

Section 1: Development and Implementation of a Comprehensive Heart Failure Practice Guideline

Introduction

Heart failure (HF) is a syndrome characterized by high mortality, frequent hospitalization, poor quality of life, and multiple comorbidities. As a result, heart failure management inevitably involves both a multidimensional assessment process and a complex therapeutic regimen. Knowledge about the pathophysiology and treatment of HF continues to accumulate very rapidly so that individual clinicians may be unable to readily and adequately synthesize new information into effective principles of care for patients with this syndrome. Trial data, though valuable, often do not give adequate direction for individual patient management.

Given the complex and changing picture of HF and the accumulation of evidence-based HF therapy, it is not possible for the clinician to rely solely on personal experience and observation to guide therapeutic decisions. This situation is exacerbated because HF is now a chronic condition in most patients, meaning that the outcome of therapeutic decisions might not be apparent for several years. The natural history and prognosis of individual patients differs considerably, making it difficult to generalize. Treatments might not dramatically improve symptoms of the disease process, yet might prevent or delay its progression and the occurrence of morbid events and deaths. The assessment of specific therapeutic outcomes is complicated by the potential differential impact of various cotherapies.

The complexity of HF, its high prevalence, and the availability of many therapeutic options make it an ideal area for practice guidelines. Additional assumptions driving the development of HF guidelines are presented in [Table 1.1](#).

The first HF guideline developed by the Heart Failure Society of America (HFSA) had a narrow scope, concentrating on the pharmacologic treatment of chronic, symptomatic left ventricular dysfunction.¹ It did not consider subsets of the clinical syndrome of HF, such as acute decompensated HF and “diastolic dysfunction,” or issues such as prevention. The subsequent comprehensive clinical practice guideline published in 2006 addressed a full range of topics including prevention, evaluation, disease management, and pharmacologic and device therapy for patients with HF.² The 2010 guideline updates and expands each of these areas and adds a section on the Genetic Evaluation of Cardiomyopathy published separately in 2009.³ The discussion of end of life management has also been considerably expanded.

HFSA Guideline Approach to Medical Evidence

Two considerations are critical in the development of practice guidelines: assessing strength of evidence and

determining strength of recommendation. Strength of evidence is determined both by the type of evidence available and the assessment of validity, applicability, and certainty of a specific type of evidence. Following the lead of previous guidelines, strength of evidence in this guideline is heavily dependent on the source or type of evidence used. The HFSA guideline process has used three grades (A, B, or C) to characterize the type of evidence available to support specific recommendations ([Table 1.2](#)).

It must be recognized, however, that the evidence supporting recommendations is based largely on population responses that may not always apply to individuals within the population. Therefore, the totality of data may support overall benefit of one treatment over another but cannot assure that all patients will respond similarly. Thus, guidelines can best serve as evidence-based recommendations for management, not as mandates for management in every patient. Furthermore, it must be recognized that trial data on which recommendations are based have often been carried out with background therapy not comparable to therapy in current use. Therefore, physician decisions regarding the management of individual patients may not always precisely match the recommendations. A knowledgeable physician who integrates the guidelines with pharmacologic and physiologic insight and knowledge of the individual being treated should provide the best patient management.

Strength of Evidence A. Randomized controlled clinical trials provide what is considered the most valid form of guideline evidence. Some guidelines require at least 2 positive randomized clinical trials before the evidence for a recommendation can be designated level A. The HFSA guideline committee has occasionally accepted a single randomized, controlled, outcome-based clinical trial as sufficient for level A evidence when the single trial is large with a substantial number of endpoints and has consistent and robust outcomes. However, randomized clinical trial data, whether derived from one or multiple trials, have not been taken simply at face value. They have been evaluated for: (1) endpoints studied, (2) level of significance, (3) reproducibility of findings, (4) generalizability of study

Table 1.1. Assumptions Underlying HFSA Practice Guideline

Clinical decisions must be made.
Correct course of action may not be readily apparent.
Multiple non-pharmacologic, pharmacologic, and device therapies are available.
Reasonably valid methods exist to address knowledge base and evaluate medical evidence.
Data beyond randomized clinical trials exist that enhance medical decision making.
Uncertainties remain concerning approaches to treatment after review of totality of medical evidence.
Expert opinion has a role in management decisions when Strength of Evidence A data are not available to guide management.
A consensus of experts remains the best method of management recommendations when Strength of Evidence A data are not available.

Table 1.2. Relative Weight of Evidence Used to Develop HFSA Practice Guideline

Hierarchy of Types of Evidence	
Level A	Randomized, Controlled, Clinical Trials May be assigned based on results of a single methodologically rigorous trial
Level B	Cohort and Case-Control Studies Post hoc, subgroup analysis, and meta-analysis Prospective observational studies or registries
Level C	Expert Opinion Observational studies-epidemiologic findings Safety reporting from large-scale use in practice

results, and (5) sample size and number of events on which outcome results are based.

Strength of Evidence B. The HFSA guideline process also considers evidence arising from cohort studies or smaller clinical trials with physiologic or surrogate endpoints. This level B evidence is derived from studies that are diverse in design and may be prospective or retrospective in nature. They may involve subgroup analyses of clinical trials or have a case control or propensity adjusted design using a matched subset of trial populations. Dose-response studies, when available, may involve all or a portion of the clinical trial population. Evidence generated from these studies has well-recognized, inherent limitations. Nevertheless, their value is enhanced through attention to factors such as pre-specification of hypotheses, biologic rationale, and consistency of findings between studies and across different populations.

Strength of Evidence C. The present HFSA guideline makes extensive use of expert opinion, or C-level evidence. The need to formulate recommendations based on expert opinion is driven primarily by a paucity of evidence in areas critical to a comprehensive guideline or by evidence derived from a study population not fully representative of the broad spectrum of HF patients. For example, the diagnostic process and the steps used to evaluate and monitor patients with established HF have not been the subject of clinical studies that formally test the accuracy of one approach versus another. In addition, trials often enroll patients that differ from the general HF population in age or gender distribution and in background therapies. In situations such as these, recommendations must be based on expert opinion or go unaddressed.

The value of expert opinion as a form of evidence remains disputed. Many contend that expert opinion is a weak form of observational evidence, based on practice experience and subject to biases and limitations. Advocates believe expert opinion represents a complex synthesis of observational insights into disease pathophysiology and the benefits of therapy in broad populations of patients. They stress the value of the interchange of experience and ideas among colleagues, who collectively treat thousands of patients. Through contact with numerous individual health care providers who may discuss patients with

them, experts are exposed to rare safety issues and gain insight into the perceptions of practitioners concerning the efficacy of particular treatments across a wide spectrum of HF.

Despite the case that can be made for its value, recommendations based on expert opinion alone have been limited to those circumstances when a definite consