to the highest tolerated dose or up to 200 mg/once daily. **Contraindications/Cautions** Contraindications include: • Severe bradycardia • Sick sinus syndrome (unless pacemaker in place) • Second- or third-degree heart block • Cardiogenic shock • Decompensated heart failure • Known allergic reaction Cautions include: • Worsening heart failure can occur in patients with severe heart failure whose beta-blocker dose is titrated up too rapidly. • Abrupt cessation of beta-blockers can lead to rebound hypertension/tachycardia, which may exacerbate existing angina and heart failure. Where possible, dosage reductions/therapy cessation should be done over one to two weeks. • Patients with bronchospastic disease may experience worsening bronchospasm while on beta-blocker therapy. • Beta-blockers may mask the signs and symptoms of hypoglycemia in diabetic patients.

Return to Algorithm *Return to Table of Contents*

Digoxin	**Role in Heart Failure** Digoxin reduces symptoms, hospitalizations and improves exercise capacity in patients with heart failure. Digoxin does not have an effect on heart failure mortality. **Clinical Indications** Digoxin has been found useful in mild-moderate systolic (EF < 40%) heart failure patients with atrial fibrillation with a rapid ventricular response, and in combination with ACE inhibitors in reducing hospitalizations *(Packer, 1993 [High Quality Evidence])*. **Supporting Literature** • In subjects in normal sinus rhythm with preserved systolic function (PSF) and mild to moderate heart failure symptoms on optimal therapy, digoxin had no effect on the endpoints of all-cause or cardiovascular mortality or hospitalization *(Ahmed, 2006 [Low Quality Evidence])*. • Serum levels of less than 1.0 ng/mL are considered therapeutic. Levels greater than 1.2 have been associated with greater side effects. Serum levels do not always correlate to symptoms of digoxin toxicity. • Digoxin should not: - be initiated in asymptomatic heart failure patients as it remains unsupported by clinical trials, or - be "loaded" either orally or intravenously. Loading doses are generally not needed and steady state generally takes one week to reach (longer in patients with renal impairment). **Contraindications/Cautions** Contraindications include: • Patients with ventricular fibrillation • Patients with a known hypersensitivity to digoxin

Return to Algorithm *Return to Table of Contents*

Digoxin (continued)	Cautions include: • **Use in Patients with Impaired Renal Function and in the Elderly:** Digoxin is primarily excreted by the kidneys; therefore, patients with impaired renal function (including the elderly) may require smaller than usual maintenance doses of digoxin. Symptoms of toxicity include nausea, confusion, visual disturbance, anorexia, reduction of renal function or conduction abnormality. To avoid digitalis toxicity, use lower doses in the elderly and those with renal impairment, check level in one to two weeks after start of therapy in elderly or renal-impaired patients, and beware of drug interactions with new medications. • **Use in Patients with Electrolyte Disorders:** In patients with hypokalemia or hypomagnesemia, digoxin toxicity may occur, despite serum digoxin concentrations below 2 ng/mL. Potassium and magnesium should be kept in the normal range in patients being treated with digoxin. • Digoxin can accumulate in patients with **renal dysfunction** and in **the elderly**. Women may experience increased mortality with digoxin compared to placebo. Randomized, prospective studies have not been completed in confirming gender-based differences, but post hoc, retrospective analysis of mortality statistics in heart failure indicate a gender gap. Practitioners may want to consider this when prescribing digoxin to women. If continuing digoxin therapy in women, it may be reasonable to recommend that lower dosing (0.125 mg per day) should be used and lower serum levels (1.0 or less) should be maintained *(Rathore, 2002 [Moderate Quality Evidence]; Digitalis Investigation Group, The, 1997 [High Quality Evidence])*. • Sinus Node Disease and AV Block**:** Digoxin may cause severe sinus bradycardia or sinoatrial block in patients with preexisting sinus node disease. In such patients, consideration should be given to the insertion of a pacemaker before treatment with digoxin. • Accessory AV Pathway (Wolff-Parkinson-White Syndrome): After intravenous digoxin therapy, some patients with paroxysmal atrial fibrillation or flutter and a coexisting accessory AV pathway have developed increased antegrade conduction across the accessory pathway bypassing the AV node, leading to a very rapid ventricular response or ventricular fibrillation. • Use in patients with Preserved Left Ventricular Systolic Function: Patients with certain disorders involving heart failure associated with preserved left ventricular ejection fraction may be particularly susceptible to toxicity of the drug. Such disorders include restrictive cardiomyopathy, constrictive pericarditis, amyloid heart disease, and acute cor pulmonale. Patients with idiopathic hypertrophic subaortic stenosis may have worsening of the outflow obstruction while on digoxin. **Lab Monitoring** • **Digoxin blood levels**: Digoxin levels are typically recommended in patients with 1) Five to seven days after starting therapy/dosage change/starting therapy with known interactions, 2) Suspected toxicity, 3) Suspected non-adherence, 4) New or existing renal dysfunction, and/or 5) Determining whether patient is in the therapeutic range. • **Serum creatinine and electrolytes:** Generally recommended at baseline and then periodically thereafter.

Return to Algorithm *Return to Table of Contents*

Hydralazine/ Nitrates	**Role in Heart Failure** Hydralazine given with isosorbide dinitrate has been shown to provides symptomatic and mortality benefit in patients with systolic heart failure. **Clinical Indications** • Hydralazine/nitrates is indicated for those patients with moderate to severe heart failure symptoms (NYHA class III-IV heart failure, EF < 40%), who are already taking ACE-inhibitors (or ARBs), beta-blockers and diuretics. • Hydralazine/nitrates can also be used IN PLACE of an ACE-inhibitor/ARB in those patients who cannot tolerate either therapy. **Supporting Literature** • Adding hydralazine/isosorbide dinitrate to digoxin and diuretics in patients with symptomatic heart failure improves left ventricular function, increases exercise tolerance and reduces mortality *(Cohn, 1991 [High Quality Evidence]; Cohn, 1986 [Moderate Quality Evidence])*. • In non-African American heart failure patients, ACE-inhibitors had more of an effect on mortality than hydralazine/isosorbide dinitrate *(Cohn, 1991 [High Quality Evidence])*. • Combining hydralazine and nitrates is recommended for patients self-described as African Americans, with moderate-severe symptoms on optimal therapy with ACE inhibitors, beta-blockers, and diuretics *(Jessup, 2009 [Guideline]; Taylor, 2004 [Moderate Quality Evidence])*. More significant gains in hypertensive cardiomyopathy have also been seen in this population. • In non-African Americans, ACE inhibitors are more effective in decreasing heart failure mortality than hydralazine/long-acting nitrate combinations *(Cohn, 1991 [High Quality Evidence])*. • Hydralazine/long-acting nitrates (usually isosorbide dinitrate) may be considered as a therapeutic option in those patients experiencing intolerance to ACE inhibitor and ARB usage. • If higher doses of ACE inhibitors or ARBs are not tolerated despite euvolemia, a lower dose should be continued and/or a trial of hydralazine/isosorbide dinitrate instituted. **Contraindications** • Hydralazine is contraindicated in patients with hypersensitivity to hydralazine and mitral valvular rheumatic heart disease. • Nitroglycerin is contraindicated in patients with hypersensitivity to nitroglycerin and in those patients who are currently using a phosphodiesterase-5 (PDE-5) inhibitor. This combination has been shown to potentiate the hypotensive effects of nitrates. **Hydralazine Cautions** • Hydralazine can produce a clinical picture simulating systemic lupus Erythematosus, including glomerulonephritis. In such patients, hydralazine should be discontinued unless the benefit outweighs risk. • Hydralazine can accentuate angina and increase pulmonary artery pressure in patients with mitral valvular disease. • Myocardial stimulation produced by hydralazine can cause anginal attacks and ECG changes of myocardial ischemia. The drug has been implicated in the production of myocardial infarction. It must, therefore, be used with caution in patients with suspected coronary artery disease. **Nitroglycerin Cautions** • Only the smallest dose required should be administered due to the potential development of tolerance. • Severe hypotension, particularly with upright posture, may occur with small doses of nitroglycerin. It should therefore be used with caution in patients who may be volume-depleted or who are already hypotensive. • Nitrate therapy may aggravate the angina caused by hypertrophic cardiomyopathy.

Return to Algorithm *Return to Table of Contents*

Loop Thiazde Diuretics	**Role in Heart Failure** • Diuretics are helpful in reducing the signs and symptoms of fluid overload (e.g., edema, dyspnea) associated with heart failure. • Diuretics have yet to demonstrate a reduction in morbidity/mortality associated with heart failure. It is therefore recommended that diuretics not be the sole therapy used to treat heart failure. **Supporting Literature** • Loop diuretics are preferred over thiazides in patients experiencing severe volume overload, severe renal insufficiency (creatinine clearance less than 30 mL/min) or persistent edema while on thiazide diuretics. • Chronic congestion may be relieved by torsemide more effectively than other loop diuretics. • If the patient remains fluid overloaded on a loop diuretic alone, adding a thiazide or metolazone in combination with the loop diuretic can be beneficial *(Funke Küpper, 1986 [Moderate Quality Evidence]; Sigurd, 1975 [Low Quality Evidence]; Whight, 1974 [Low Quality Evidence])*. When used together, the thiazide diuretic should be given 30-60 minutes before the loop diuretic to increase overall diuretic effectiveness. • Studies of furosemide have not shown a significant difference in outcomes when using either continuous infusion or bolus intravenous injections *(Felker, 2011 [Meta-analysis])*. In acute decompensated heart failure, there also has been shown to be no difference in the use of low dose (equivalent to the patient's previous oral dose) or high dose (2.5 times the previous oral dose) in terms of the patient's global assessment of symptoms or in the change in renal function. Therefore, no benefit is seen to either low vs. high dose or infusion vs. bolus injection as long as adequate naturesis is maintained. • The work group cannot recommend a single, ideal diuretic for heart failure patients other than to use the lowest possible dose, at least until more rigorous studies are completed. However, the TORIC study suggested a lower mortality among congestive heart failure patients treated with torsemide compared to furosemide/other diuretics *(Cosin, 2002 [Low Quality Evidence])*. Another small, open-label study comparing furosemide with torsemide confirmed that torsemide-treated patients were less likely to be readmitted for heart failure and all cardiovascular causes, and appeared to be less fatigued. **Contraindications/Cautions** Contraindications include: Anuria and existing allergic reaction to the active diuretic component Cautions include: • High doses of loop diuretics can cause ototoxicity/tinnitus. This typically occurs with rapid infusion, severe renal impairment, higher than recommended dosing and/or concomitant use of other ototoxic medications. • Excessive diuresis can cause dehydration, blood volume depletion, hypotension and renal failure. • Loop/thiazide diuretics can cause significant hypokalemia. Potassium levels should be closely monitored during loop/thiazide diuretic therapy. (See lab monitoring.)

Return to Algorithm *Return to Table of Contents*

Loop Thiazde Diuretics (continued)	**Lab Monitoring** • **Sodium, Potassium, Chloride/Bicarb, BUN/Scr (basic chemistry panel)** - Before diuretic initiation - During the diuretic initiation phase, the first lab after starting therapy is dependent on the degree of diuresis: • Outpatients on oral diuretic therapy should have their labs repeated once approximately five to seven days after diuretic initiation. If lab abnormalities are present, labs should be repeated weekly until they have stabilized. • Inpatients on large IV doses of diuretics (including drips) or who require frequent dose changes should have labs repeated daily at a minimum. - Following the initiation phase, stable outpatient diuretic patients should have a basic chemistry panel drawn every four months for the duration of therapy. If any of the following occurs, lab monitoring will need to be more frequent until the patient stabilizes: • Changes in diuretic dose, route or frequency • The patient's condition worsens • The patient develops signs/symptoms of electrolyte abnormalities - If lab abnormalities are found at any time, increased lab surveillance is warranted until levels have normalized. • **Magnesium, calcium** - Before diuretic initiation, then every four months for the duration of therapy • **Glucose checks (diabetic patients)** - Before diuretic initiation - During the initiation phase (the first week of therapy), the glucose checks should be increased above baseline to ascertain the effect of the diuretic on glucose trends - Following the initiation phase, stable outpatient diuretic patients should follow their normal glucose monitoring. If any of the following occurs, glucose checks will need to be more frequent until the patient stabilizes: • Dose/route/frequency changes • The patient's condition worsens • The patient develops signs/symptoms of electrolyte abnormalities • **Uric acid** - Patients without active gout or a history of gout do not need uric acid levels unless signs/symptoms of gout are present. - Patients with gout or a history of gout should have a uric acid level drawn: • Before diuretic initiation • One week after the diuretic is initiated and annually thereafter. - If any of the following occurs, uric acid levels will need to be more frequent until the patient stabilizes: • Changes in diuretic dose, route or frequency • The patient's condition worsens • The patient develops signs/symptoms of worsening gout - If lab abnormalities are found at any time, increased lab surveillance is warranted until levels have normalized.

Return to Algorithm *Return to Table of Contents*

14. Non-Pharmacologic Management

- Engage the patient in his or her care and include shared decision-making between patient, family and the physician.

  Shared decision-making is a process in which patients and clinicians collaborate to clarify all acceptable options, ensure the patient is well-informed, and choose a course of care consistent with patient values and preferences, and the best available medical evidence.

  Please refer to Appendix C, "ICSI Shared Decision-Making Model."

- All patients with heart failure should receive dietary instruction regarding sodium intake since dietary indiscretion is the most common cause of exacerbation of heart failure.
- Provide dietary counseling for patients to learn the need for fluid balance management, avoiding excess sodium and/or water intake. Refer to a dietitian for patients with comorbid conditions or repeat episodes of edema. Sodium restriction alone may provide substantial benefits for heart failure patients.
- Patients should call their clinician about a two-pound or greater weight gain overnight or a five-pound or greater weight gain in a week. Daily weights are important for managing heart failure and early detection of increases in fluid retention.
- Simplifying medication regimes as much as possible should be explored. All medications, including over-the-counter (OTC) medications, should be reviewed at each visit.
- Patients should be assessed for depression. Major depression is common in patients hospitalized with heart failure and is independently associated with a poor prognosis. Additionally, depression is independently associated with a substantial increased risk of heart failure in older patients with isolated systolic hypertension.
- Consider utilizing a heart failure clinic or case management for patients with medical problems or at high risk for rehospitalization.
- Exercise instruction should be included as part of a comprehensive heart failure program *(Davies, 2010 [Systematic Review])*.

Finding the right time to discuss patient preferences, medical options and prognosis is challenging. Difficult decisions now will simplify difficult decisions in the future. It is best for these discussions to be had and documented in the ambulatory setting rather than during an emergent situation, e.g. hospitalization, when thoughtful decision-making may be impaired. During an admission for heart failure it is easier to review rather than introduce advanced care decisions. Consider an annual heart failure, review done by the primary care provider or cardiology team with patient and family. It should include discussion of current and potential therapies for anticipated and unanticipated events. Topics for discussion would include symptom burden and quality of life. It is important to review goals and preferences for the coming year on outcomes like survival and functional capacity. Also discuss what the patient may be eligible for in regards to current treatment with drugs and devices dependent upon the type of heart failure, stage and trajectory *(Allen, 2012 [Guideline])*.

Patient education for early symptom recognition and counseling about the disease process should be initiated at this time. See the Implementation Tools and Resources Table section for Web sites and tools to assist the clinician and patient with non-pharmacologic management of heart failure.

Dietary Recommendations

Dietary indiscretion remains a common cause of exacerbation of heart failure and reinforcement of the importance of dietary compliance should occur at each interaction *(Dracup, 1994 [Low Quality Evidence])*.

Return to Algorithm *Return to Table of Contents*

Assess usual diet, plan dietary modifications in accordance with checking for commonly used foods, ethnic foods or special dietary restrictions and practices. Avoid overly restrictive diet regimens unless medically necessary.

Sodium restriction

A reduction in dietary sodium intake of 2,000 mg per day alone may provide substantial hemodynamic and clinical benefits for heart failure patients. Unfortunately patients (and physicians) frequently rely solely on diuretics to control symptoms. Stress the importance of reading labels, and a no-salt-added diet. If patient has repeat episodes of edema or failure, a daily sodium intake of less than 2,000 mg is recommended. Patients should be referred to a registered dietitian if there are repeat episodes of edema or for comorbid conditions such as diabetes, dyslipidemia and renal failure. It is important to help patients prioritize dietary modifications. For example, low-fat, low-saturated fat diets would be appropriate for patients with hypercholesterolemia. Dietary modifications may need to be liberalized to increase compliance with dietary restrictions required for management of heart failure.

There are specific recommendations for sodium restrictions; these are the generally accepted and utilized guidelines. A goal of 2 grams of sodium daily is reasonable for many patients and is achieved by following a no-added-salt (NAS) diet and the judicious use of processed foods. A stricter recommendation of less than 2,000 mg sodium/day may be considered for patients with moderate to severe heart failure. Education on alternatives for flavoring and label reading should be conducted by a registered dietitian. Caution patients about the use of potassium-containing salt substitutes and potassium intake in general, which could contribute to the development of significant hyperkalemia.

(Adams, 2006 [Guideline])

When appropriate, patients should have dietary counseling to teach them about the need for management of fluid balance, and the importance of avoiding excess sodium and/or water intake.

Fluid management

Patients should be advised to avoid excessive fluid intake. Not all patients require a fluid restriction; however, if patient is edematous or hyponatremic (serum sodium less than 130 mEq/L), a 1,500 or 2,000 cc/day fluid restriction should be recommended.

Alcohol intake

Since even moderate usage may be associated with decreasing ventricular systolic function, alcohol use should be discouraged, or at the least, saved for special occasions. One drink is considered 10 oz. of beer, 5 oz. of wine or 1.5 oz. of hard liquor. In severe heart failure or those with alcoholic cardiomyopathy, complete abstinence is recommended.

Dietary Supplements and Vitamins

Formerly called herbals, any over-the-counter dietary supplement or vitamin product should be discussed with a health care clinician to make sure there is not an interaction with the disease condition or other medications.

Multiple vitamin-mineral supplementation should be considered for those on diuretic therapy and restricted diets to ensure an adequate intake of the recommended daily equivalent of essential nutrients *(Adams, 2006 [Guideline])*.

Daily Weights

Daily weights should be taken upon rising in the morning (before eating and after urinating), on the same scale, wearing the same amount of clothing. Patients should report significant gains or losses to their care clinician, along with any new or worsening symptoms. Patients should be instructed to keep an ongoing

record of these weights and bring these values to all medical appointments. Patients should call about a greater than or equal to two-pound weight gain overnight or a five pound or greater weight gain in a week. To avoid dehydration, patients should additionally call their health care clinician if they have decreased oral intake of fluids and are experiencing unanticipated weight loss of greater than three to five pounds. Daily weights are critical for managing heart failure and early detection of fluid retention. Increases in body weight are associated with hospitalization for heart failure and begin at least one week before admission. Daily information about the patient's body weight identifies a high-risk period during which interventions to avert decompensated heart failure and subsequent hospitalization may be beneficial.

(Chaudhry, 2007 [Low Quality Evidence]; Heart Failure Society of America, 2010 [Guideline]; Agency for Health Care Policy and Research, 1994a [Low Quality Evidence])

Medications

Because of the advanced age of this population and the complexity of medication regimes, every effort should be made to simplify and clarify a patient's medications.

- Group medications so they are taken together.
- Cut down on the frequency of each medication taken per day (i.e., twice daily versus three times daily if bioequivalent).
- Emphasize taking medications at the appropriate time to maximize symptom control (e.g., take nitrates on an empty stomach; however, caution regarding the increased risk of syncope with elderly patients).
- NSAIDs and COX-2 inhibitors are not recommended in patients with chronic heart failure.

All medication instructions, including over-the-counter medications, should be reviewed at each interaction, written clearly and reinforced verbally. The indications and possible side effects of each medication should be explained, and patients should be reminded not to stop or change their medications without talking to their clinician or nurse.

Vaccinations

Pneumococcal vaccine and annual influenza vaccination are recommended in all patients with heart failure in the absence of contraindications *(Adams, 2006 [Guideline])*.

Exercise and Activity Guidelines

Bed rest may be prescribed for patients with decompensated heart failure, because it encourages diuresis and reduced myocardial oxygen consumption. However, investigators have now shown that regular exercise in heart failure patients produces positive effects and represents minimal risks. Regular physical activity increases functional status and decreases symptoms of heart failure, except in patients with acute myocarditis or recent MI for whom exercise restrictions are appropriate. Exercise training programs have demonstrated a reduction in hospitalizations related to systolic heart failure and an improvement in health related quality of life.

Exercise instruction should be included as a part of a comprehensive heart failure program. Referral to a cardiac rehabilitation program is recommended for exercise prescription and modeling and will contribute to patients' compliance with exercise, functional improvement and quality of life. Participation in a formal program may also contain education and compliance monitoring of lifestyle management components for heart failure.

Patients should be counseled about the benefits of a low-intensity aerobic exercise and lightweight conditioning programs. Abnormal responses to exercise, such as lightheadedness, chest pain, marked dyspnea

Return to Algorithm *Return to Table of Contents*

or unusual fatigue should also be discussed with the patient. Increased workload on the heart, either too heavy or too sustained, may result in decompensation of heart failure. Modifications to a patient's daily work schedule and duties may become necessary to accommodate the need for more frequent rest breaks and decreased functional abilities.

Patients should be advised that if they are overly tired the day following an exercise session, modifications are in order. Patients should incorporate an appropriate warm-up and cool-down period.

Note: It is not uncommon for patients who have been exercising for approximately six weeks to need an increase in diuretic dosage. Care should be taken that this does not discourage the patient from continuing exercise training.

(Flynn, 2009 [Moderate Quality Evidence]; Piña, 2003 [Low Quality Evidence]; Fletcher, 2001 [Guideline]; Sullivan, 1996 [Low Quality Evidence]; Belardinelli, 1995 [Low Quality Evidence]; Coats, 1992 [Moderate Quality Evidence])

Make sure to arrange home care services or home care physical therapy to aid the patient with activities of daily living.

In patients with stable heart failure, a low-intensity home walking exercise (HWE) program is effective in improvement of functional status and symptoms. HWE programs involving home visits will have more compliance from the patients compared to hospital-based cardiac rehabilitation programs *(Corvera-Tindel, 2004 [Moderate Quality Evidence])*. Comprehensive outpatient rehabilitation programs for patients with NYHA class II and III heart failure for even 12 weeks improve exercise capacity and quality of life *(Meyer, 2003 [Low Quality Evidence])*.

Phase 3 Cardiac Rehabilitation

Phase 3 programs are outpatient non-monitored. At hospital discharge, patients should receive an exercise prescription based on tolerance to in-hospital activity, risk factors and stress testing (if done). Unless there is a long-term effort of encouragement, most patients will revert to previous sedentary activities *(Holmback, 1994 [Moderate Quality Evidence])*.

Phase 3 cardiac rehabilitation emphasizes exercise training and activity prescription, risk factor modification, and psychosocial evaluation and counseling in an attempt to lower morbidity and mortality. The following should be considered when writing an exercise prescription:

- **Exercise treatment.** Education about the signs and symptoms of overexertion, angina and cardiopulmonary distress is important.
- **Type of exercise.** Aerobic exercise is emphasized. It includes any activity that preferentially uses large muscle groups and can be maintained for a prolonged period (e.g., walking). Pure isometric exercise should be minimized because it may result in LV decompensation in patients with poor LV function.
- **Intensity of exercise.** This should be based on an exercise tolerance test or the MET level at discharge from phase 2 rehabilitation. In patients with an angina threshold of 2-3 METs, exercise training may not be appropriate. In general, moderate intensity (to 40-60% of functional capacity) is advisable during the first weeks of conditioning, with a goal to reach 40-85%, or that of the functional capacity of the population at large *(Lavie, 2009 [Low Quality Evidence])*.
- **Target heart rate.** This should be determined from an exercise test or a monitored exercise session. If this is not feasible, target heart rate can be calculated as follows:

  220 – age = maximum heart rate

  65% x maximum heart rate = target heart rate

This applies to patients who are not taking a beta-blocker and who have been shown to tolerate the heart rate without ischemia.

- **Monitoring rate of perceived exertion (RPE).** The Borg scale of perceived exertion is a useful tool in guiding exercise programs. It is used in conjunction with the target heart rate when instructing patients on exercise tolerance. Target rates are usually between 11 (fairly light) and 14 (somewhat hard to hard). RPE is advantageous for many reasons: it is unaffected by negative chronotropic medications, unlike heart rate monitoring; it is quite reproducible across age, gender and cultural origin; and lastly, it requires only patient attunement to symptoms *(Squires, 1990 [Low Quality Evidence])*.
- **Duration of exercise.** Initially, multiple 10-minute bouts distributed throughout the day may be optimal for some patients. During the first two to six weeks of participation, exercise duration should be gradually increased from 30 minutes to 45 minutes or more. (This does not include the warm-up, cool-down or stretching periods crucial to any workout.) Duration should be increased to 20-30 minutes before intensity is increased. A steady rate of perceived exertion should be maintained by increasing frequency as tolerated. Patients should exercise for 200 minutes per week in four to five divided sessions.
- **Frequency of exercise.** From the onset, exercise frequency should be three to five times per week.

Smoking Cessation

Cigarette smoking increases the incidence of congestive heart failure *(American Heart Association, 2005 [Guideline])*. In fact, using the NHANES I epidemiologic data, cigarette smoking is clearly an independent risk factor for heart failure *(He, 2001 [Low Quality Evidence])*. As heart failure continues to increase in prevalence in the U.S., we will need to look at how to prevent decompensation of previously stable heart failure patients. Several precipitating factors have been suggested in the relapse of heart failure, and one of them includes cigarette smoking *(He, 2001 [Low Quality Evidence])*. Cigarette smoking activates the sympathetic nervous system, which causes an elevation in blood pressure and heart rate, which in turn increases myocardial oxygen consumption *(Narkiewicz, 1998 [Low Quality Evidence]; Winniford 1986 [Low Quality Evidence])*. Smoking also decreases myocardial oxygen supply, due to reduced diastolic filling time and increased carboxyhemoglobin level *(Nicolozakes, 1988 [Low Quality Evidence])*. Both increases in myocardial oxygen consumption and decreased myocardial oxygen supply have adverse effects on the heart. Research on the participants in the Study of Left Ventricular Dysfunction (SOLVD) showed that current cigarette smoking increased mortality and hospitalization compared to ex-smokers and those who never smoked *(Suskin, 2001 [Low Quality Evidence])*. Quitting smoking is associated with a significant decrease in risk of all-cause mortality among patients with coronary heart disease *(Critchley, 2003 [Systematic Review])*. In fact, in patients with left ventricular dysfunction, research on the SOLVD participants showed that quitting smoking substantially decreases morbidity and mortality within two years *(Suskin, 2001 [Low Quality Evidence])*.

For more information, see the ICSI Preventive Services for Adults guideline.

Self-management and treatment adherence

Comprehensive education and counseling are essential for patients and caregivers to gain an understanding of disease process and recommendations for disease management. The goals should focus on giving patients, family and caregivers the knowledge and self-care tools to effectively engage in treatment plans. Emphasis should be placed on understanding the definition and cause of the patient's heart failure, symptom recognition, medication usage and indications, risk factor modification, diet, activity and lifestyle recommendations, and the importance of treatment adherence. Social factors should also be considered: does the patient have resources to obtain medications, a system for setting up medications and remembering to take them. Does the patient have transportation to appointments for follow-up? Community resources may need to be utilized to support the patient and the family in engaging in the plan-of-care.

Return to Algorithm *Return to Table of Contents*

Hospital discharge and reducing hospital readmissions

Comprehensive discharge planning with detailed written instructions for the patient and caregivers should be implemented to promote compliance and understanding of treatment and educational goals. Family and caregivers must be considered as part of the care team and engaged in follow-up planning during the hospitalizations. Discharge preparation should assess the patient and family understanding of the illness, and their ability to state what they will need to do in the next care setting rather than delivery of information alone. Discharge instructions should address medication regimes, dietary instructions for sodium and fluid restriction recommendations, activity level, weight monitoring and what to do if symptoms return or worsen. A discharge follow-up appointment should be scheduled within 7-10 days *(Lindenfeld, 2010 [Guideline])* to assess the patient's status, titrate medications toward recommended target doses, and to reinforce and supplement education initiated in the hospital. Communication with the next care clinician needs to be completed in a timely manner so all care team members in all settings have the information necessary to care for the patient. Accountability needs to be assigned for care so the patient doesn't "fall between the cracks" of hospital, primary and specialty care *(Jessup, 2009 [Guideline]; Arnold, 2008 [Guideline]; Adams, 2006 [Guideline]; Arnold, 2006 [Low Quality Evidence]; Koelling, 2005 [Moderate Quality Evidence])*.

For patients with advanced heart failure or recurrent admissions for heart failure, consider the following post-discharge management plan: a scale present in the home; visiting nurse or telephone follow-up generally no longer than three days after discharge; and referral for disease management, if available *(Lindenfeld, 2010 [Guideline]; Rich, 1995 [Moderate Quality Evidence])*.

The ability of a patient to understand disease process and actively participate in heart failure management is linked to better outcomes. It is important for health care clinicians to assess health literacy and provide additional support, educational resources to enhance self-care and optimize heart failure treatment in those with low health literacy *(Evangelista, 2010 [Low Quality Evidence]; Driscoll, 2009 [Low Quality Evidence])*.

Readmissions

In this ever-changing health care environment, both quality and cost measures have forced us to focus on readmissions. Heart failure is a key targeted diagnosis to help reduce readmissions. Emergency center-based observation units are a cost-effective way to treat heart failure patients. They can save money for the organization, while improving quality of care and reducing length of stay for the patient.

Heart failure is the leading cause of hospitalization and readmission among older adults *(Roger, 2012 [Low Quality Evidence])*. Medicare patients who are hospitalized for heart failure have a 30-day readmission rate of 20-25% *(Jencks, 2009 [Low Quality Evidence])*. Readmissions are complex, involving complex patients with multiple comorbidities. Most of the information that evaluates the cause of readmissions is based on retrospective chart analysis *(Ashton, 1995 [Low Quality Evidence])*. A recently published study looked at patient perspectives regarding the causes of readmission. There were five main themes gathered from the study: "distressing symptoms, unavoidable progression of illness, psychosocial factors, adherence with self-care recommendations, and health system failures" *(Retrum, 2012 [Low Quality Evidence])*. The patient's perspectives were that there was a lack of clinician communication between the inpatient and outpatient environment; they may have been discharged too early; and broad concerns including medical, social, and economic in the post-discharge environment that were not adequately addressed *(Retrum, 2012 [Low Quality Evidence])*.

The RARE (Reduce Avoidable Readmissions Effectively) campaign was established to reduce avoidable readmissions. They have devised five key areas that must be addressed to reduce readmissions: 1) Patient and family engagement and activation, 2) Medication management, 3) Comprehensive transition planning, 4) Care transition support, and 5) Transition communication.

This complex subject requires additional study prior to the development of specific recommendations. However, individual clinicians and health systems will likely need to create coordinated, multidisciplinary

and customized programs that meets the specific needs of the patients that they care for. The programs should be designed to be within the scope and resources of the health care clinicians and health systems.

General discharge criteria for patients admitted with Heart Failure and recognized by other Heart Failure Guidelines (HSFA and AHA Get with the Guidelines-Heart Failure) prior to discharge are:

- Heart failure exacerbating factors addressed
- Near optimum fluid status and pharmacologic therapy achieved, have transition from IV to oral diuretic completed
- Patient education completed with clear discharge instructions
- Follow-up clinic visit scheduled within seven days

The relationship between early physician follow-up and 30-day readmission among Medicare beneficiaries hospitalized for heart failure was looked at by Dr. Fonarow and Dr. Yancy, who found that only 38% of discharge heart failure patients were seen within seven days and for patients seen earlier, readmissions were decreased from 23 to 20% *(Hernandez, 2010 [Low Quality Evidence])*.

Other recommendations prior to discharge that should be considered in patient with advanced heart failure to decrease risk of recurrent admissions:

- Oral regimen stable for 24 hours
- No IV inotrope or vasodilator for 24 hours
- Ambulation before discharge to assess functional capacity
- Plans for post-discharge management
- Referral for disease management, if available

Transitional care

Transitional care, or transition of care, is an important aspect of patient management that promotes continuity of care and facilitates safe and timely transfer of patient from one level of care to another. Transitional care particularly targets patients at high risk for heart failure readmission: for example, those with previous hospitalizations, multiple comorbidities or medications, cognitive or functional impairment, depression or limited social support. Patients with new-onset heart failure should be assessed and treated with some urgency, because their mortality is high within the first few weeks after diagnosis. Referral to a comprehensive heart failure disease management program should be considered for these high-risk patients. Many models of chronic disease management programs in primary care are being developed across the country and offer great opportunities for innovative care delivery to heart failure patients and their families. This heart failure care, however, should complement but not replace the other comprehensive care delivered by the PCP *(Arnold, 2008 [Guideline])*.

Heart failure management programs

Referral to a comprehensive heart failure clinic or heart failure disease management program should be considered if the patient has complex medical problems or is at high risk for rehospitalization. High-risk patients include those with renal insufficiency, low output state, diabetes, chronic obstructive pulmonary disease, persistent NYHA class III or IV symptoms, frequent hospitalizations for any cause, multiple comorbidities, a history of depression, cognitive impairment, inadequate social support, poor health literacy, or persistent non-adherence to therapeutic regimens. Heart failure disease management programs have been shown to improve patient outcomes. Studies have been done looking at the management of heart failure using both clinic and home-based care models. These have shown convincing evidence that the rate of

Return to Algorithm *Return to Table of Contents*

rehospitalization and costs can be significantly reduced as well as quality of life improved *(Lindenfeld, 2010 [Guideline]; Whellan, 2007 [Cost-Effective Analysis]; Adams, 2006 [Guideline]; Gregory, 2006 [Low Quality Evidence]; Sisk, 2006 [Low Quality Evidence]; Hartmann, 2004 [Low Quality Evidence]; Logeart, 2004 [Low Quality Evidence]; Redfield, 2002 [Low Quality Evidence]; Philbin, 1999 [Low Quality Evidence]; Rich, 1995 [Moderate Quality Evidence])*.

The Heart Failure Society of America (HFSA) recommends that HF disease management programs include multiple components based on patient characteristics and needs. Disease management components in a HF clinic include but are not limited to the following:

Comprehensive education and counseling individualized to patient needs

Promotion of self-care, including self-adjustment of diuretic therapy in appropriate patients (or with family member/caregiver assistance)

Emphasis on behavioral strategies to increase adherence

Vigilant follow-up after hospital discharge or after periods of instability

Optimization of medical therapy

Increased access to clinicians

Early attention to signs and symptoms of fluid overload

Assistance with social and financial concerns

(Hauptman, 2008 [Guideline])

Telemonitoring programs

Telephone intervention programs that include education to improve dietary and treatment compliance along with teaching self-monitoring of heart failure signs and symptoms have been shown to decrease heart failure admissions. However, telephone-based interactive voice response systems that collect daily information about symptoms and weight for review by the patient's clinicians have not been shown to improve outcomes. A multicenter, randomized, controlled trial of 1,653 patients found no reduction in the risk of readmission or death from any cause with telemonitoring as compared to usual care. Moreover, there were no reductions in the risk of hospitalization for heart failure, the number of days in the hospital, or the time to readmission or death. The study authors conclude that there remains a need for strategies to improve heart failure outcomes. The findings also indicate the importance of a thorough, independent evaluation of disease-management strategies before their widespread adoption *(Chaudhry, 2010 [Low Quality Evidence]; Ferrante, 2010 [Moderate Quality Evidence])*.

Stress reduction

Stress reduction and relaxation techniques may be of benefit to some patients and should be encouraged.

(Mandle, 1996 [Low Quality Evidence]; Beary, 1974 [Low Quality Evidence])

Depression, adjustment disorder with depression

Major depression is common in patients hospitalized with heart failure and is independently associated with a poor prognosis *(Jiang, 2001 [Low Quality Evidence])*. It is independently associated with a substantial increase in the risk of heart failure among older persons with isolated systolic hypertension. This association does not appear to be mediated by myocardial infarction *(Abramson, 2001 [Moderate Quality Evidence])*. An increasing number of depressive symptoms is a negative prognostic factor for patients with heart failure, just as it is for patients with CHD *(Vaccarino, 2001 [Moderate Quality Evidence])*. It is an independent risk factor for functional decline and death in heart failure patients *(Guck, 2003 [Low Quality Evidence])*.

Return to Algorithm *Return to Table of Contents*

Frequently, depression is not assessed, or not treated adequately or at all. As yet there are no data to support the hypothesis that antidepressant treatment improves cardiac morbidity and mortality *(Jiang, 2005 [Low Quality Evidence])*. Nevertheless, consensus opinion is to treat depressed cardiac patients with a safe drug rather than watchful waiting since they would benefit from symptomatic relief of their depressive symptoms, and there is a potential improvement in their cardiovascular risk profile *(Ballenger, 2001 [Low Quality Evidence])*.

Although tricyclic antidepressants are affective against depression, they are associated with cardiovascular side effects including orthostatic hypotension, slowed cardiac conduction, antiarrythmic activity and increased heart rate. SSRIs, by contrast, are well tolerated and have a more benign cardiovascular profile and would be preferred initial agents for treatment of depression in individuals with cardiovascular disease *(Jiang, 2005 [Low Quality Evidence])*.

Though well-designed studies are still needed, it would appear that the newer agents, particularly the SSRIs, are preferred over the TCAs, which should be avoided when treating depression in patients with heart failure *(Jessup, 2009 [Guideline]; Guck, 2003 [Low Quality Evidence])*.

Advance Directives

The Minnesota Health Care Directive, established by the Minnesota Legislature in 1998, is a written document that satisfies State of Minnesota requirements for advance directives. This document combines the functions of a Living Will and Durable Power of Attorney for Health Care into a single legal declaration; i.e., it informs others of the individual's wishes about health care and allows naming a person ("agent") to decide if the individual is unable to decide or if the individual wants someone else to decide. An attorney is not required for document completion, and the document can be amended or revoked at any time by the author.

See the ICSI Preventive Services for Adults guideline for more information.

End-of-Life Considerations

Ongoing patient and family education regarding prognosis for functional capacity and survival is recommended for patients with heart failure as part of disease management. A patient's status should be medically and psychologically optimized before discussing end-of-life care.

Discussion regarding end-of-life care should be considered for those with advanced heart failure with symptoms at rest despite repeated attempts to optimize medical treatments. Whenever possible, the patient should be involved in the decision-making process. Discussion is recommended regarding the option of inactivating ICDs for patients with heart failure at the end-of-life. Aggressive procedures performed within the final days of life are not appropriate. See the ICSI Palliative Care guideline for more information.

(Hunt, 2005 [Guideline])

Return to Algorithm *Return to Table of Contents*

15. Symptom Control Satisfactory?

Recommendation:

- Consider reassessment of ventricular function (echocardiography or radionuclide ventriculography) if the symptoms persist despite changes in pharmacologic management, or if symptoms markedly change.

Return to Algorithm *Return to Table of Contents*

16. Consider Specialty Referral

Recommendations:

- Primary care clinicians should continue to be involved in the decision-making process when subspecialty consultation and management are necessary.
- Communication between the primary caregiver and the cardiologist is key and should be encouraged even before the need for a referral in order to integrate seamless diagnostic and therapeutic care.

Once it has been determined that the patient is a candidate for revascularization, the next step is angiography performed by a cardiologist. Subspecialty consultation will generally involve not only performance of the procedure but also recommendations for further management. Primary care clinicians should continue to be involved in the decision-making process. Primary care clinicians should also be familiar with risks associated with various patterns of disease distribution seen on angiogram. The decision to proceed with revascularization must be determined on an individual basis. Consultation should take place among the patient, primary care clinician, cardiologist and cardiovascular surgeon to determine the most appropriate course of action.

If the results of the angiogram do not show significant CAD or if the decision is made not to proceed with revascularization, pharmacological management should be continued (see Annotation #13, "Pharmacologic Management").

Device/surgical

- Cardiac transplantation is the only established surgical approach to treatment of refractory heart failure. Additional interventional and surgical treatments of refractory heart failure are experimental.
- Cardiac resynchronization therapy (CRT)

  CRT or biventricular pacing can be used to treat individuals who remain symptomatic despite optimal medical therapy. By treating ventricular dyssynchrony (as evidenced by wide QRS greater than or equal to 0.12 sec), CRT has resulted in significant improvements in measurements of quality of life, functional class, exercise capacity, degree of mitral regurgitation, and left ventricular ejection fraction (LVEF). Meta-analyses of several CRT trials have shown reduced heart failure hospitalizations and a reduction of mortality with or without implantable cardioversion defibrillator (ICD) therapy.

  Indications for CRT are outlined in the ACC/AHA/HRS 2008 Guidelines for Device-Based Therapy of Cardiac Rhythm abnormalities. CRT with or without ICD is indicated for patients with NYHA functional class III or IV symptoms of heart failure despite optimal medical therapy, LVEF less than or equal to 35%, a QRS duration of greater than or equal to 0.12 seconds, and sinus rhythm *(Epstein, 2008 [Guideline])*.

  For patients who meet the criteria for CRT with or without ICD but who are in atrial fibrillation or who have frequent dependence on right ventricular pacing, CRT is a reasonable therapy.

  Patients with dyssynchrony but only New York Heart Associations Class I or II symptoms of heart failure do not currently have indications for CRT therapy.

  CRT is not recommended for patients whose functional status or life expectancy is limited by chronic non-cardiac conditions.

Return to Algorithm *Return to Table of Contents*

- Implantable cardioverter defibrillator (ICD)

  ICD therapy is indicated for both primary and secondary prevention of sudden cardiac death in qualifying heart failure patients.

- Secondary prevention

  Patients with prior cardiac arrest or documented sustained ventricular arrhythmias (ventricular fibrillation [VF] or hemodynamically unstable sustained ventricular tachycardia [VT]) have a high risk of recurrence. If completely reversible causes are excluded, an ICD placement is indicated for secondary prevention to reduce mortality in this patient population.

- Primary prevention

  Patients with low LVEF, from either ischemic or non-ischemic etiology, are at increased risk of sudden cardiac death. Primary prevention refers to placement of an ICD for those individuals at risk but who have not yet had an episode of sustained VT, VF or cardiac arrest.

  ICD therapy is indicated for primary prevention in patients with LVEF less than 35% due to prior MI who are at least 40 days post-MI and are in NYHA functional Class II or III.

  For similar patients in NYHA functional Class I, the LVEF requirement for ICD placement for primary prevention is less than 30%.

  For similar patients with LVEF less than 40% and non-sustained VT, ICD placement is indicated if inducibile VF or sustained VT can be demonstrated at electrophysiology study.

  For patients with non-ischemic dilated cardiomyopathy, in NYHA functional Class II or III, ICD therapy is indicated for LVEF less than or equal to 35% and may be considered for those in NYHA functional Class I (Class IIb indication).

  Patients with reduced left ventricular systolic function, heart failure and syncope of unclear origin have an increased rate of subsequent death. These patients should be considered for referral for ICD evaluation.

  ICD placement should not be considered for patients who do not have a life expectancy of at least one year with acceptable functional status.

- Radiofrequency catheter ablation may be indicated in patients with heart failure and reciprocating tachycardias or selected patients with atrial fibrillation. However, there is insufficient data on the role of ablation on sustained ventricular tachycardias in patients with heart failure.

- Revascularization should be considered in patients with significant coronary artery disease defined as left main disease, three-vessel disease, or two-vessel disease with proximal LAD involvement, with evidence of ischemia.

- Valve surgery is indicated in patients with severe left ventricular dysfunction, and severe mitral valve insufficiency or aortic stenosis surgery may lead to symptomatic improvement in selected heart failure patients.

Left ventricular assist devices

- The devices provide hemodynamic support as a bridge to cardiac transplantation and for the treatment of severe myocarditis.

- Left ventricular assist devices as destination therapy are reasonable in selected patients with refractory end stage heart failure who are not candidates for heart transplant, and who have an estimated one year mortality over 50% with medical therapy (Class IIa indication) *(Epstein, 2008 [Guideline])*.

Return to Algorithm *Return to Table of Contents*

Continuous (not intermittent) infusions of positive inotropic agents such as dobutamine, dopamine or milrinone

- Patients who cannot be weaned from IV to oral therapy on multiple attempts may require placement of a central line to allow for continuous infusion of the above agents.
- This therapy may reduce symptoms at end of life, though it does not prolong life.

Palliative care and hospice

- At the point that heart failure is diagnosed, referral to palliative care services will provide patients and families with access to resources helpful throughout the entire course of illness.
- Hospice should be considered for patients dying of heart failure.
- Palliative use of continuous or intermittent inotropic agents may be helpful in alleviating symptoms.

Please see the ICSI Palliative Care guideline for more information.

(Hunt, 2005 [Guideline]; Abraham, 2002 [Moderate Quality Evidence]; Moss, 2002 [Moderate Quality Evidence]; Hunt, 2001 [Guideline]; Remme, 2001 [Guideline]; Agency for Health Care Policy and Research, 1994a [Low Quality Evidence]; Agency for Health Care Policy and Research, 1994b [Low Quality Evidence])

If patients continue to have symptoms refractory to care, consider referral to subspecialist for/when:

- Patient has progressive or persistent symptoms of heart failure despite optimal medical therapy.
- Patient has severe valvular disease associated with heart failure.
- Patient is intolerant of medical therapy.
- Patient has significant or symptomatic atrial or ventricular arrhythmias.
- Patient has angina refractory to medical therapy or evidence of signficant ischemia on stress testing.
- Patient is likely to need invasive testing such as angiography and subsequent coronary intervention or surgery.
- Patient has LVEF of less than or equal to 40% despite optimal medical therapy.
- Referral to a specialist should be considered if patient is a candidate for CRT therapy (see "Device/surgical" earlier in this section).
- Referral to a specialist should be considered if patient is a candidate for ICD therapy (see "Device/surgical" earlier in this section).
- NYHA Class III or IV symptoms are refractory to medical management, including those patients exhibiting signs of diuretic resistance.
- Symptoms are rapidly progressive in spite of maximal medical management.
- Patients with syncope of unknown cause or those who have undergone cardioversion for ventricular tachycardia or fibrillation should be referred to a cardiologist.
- Patients in whom moderate doses of vasodilating drugs cannot be tolerated for whatever reason.
- Younger people (i.e., less than 60) with NYHA Class I-II heart failure with either severe left ventricular dysfunction, severe left ventricular dilatation, or significant valvular regurgitation. Many of these patients may be candidates for cardiac transplantation or other cardiac surgical procedures. Consultation with a cardiologist should be strongly considered, as well as a diagnostic workup, even in patients with minimal symptoms.

Return to Algorithm *Return to Table of Contents* **www.icsi.org**

- Consider referral for biventricular pacing of NYHA Class III-IV patients who are already on optimized medical therapy and have an LVEF less than or equal to 35% with intraventricular conduction delay (as defined by QRS greater than or equal to 0.12 seconds).
- Patients with NYHA Class I and II symptoms, but EF less than 30% and dyssynchrony, manifested by QRS interval greater than 130 msec, benefited from CRT with or without ICD. CRT did not impact mortality over ICD alone *(Moss, 2009 [Moderate Quality Evidence])*.
- Consider referral for ICD placement in patients with both ischemic and non-ischemic cardiomyopathy that meet guideline recommendations, for the primary prevention of sudden cardiac death.

(Epstein, 2008 [Guideline])

Return to Algorithm *Return to Table of Contents*

17. Ongoing Assessment of Response to Treatment and Evaluation for Symptom Exacerbation

Recommendations:

- After initial evaluation and diagnosis, follow-up of heart failure patients in the ambulatory setting should focus on optimizing pharmacologic therapy and prevention of heart failure exacerbations.
- Patient education should be ongoing and consistently reinforced, and family members should be a part of this process whenever possible. Symptoms of worsening heart failure should be explained, and patients should be advised to contact their clinician or nurse if these symptoms develop.
- Patients should be advised to call their clinician about a greater or equal to two pounds/day weight gain or five or more pounds/week. They can expect the clinician to assess symptoms, adjust diuretics if appropriate, discuss dietary sodium compliance/restriction, review treatment plan, and recommend appropriate level of care (office visit, ER, etc.).
- Also refer to Appendix B, "Strategies to Address Adherence to Treatment Plan."

Accessibility

Intimidation by or frustration with large health care systems and social isolation are factors that distance patients from their health care clinicians. A patient's failure to maintain this contact, as well as inadequate patient education, contribute to poor patient compliance and high hospital admission and readmission rates in this population *(Moser, 1996 [Low Quality Evidence]; Rich, 1995 [Moderate Quality Evidence])*.

- To prevent heart failure exacerbation, efforts and resources should be directed toward early intervention in the form of increased accessibility to care and education aimed at symptom recognition and treatment plan adherence.
- Frequently, patients wait until they are in crisis before seeking medical assistance, bypassing the clinician's office and going straight to the emergency department (ED). Limited hours and limited/untrained staff at clinicians' offices have been cited as reasons patients seek acute care with worsening symptoms of heart failure.
- Case managers and heart failure clinics have been shown to be effective strategies to avert ED visits and hospitalizations by providing patients with a contact person who is familiar with their

Return to Algorithm *Return to Table of Contents*

care in order to expedite treatment alternatives. This contact person, usually a nurse, is available to answer questions and clarify instructions, potentially increasing treatment plan compliance. The nurse should have adequate ancillary support services available, e.g., social workers, dietary.

- A midlevel clinician can provide an appropriate level of care adjusting medications and dosages.
- NTproBNP has been shown to be useful in determining the long-term prognosis of patients with congestive heart failure. After hospital admission, the euvolemic/dry BNP value at discharge can be used as a meaningful and valuable baseline level for subsequent monitoring and management of patients with heart failure.

• Time between visits is important for the patient to formulate questions and assimilate the previously presented information. Family members and caregivers should also be involved in education to support the patient's efforts.

(Kasper, 2002 [Moderate Quality Evidence]; Hafferkamp, 1996 [Low Quality Evidence]; Moser, 1996 [Low Quality Evidence]; Rich, 1995 [Moderate Quality Evidence])

Return to Algorithm *Return to Table of Contents*

The Aims and Measures section is intended to provide protocol users with a menu of measures for multiple purposes that may include the following:

- population health improvement measures,
- quality improvement measures for delivery systems,
- measures from regulatory organizations such as Joint Commission,
- measures that are currently required for public reporting,
- measures that are part of Center for Medicare Services Physician Quality Reporting initiative, and
- other measures from local and national organizations aimed at measuring population health and improvement of care delivery.

This section provides resources, strategies and measurement for use in closing the gap between current clinical practice and the recommendations set forth in the guideline.

The subdivisions of this section are:

- Aims and Measures
- Implementation Recommendations
- Implementation Tools and Resources
- Implementation Tools and Resources Table

Aims and Measures

1. Decrease the readmission rate for patients age 18 years and older and with heart failure diagnosis, within 30 days of discharge following hospitalization for heart failure. *(Annotation #14)*

   Measure for accomplishing this aim:

   a. CMS readmissions measure for heart failure patients at Hospital Compare at http://www.medicare.gov/hospitalcompare/. *(CMS Hospital Compare)*

2. Increase the rate of heart failure patients age 18 years and older who receive optimum evidence-based pharmacologic treatment. *(Annotation #13)*

   Measures for accomplishing this aim:

   a. Percentage of patients aged 18 years and older with a diagnosis of heart failure with a current or prior left ventricular ejection fraction (LVEF < 40%) who were prescribed ACE inhibitor or ARB therapy either within a 12-month period when seen in the outpatient setting OR at **each** hospital discharge. *(AMA-PCPI/ACCF/AHA, Physician Quality Reporting-PQRS 2013 #5)*

   b. Percentage of patients aged 18 years and older with a diagnosis of heart failure with a current or prior left ventricular ejection fraction (LVEF < 40%) who were prescribed beta-blocker therapy either within a 12-month period when seen in the outpatient setting OR at **each** hospital discharge. *(AMA-PCPI/ACCF/AHA, Physician Quality Reporting-PQRS 2013 #8)*

   c. Percentage of patients with heart failure diagnosis and LVSD who at the last clinic visit met the following (if eligible):

      - prescribed or were on ACEI/ARB,
      - prescribed or were on beta-blocker therapy, and
      - a non-smoker.

3. Improve the use of diagnostic testing in order to identify and then appropriately treat adult patients with heart failure. *(Annotation #2)*

   Measure for accomplishing this aim:

   a. Percentage of patients aged 18 years and older with a diagnosis of heart failure for whom the quantitative or qualitative results of a recent or prior (any time in the past) LVEF assessment is documented within a 12-month period. *(AMA-PCPI/ACCF/AHA, Physician Quality Reporting-PQRS 2013 #198)*

Return to Table of Contents

4. Increase the rate of heart failure patients age 18 years and older who have comprehensive patient education and follow-up care. *(Annotation #14)*

   Measures for accomplishing this aim:

   a. Percentage of heart failure patients who have education on the following about their condition:

      - physical activity levels
      - proper diet
      - medications
      - daily weight monitoring
      - what to do if their symptoms worsen

   b. Percentage of heart failure patients who have a follow-up appointment with their primary care clinician within seven days of hospital discharge.

   c. Percentage of heart failure patients who are current smokers or tobacco users who received smoking cessation advice or counseling in primary care.

Return to Table of Contents

Measurement Specifications

Measurement #1a

CMS readmissions measure for heart failure patients at Hospital Compare at http://www.medicare.gov/hospitalcompare/.

Notes

Hospital readmissions data for heart failure patients – Medicare Hospital Compare Web site at http://www.medicare.gov/hospitalcompare/.

Web site link up-to-date as of May 16, 2013.

Return to Table of Contents

Measurement #2a

Percentage of patients aged 18 years and older with a diagnosis of heart failure with a current or prior left ventricular ejection fraction (LVEF < 40%) who were prescribed ACE inhibitor or ARB therapy either within a 12-month period when seen in the outpatient setting OR at **each** hospital discharge.

Notes

This measure is from Centers for Medicare and Medicaid Services (CMS) Physician Quality Reporting System (PQRS). Initiative and information can be found at http://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/PQRS/index.html?redirect=/PQRS/.

Web site link up-to-date as of May 16, 2013.

Return to Table of Contents

Measurement #2b

Percentage of patients aged 18 years and older with a diagnosis of heart failure with a current or prior left ventricular ejection fraction (LVEF < 40%) who were prescribed beta-blocker therapy either within a 12-month period when seen in the outpatient setting OR at **each** hospital discharge.

Notes

This measure is from Centers for Medicare and Medicaid Services (CMS) Physician Quality Reporting System (PQRS). Initiative and information can be found at http://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/PQRS/index.html?redirect=/PQRS/.

Web site link up-to-date as of May 16, 2013.

Return to Table of Contents

Measurement #2c

Percentage of patients with heart failure diagnosis and LVSD who at the last clinic visit met the following (if eligible):

- prescribed or were on ACEI/ARB,
- prescribed or were on beta-blocker therapy, and
- a non-smoker.

Population Definition

Patients age 18 years and older with a diagnosis of heart failure and who have left ventricular systolic dysfunction (LVSD)* and did not have a device.

Data of Interest

$$\frac{\text{\# of patients who were prescribed or were taking ACEI/ARB, beta-blocker therapy and a non-smoker}}{\text{\# of patients with heart failure and LVSD}}$$

Numerator/Denominator Definitions

Numerator: Number of patients 18 years and older with a diagnosis of heart failure and LVSD who were prescribed or were taking ACEI/ARB, beta-blocker therapy and a non-smoker.

Denominator: Number of patients age 18 years and older with a diagnosis of heart failure and LVSD.

ICD-9 codes included: 428.0, 428.1, 428.20, 428.21, 428.22, 428.23, 428.40, 428.41, 428.42, 428.43, 428.9.

Exclusions:

- Patients less than 18 years of age
- Patients with potential contraindications or other reasons for the clinician to not prescribe an ACEI/ARB and/or beta-blocker
- Patients transferred to another care system
- Patients with devices
- Deceased patients

Potential contraindication or other reason for not prescribing an ACEI and a potential reason or other reason for not prescribing an ARB at discharge include:

- ACEI and ARB allergy
- Moderate or severe aortic stenosis
- Physician, nurse practitioner or physician assistant documentation of reasons for not prescribing both an ACEI and ARB at discharge
- ACEI allergy and physician, nurse practitioner or physician assistant documentation for not prescribing an ARB at discharge
- ARB allergy and physician, nurse practitioner or physician assistant documentation for not prescribing an ACEI at discharge
- Patient had a left ventricular assistive device (LVAD) or heart transplant procedure during hospitalization
- Patient reasons (refusal, financial hardship, side effects, etc.)
- Pregnancy

Return to Table of Contents

- Hyperkalemia (ARB)
- Renal insufficiency (ACEI) or renal dysfunction (ARB)

Potential contraindication or other reason for not prescribing a beta-blocker include:

- Allergy to beta-blocker
- Bradycardia less than 50 bpm without beta-blocker therapy
- Advanced heart block (greater than one first-degree AV block) unless treated by pacemaker
- Severe bronchospasms/COPD/asthma/reactive airway disease
- Patient reasons (refusal, financial hardship, side effects, etc.)

Definition of Terms

* Left ventricular systolic dysfunction is defined quantitatively as left ventricular ejection fraction less than 40%, and qualitatively as moderately or severely depressed left ventricular systolic function.

Method of Data Collection

Query EMR records for clinic visits within the last 12 months for patients age 18 years and older with heart failure diagnosis and LVSD. Determine if patients meet any exclusions criteria. For patients who meet exclusions criteria, exclude them from the analysis. For patients who can be included in the analysis, determine whether they were prescribed or were taking ACEI/ARB, and/or beta-blocker therapy and a non-smoker within the last 12 months of the measurement date.

Measurement Period

Quarterly.

Notes

This is a process measure, and improvement is noted as an increase in the rate.

Return to Table of Contents

Measure #3a

Percentage of patients aged 18 years and older with a diagnosis of heart failure for whom the quantitative or qualitative results of a recent or prior (any time in the past) LVEF assessment is documented within a 12-month period.

Notes

This measure is from Centers for Medicare and Medicaid Services (CMS) Physician Quality Reporting Initiative System (PQRS) Initiative and information can be found at http://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/PQRS/index.html?redirect=/PQRS/.

Return to Table of Contents

Measurement #4a

Percentage of heart failure patients who have education on the following about their condition:

- Physical activity levels
- Proper diet
- Medications
- Daily weight monitoring
- What to do if their symptoms worsen

Population Definition

Patients age 18 years and older with a diagnosis of heart failure.

Data of Interest

$$\frac{\text{\# of patients who were educated on the management of their condition}}{\text{\# of patients with heart failure}}$$

Numerator/Denominator Definitions

Numerator: Number of patients 18 years and older with a diagnosis of heart failure who were educated on the management of their condition to include:

- Physical activity levels
- Proper diet
- Medications
- Faily weight monitoring
- What to do if their symptoms worsen

Denominator: Number of patients age 18 years and older with a diagnosis of heart failure.

ICD-9 codes included: 428.0, 428.1, 428.20, 428.21, 428.22, 428.23, 428.40, 428.41, 428.42, 428.43, 428.9.

Method of Data Collection

Query EMR records for clinic visits within the last 12 months for patients age 18 years and older with heart failure diagnosis. Determine whether they had education on the management of their condition to include: physical activity levels, proper diet, medicaitons, daily weight monitoring and what to do if symptoms worsen within the last 12 months of the measurement date.

Measurement Period

Monthly.

Notes

This measure is a process measure, and improvement is noted as an increase in the rate.

Return to Table of Contents

Measurement #4b

Percentage of heart failure patients who have a follow-up appointment with their primary care clinician within seven days of hospital discharge.

Population Definition

Patients age 18 years and older with a diagnosis of heart failure.

Data of Interest

$$\frac{\text{\# of patients who have a follow-up appointment with their primary care clinician within seven days of hospital discharge}}{\text{\# of patients with heart failure}}$$

Numerator/Denominator Definitions

Numerator: Number of patients 18 years and older with a diagnosis of heart failure who have a follow-up appointment with their primary care clinician within seven days of hospital discharge.

Denominator: Number of patients age 18 years and older with a diagnosis of heart failure.

ICD-9 codes included: 428.0, 428.1, 428.20, 428.21, 428.22, 428.23, 428.40, 428.41, 428.42, 428.43, 428.9.

Method of Data Collection

Query EMR records for clinic visits within the last month for patients age 18 years and older with heart failure diagnosis and a hospital discharge within the last month. Determine whether they have a follow-up appointment with their primary care clinician within seven days of hospital discharge.

Measurement Period

Monthly.

Notes

This measure is a process measure, and improvement is noted as an increase in the rate.

Return to Table of Contents

Measurement #4c

Percentage of heart failure patients who are current smokers or tobacco users who received smoking cessation advice or counseling in primary care.

Population Definition

Patients age 18 years and older with a diagnosis of heart failure and current tobacco users.

Data of Interest

$$\frac{\text{\# of patients who received counseling or advice on smoking cessation}}{\text{\# of patients with heart failure and current smokers or tobacco users}}$$

Numerator/Demoninator Definitions

Numerator: Number of patients 18 years and older with a diagnosis of heart failure and current smokers or tobacco users who received counseling or advice on smoking cessation at the last clinic visit.

Denominator: Number of patients age 18 years and older with a diagnosis of heart failure and current smokers or tobacco users.

Method of Data Collection

Query EMR records for clinic visits within the last month for patients age 18 years and older with heart failure diagnosis and are also current smokers or tobacco users. Then, determine the number of patients who received smoking cessation advice or counseling on smoking cessation.

Measurement Period

Monthly.

Notes

This measure is a process measure, and improvement is noted as an increase in the rate.

Descriptions of ICD-9 Codes

ICD-9-CM Code	Description
402.01	Malignant, hypertensive heart disease with heart failure
402.11	Benign, hypertensive heart disease with heart failure
402.91	Unspecified, hypertensive heart disease with heart failure
404.01	Malignant, hypertensive heart and renal disease with heart failure
404.03	Malignant, hypertensive heart and renal disease with heart failure and renal failure
404.11	Benign, hypertensive heart and renal disease with heart failure
404.13	Benign, hypertensive heart and renal disease with heart failure and renal failure
404.91	Unspecified, hypertensive heart and renal disease with heart failure

Return to Table of Contents

404.93	Unspecified, hypertensive heart and renal disease with heart failure and renal failure
428.0	Unspecified, congestive heart failure
428.1	Left heart failure
428.20	Unspecified, systolic heart failure
428.21	Acute systolic heart failure
428.22	Chronic systolic heart failure
428.23	Acute or chronic systolic heart failure
428.30	Unspecified, diastolic heart failure
428.31	Acute diastolic heart failure
428.32	Chronic diastolic heart failure
428.33	Acute or chronic diastolic heart failure
428.40	Unspecified, combined systolic and diastolic heart failure
428.41	Acute combined systolic and diastolic heart failure
428.42	Chronic combined systolic and diastolic heart failure
428.43	Acute or chronic combined systolic and diastolic heart failure
428.9	Unspecified, heart failure

Return to Table of Contents

Implementation Recommendations

Prior to implementation, it is important to consider current organizational infrastructure that address the following:

- System and process design
- Training and education
- Culture and the need to shift values, beliefs and behaviors of the organization

The following system changes were identified by the guideline work group as key strategies for health care systems to incorporate in support of the implementation of this guideline:

- Develop a process that will allow primary care clinicians to identify patients who have been readmitted to the hospital with a diagnosis of heart failure.
- Emphasize patient self-management strategies. These may include heart failure education and other actions designed to sustain engagement of patients with their heart failure care.
- Develop a process to provide education to the patient and/or caregiver in the area of:
  - diet
  - weight monitoring (to include: clinician should be contacted about a two-pound or greater weight gain overnight or a five-pound or greater weight gain during the week)
  - activity level
  - medications
  - the importance of follow-up appointments
  - what to do if symptoms worsen
- Develop a process for timely, early specialty referral for patients with ischemia or those who are refractory despite optimal medical therapy.

Return to Table of Contents

Implementation Tools and Resources

Criteria for Selecting Resources

The following tools and resources specific to the topic of the guideline were selected by the work group. Each item was reviewed thoroughly by at least one work group member. It is expected that users of these tools will establish the proper copyright prior to their use. The types of criteria the work group used are:

- The content supports the clinical and the implementation recommendations.
- Where possible, the content is supported by evidence-based research.
- The author, source and revision dates for the content are included where possible.
- The content is clear about potential biases and when appropriate conflicts of interests and/or disclaimers are noted where appropriate.

Return to Table of Contents

Implementation Tools and Resources Table

Author/Organization	Title/Description	Audience	Web Sites/Order Information
American Association of Heart Failure Nurses	Specialty organization dedicated to advancing nursing education, clinical practice and research, to improve heart failure patient outcomes.	Health Care Professionals	http:// www.AAHFN.org
American College of Cardiology	Offers clinical statements and guidelines to help address contemporary practice issues within the field of cardiology.	Health Care Professionals	http://www.acc.org
American Heart Association	"Go Red for Women" – Physician Tool Kit	Health Care Professionals	http://www.goredforwomen.org
Channing L. Bete, Co.	Learning to Live with Heart Failure; 31-pg handbook	Patients and Families	800-628-7733 item # 93612
Healthwise, Ottawa Hospital Research Institute	Decision aid questions and scoring for "Heart Failure: Should I Get a Pacemaker (cardiac resyncrhonization therapy)?"	Patients and Families	http://decisionaid.ohri.ca/AZsumm.php?ID=1328
Healthwise, Ottawa Hospital Research Institute	Decision aid questions and scoring for "Heart failure: Should I get an implantable cardioverter-defibrillator (ICD)?"	Patients and Families	http://decisionaid.ohri.ca/AZsumm.php?ID=1310
Heart Failure Matters	Offers information to understand heart failure.	Patients and Families	http://www.heartfailurematters.org
Heart Failure Society of America	The Heart Failure Society of America, Inc. (HFSA) represents the first organized effort by heart failure experts from the Americas to provide a forum for all those interested in heart function, heart failure and congestive heart failure research and patient care. Go to the Web site for more information.	Health Care Professionals	http://www.hfsa.org http://www.hfsa.org/journal.asp
Krames Communications	Krames: Cardiac Resynchronization Therapy; 16-pg booklet	Patients and Families	800-333-3032 #11468

Return to Table of Contents

Author/Organization	Title/Description	Audience	Web Sites/Order Information
Mayo Clinic	Heart failure, also known as congestive heart failure (CHF) means your heart can't pump enough blood to meet your body's needs. Any number of underlying heart conditions can lead to heart failure. Over time, conditions such as coronary artery disease or high blood pressure gradually sap your heart of its strength, leaving it too weak or too stiff to fill and pump efficiently. Information at this site include signs and symptoms, causes, risk factors, when to seek medical advice, screening and diagnosis, complications, treatment, prevention, self-care and coping skills.	Health Care Professionals; Patients and Families	http://www.mayoclinic.com/health/heart-failure/DS00061
Minnesota Health Care Directive	MN Department of Health/MN Board of Aging's Senior Linkage	Patients and Families	http://www.health.state.mn.us/divs/fpc/adbrochure.pdf 1-800-333-2433
NIH – Medline Plus	Interactive patient tutorial. Also for print.	Patients and Families	http://www.nlm.nih.gov/medlineplus/tutorials/congestiveheartfailure/htm/index.htm
Ottawa Hospital Research Institute	Ottawa Personal Decision Guides for any patient decision. Includes interactive PDF guide, iShould application for Facebook, Ottawa Family Decision Guide	Patients and Families	http://decisionaid.ohri.ca/decguide.html

Return to Table of Contents

* Available to ICSI members only.

The subdivisions of this section are:

- References
- Appendices

References

Links are provided for those new references added to this edition (author name is highlighted in blue).

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Return to Table of Contents

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Return to Table of Contents

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Return to Table of Contents

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Return to Table of Contents

Appendix A – Heart Failure Classification Comparison

ACC/AHA 2001 Staging Compared to NYHA Functional Classification

ACC/AHA, 2001		NYHA	
A	At high risk of developing HF, but without structural heart disease or symptoms of HF	**None**	
B	Structural heart disease, but without symptoms of HF	**I**	Asymptomatic
C	Structural heart disease with prior or current symptoms of HF	**II**	Symptomatic with moderate exertion
		III	Symptomatic with minimal exertion
		IV	Symptomatic at rest
D	Refractory HF requiring specialized interventions	**IV**	

ACC = American College of Cardiology, AHA = American Heart Association
NYHA = New York Heart Association, HF = Heart Failure

The New York Heart Association (NYHA) classification is a four-level scheme for grading the functional incapacity of patients with cardiac disease. Although criticized for lack of reliability, this system is still widely used.

The ACC/AHA heart failure grading scheme takes into consideration the natural history and progressive nature of heart failure.

For each of these classes, recommendations for prevention, ongoing surveillance for disease progression and specific medical therapy are outlined. This new classification scheme is often used in conjunction with the NYHA classification described above.

Return to Table of Contents

Appendix B – Strategies to Address Adherence to Treatment Plan

The clinician-patient relationship plays an essential role in improving compliance. A member of the health care team should assess for medication and dietary adherence at every visit. The preferred approach is to ask non-threatening, open-ended questions. Include probes for factors that contribute to non-adherence, including adverse reactions, misunderstanding of treatment plan, depression, cognitive impairment, complex dosing regimens and financial constraints.

1. **Assess knowledge of heart failure**
   - When you hear the words "heart failure," what does that mean to you?
   - What do you know about your heart and the type of heart failure you have, and what may have caused it to develop?
2. **Assess adherence to daily weights**
   - Can you tell me why it is important to weigh yourself every day?
   - Are you willing and able to weigh yourself at home every day to monitor for fluid weight gain?
   - Is there anything that might keep you from being able to weigh yourself at home on a regular basis?
3. **Assess adherence to medication regimen**
   - Can you tell me why you are taking this medicine?
   - Do you have a reminder system for taking your medications?
   - In general, how often do you miss or skip doses of any of your prescribed medications for any reason?
   - What is the most common reason(s) you might miss or skip a dose?
   - What do you do when you miss a dose?
   - Is the cost of medication interfering with your treatment?
   - Are you experiencing any unusual symptoms that you think may be due to your medications?
4. **Assess adherence to low-sodium diet**
   - Has your doctor (clinician) ever talked with you about limiting the amount of salt, or sodium you eat?
   - Did your doctor (clinician) tell you how much sodium you can have in a day?
   - Why is it so important for you to follow a low-sodium diet?
   - Who prepares your meals? Is he/she aware of your sodium restriction?
   - What steps do you take to limit the sodium in your diet?
   - What things do you/might you find (most) difficult about following a low-sodium diet?
5. **Assess adherence to treatment plan**
   - Are you comfortable with your ability to follow the treatment plan we have designed for you?
   - What is the most difficult task you have in following your treatment plan?

Source: Marshfield Clinic

Return to Table of Contents BACK

Appendix C – ICSI Shared Decision-Making Model

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The technical aspects of Shared Decision-Making are widely discussed and understood.

- **Decisional conflict** occurs when a patient is presented with options where no single option satisfies all the patient's objectives, where there is an inherent difficulty in making a decision, or where external influencers act to make the choice more difficult.
- **Decision support** clarifies the decision that needs to be made, clarifies the patient's values and preferences, provides facts and probabilities, guides the deliberation and communication and monitors the progress.
- **Decision aids** are evidence-based tools that outline the benefits, harms, probabilities and scientific uncertainties of specific health care options available to the patient.

However, before decision support and decision aids can be most advantageously utilized, a Collaborative Conversation™ should be undertaken between the clinician and the patient to provide a supportive framework for Shared Decision-Making.

Collaborative Conversation™

A collaborative approach toward decision-making is a fundamental tenet of Shared Decision-Making (SDM). The Collaborative Conversation™ is an inter-professional approach that nurtures relationships, enhances patients' knowledge, skills and confidence as vital participants in their health, and encourages them to manage their health care.

Within a Collaborative Conversation™ , the perspective is that both the patient and the clinician play key roles in the decision-making process. The patient knows which course of action is most consistent with his/her values and preferences, and the clinician contributes knowledge of medical evidence and best practices. Use of Collaborative Conversation™ elements and tools is even more necessary to support patient, care clinician and team relationships when patients and families are dealing with high stakes or highly charged issues, such as diagnosis of a life-limiting illness.

The overall framework for the Collaborative Conversation™ approach is to create an environment in which the patient, family and care team work collaboratively to reach and carry out a decision that is consistent with the patient's values and preferences. A rote script or a completed form or checklist does not constitute this approach. Rather it is a set of skills employed appropriately for the specific situation. These skills need to be used artfully to address all aspects involved in making a decision: cognitive, affective, social and spiritual.

Key communication skills help build the Collaborative Conversation™ approach. These skills include many elements, but in this appendix only the questioning skills will be described. (For complete instruction, see O'Connor, Jacobsen "Decisional Conflict: Supporting People Experiencing Uncertainty about Options Affecting Their Health" [2007], and Bunn H, O'Connor AM, Jacobsen MJ "Analyzing decision support and related communication" [1998, 2003].)

1. **Listening skills:**

   Encourage patient to talk by providing prompts to continue such as "*go on, and then?, uh huh,*" or by repeating the last thing a person said, "*It's confusing*."

Return to Table of Contents

Paraphrase content of messages shared by patient to promote exploration, clarify content and to communicate that the person's unique perspective has been heard. The clinician should use his/her own words rather than just parroting what he/she heard.

Reflection of feelings usually can be done effectively once trust has been established. Until the clinician feels that trust has been established, short reflections at the same level of intensity expressed by the patient without omitting any of the message's meaning are appropriate. Reflection in this manner communicates that the clinician understands the patient's feelings and may work as a catalyst for further problem solving. For example, the clinician identifies what the person is feeling and responds back in his/her own words like this: "*So, you're unsure which choice is the best for you*."

Summarize the person's key comments and reflect them back to the patient. The clinician should condense several key comments made by the patient and provide a summary of the situation. This assists the patient in gaining a broader understanding of the situations rather than getting mired down in the details. The most effective times to do this are midway through and at the end of the conversation. An example of this is, "*You and your family have read the information together, discussed the pros and cons, but are having a hard time making a decision because of the risks*."

Perception checks ensure that the clinician accurately understands a patient or family member, and may be used as a summary or reflection. They are used to verify that the clinician is interpreting the message correctly. The clinician can say "*So you are saying that you're not ready to make a decision at this time. Am I understanding you correctly?*"

2. **Questioning Skills**

Open and closed questions are both used, with the emphasis on open questions. Open questions ask for clarification or elaboration and cannot have a yes or no answer. An example would be "*What else would influence you to choose this?*" Closed questions are appropriate if specific information is required such as "*Does your daughter support your decision?*"

Other skills such as summarizing, paraphrasing and reflection of feeling can be used in the questioning process so that the patient doesn't feel pressured by questions.

Verbal tracking, referring back to a topic the patient mentioned earlier, is an important foundational skill (Ivey & Bradford-Ivey). An example of this is the clinician saying, "*You mentioned earlier…*"

3. **Information-Giving Skills**

Providing information and **providing feedback** are two methods of information giving. The distinction between providing information and giving advice is important. Information giving allows a clinician to supplement the patient's knowledge and helps to keep the conversation patient centered. Giving advice, on the other hand, takes the attention away from the patient's unique goals and values, and places it on those of the clinician.

Providing information can be sharing facts or responding to questions. An example is "*If we look at the evidence, the risk is…*" Providing feedback gives the patient the clinician's view of the patient's reaction. For instance, the clinician can say, "*You seem to understand the facts and value your daughter's advice*."

Additional Communication Components

Other elements that can impact the effectiveness of a Collaborative Conversation™ include:

- Eye contact
- Body language consistent with message

Return to Table of Contents

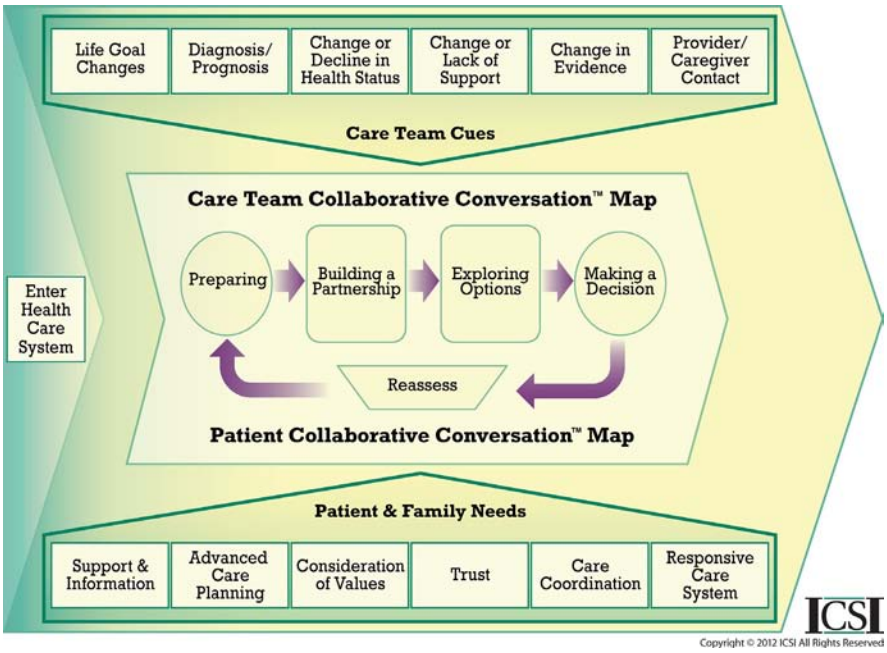
- Respect
- Empathy
- Partnerships

Self-examination by the clinician involved in the Collaborative Conversation™ can be instructive. Some questions to ask oneself include:

- Do I have a clear understanding of the likely outcomes?
- Do I fully understand the patient's values?
- Have I framed the options in comprehensible ways?
- Have I helped the decision-makers recognize that preferences may change over time?
- Am I willing and able to assist the patient in reaching a decision based on his/her values, even when his/her values and ultimate decision may differ from my values and decisions in similar circumstances?

When to Initiate a Collaborative Conversation™

A Collaborative Conversation™ can support decisions that vary widely in complexity. It can range from a straightforward discussion concerning routine immunizations to the morass of navigating care for a life-limiting illness. Table 1 represents one health care event. This event can be simple like a 12 year-old coming to the clinic for routine immunizations, or something much more complex like an individual receiving a diagnosis of congestive heart failure. In either case, the event is the catalyst that starts the process represented in this table. There are cues for clinicians and patient needs that exert influence on this process. They are described below. The heart of the process is the Collaborative Conversation™. The time the patient spends within this health care event will vary according to the decision complexity and the patient's readiness to make a decision.



Regardless of the decision complexity there are cues applicable to all situations that indicate an opportune time for a Collaborative Conversation™. These cues can occur singularly or in conjunction with other cues.

Cues for the Care Team to Initiate a Collaborative Conversation™

- **Life goal changes:** Patient's priorities change related to things the patient values such as activities, relationships, possessions, goals and hopes, or things that contribute to the patient's emotional and spiritual well-being.
- **Diagnosis/prognosis changes:** Additional diagnoses, improved or worsening prognosis.
- **Change or decline in health status:** Improving or worsening symptoms, change in performance status or psychological distress.
- **Change or lack of support:** Increase or decrease in caregiver support, change in caregiver, or caregiver status, change in financial standing, difference between patient and family wishes.
- **Change in medical evidence or interpretation of medical evidence:** Clinicians can clarify the change and help the patient understand its impact.
- **Clinician/caregiver contact:** Each contact between the clinician/caregiver and the patient presents an opportunity to reaffirm with the patient that his/her care plan and the care the patient is receiving are consistent with his/her values.

Patients and families have a role to play as decision-making partners, as well. The needs and influencers brought to the process by patients and families impact the decision-making process. These are described below.

Patient and Family Needs within a Collaborative Conversation™

- **Request for support and information:** Decisional conflict is indicated by, among other things, the patient verbalizing uncertainty or concern about undesired outcomes, expressing concern about choice consistency with personal values and/or exhibiting behavior such as wavering, delay, preoccupation, distress or tension. Generational and cultural influencers may act to inhibit the patient from actively participating in care discussions, often patients need to be given "permission" to participate as partners in making decisions about his/her care.

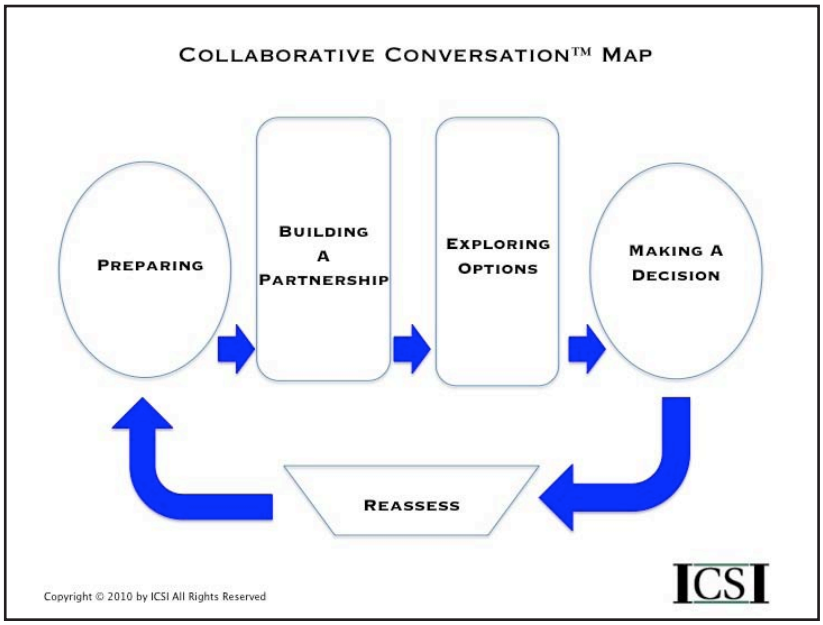
  Support resources may include health care professionals, family, friends, support groups, clergy and social workers. When the patient expresses a need for information regarding options and his/her potential outcomes, the patient should understand the key facts about options, risks and benefits, and have realistic expectations. The method and pace with which this information is provided to the patient should be appropriate for the patient's capacity at that moment.

- **Advance Care Planning:** With the diagnosis of a life-limiting illness, conversations around advance care planning open up. This is an opportune time to expand the scope of the conversation to other types of decisions that will need to be made as a consequence of the diagnosis.
- **Consideration of Values:** The personal importance a patient assigns potential outcomes must be respected. If the patient is unclear how to prioritize the preferences, value clarification can be achieved through a Collaborative Conversation™ and by the use of decision aids that detail the benefits and harms of potential outcomes in terms the patient can understand.
- **Trust:** The patient must feel confident that his/her preferences will be communicated and respected by all caregivers.
- **Care Coordination:** Should the patient require care coordination, this is an opportune time to discuss the other types of care-related decisions that need to be made. These decisions will most likely need to be revisited often. Furthermore, the care delivery system must be able to provide coordinated care throughout the continuum of care.

Return to Table of Contents

- **Responsive Care System:** The care system needs to support the components of patient- and family-centered care so the patient's values and preferences are incorporated into the care he/she receives throughout the care continuum.

The Collaborative Conversation™ Map is the heart of this process. The Collaborative Conversation™ Map can be used as a stand-alone tool that is equally applicable to clinicians and patients as shown in Table 2. Clinicians use the map as a clinical workflow. It helps get the Shared Decision-Making process initiated and provides navigation for the process. Care teams can used the Collaborative Conversation™ to document team best practices and to formalize a common lexicon. Organizations can build fields from the Collaborative Conversation™ Map in electronic medical records to encourage process normalization. Patients use the map to prepare for decision-making, to help guide them through the process and to share critical information with their loved ones.



Evaluating the Decision Quality

Adapted from O'Connor, Jacobsen "Decisional Conflict: Supporting People Experiencing Uncertainty about Options Affecting Their Health" [2007].

When the patient and family understand the key facts about the condition and his/her options, a good decision can be made. Additionally, the patient should have realistic expectations about the probable benefits and harms. A good indicator of the decision quality is whether or not the patient follows through with his/her chosen option. There may be implications of the decision on patient's emotional state such as regret or blame, and there may be utilization consequences.

Decision quality can be determined by the extent to which the patient's chosen option best matches his/her values and preferences as revealed through the Collaborative Conversation™ process.

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ICSI has long had a policy of transparency in declaring potential conflicting and competing interests of all individuals who participate in the development, revision and approval of ICSI guidelines and protocols.

In 2010, the ICSI Conflict of Interest Review Committee was established by the Board of Directors to review all disclosures and make recommendations to the board when steps should be taken to mitigate potential conflicts of interest, including recommendations regarding removal of work group members. This committee has adopted the Institute of Medicine Conflict of Interest standards as outlined in the report, Clinical Practice Guidelines We Can Trust (2011).

Where there are work group members with identified potential conflicts, these are disclosed and discussed at the initial work group meeting. These members are expected to recuse themselves from related discussions or authorship of related recommendations, as directed by the Conflict of Interest committee or requested by the work group.

The complete ICSI policy regarding Conflicts of Interest is available at http://bit.ly/ICSICOI.

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ICSI facilitates and coordinates the guideline development and revision process. ICSI, member medical groups and sponsoring health plans review and provide feedback but do not have editorial control over the work group. All recommendations are based on the work group's independent evaluation of the evidence.

Return to Table of Contents

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Return to Table of Contents

All ICSI documents are available for review during the revision process by member medical groups and sponsors. In addition, all members commit to reviewing specific documents each year. This comprehensive review provides information to the work group for such issues as content update, improving clarity of recommendations, implementation suggestions and more. The specific reviewer comments and the work group responses are available to ICSI members at http://www.HeartFailure.

Return to Table of Contents

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Invited Reviewers

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Return to Table of Contents

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Released in July 2013 for Thirteenth Edition.
The next scheduled revision will occur within 24 months.

Document History

In the Twelfth Edition the following changes were made:

- GRADE was used to classify literature.
- Key Points were changed to Recommendations.
- Order Sets were incorporated into the guideline (2011).

In the Thirteenth Edition the following changes were made:

- The scope and target population of this document no longer include treatment of the adult patient with suspected heart failure in the Emergency Department or in the inpatient hospital setting. The focus was set to the Outpatient management of heart failure.
- The order sets were removed (2013).

Return to Table of Contents

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ICSI Document Development and Revision Process

Overview

Since 1993, the Institute for Clinical Systems Improvement (ICSI) has developed more than 60 evidence-based health care documents that support best practices for the prevention, diagnosis, treatment or management of a given symptom, disease or condition for patients.

Audience and Intended Use

The information contained in this ICSI Health Care Guideline is intended primarily for health professionals and other expert audiences.

This ICSI Health Care Guideline should not be construed as medical advice or medical opinion related to any specific facts or circumstances. Patients and families are urged to consult a health care professional regarding their own situation and any specific medical questions they may have. In addition, they should seek assistance from a health care professional in interpreting this ICSI Health Care Guideline and applying it in their individual case.

This ICSI Health Care Guideline is designed to assist clinicians by providing an analytical framework for the evaluation and treatment of patients, and is not intended either to replace a clinician's judgment or to establish a protocol for all patients with a particular condition.

Document Development and Revision Process

The development process is based on a number of long-proven approaches and is continually being revised based on changing community standards. The ICSI staff, in consultation with the work group and a medical librarian, conduct a literature search to identify systematic reviews, randomized clinical trials, meta-analysis, other guidelines, regulatory statements and other pertinent literature. This literature is evaluated based on the GRADE methodology by work group members. When needed, an outside methodologist is consulted.

The work group uses this information to develop or revise clinical flows and algorithms, write recommendations, and identify gaps in the literature. The work group gives consideration to the importance of many issues as they develop the guideline. These considerations include the systems of care in our community and how resources vary, the balance between benefits and harms of interventions, patient and community values, the autonomy of clinicians and patients and more. All decisions made by the work group are done using a consensus process.

ICSI's medical group members and sponsors review each guideline as part of the revision process. They provide comment on the scientific content, recommendations, implementation strategies and barriers to implementation. This feedback is used by and responded to by the work group as part of their revision work. Final review and approval of the guideline is done by ICSI's Committee on Evidence-Based Practice. This committee is made up of practicing clinicians and nurses, drawn from ICSI member medical groups.

Implementation Recommendations and Measures

These are provided to assist medical groups and others to implement the recommendations in the guidelines. Where possible, implementation strategies are included that have been formally evaluated and tested. Measures are included that may be used for quality improvement as well as for outcome reporting. When available, regulatory or publicly reported measures are included.

Document Revision Cycle

Scientific documents are revised every 12-24 months as indicated by changes in clinical practice and literature. ICSI staff monitors major peer-reviewed journals every month for the guidelines for which they are responsible. Work group members are also asked to provide any pertinent literature through check-ins with the work group midcycle and annually to determine if there have been changes in the evidence significant enough to warrant document revision earlier than scheduled. This process complements the exhaustive literature search that is done on the subject prior to development of the first version of a guideline.

Return to Table of Contents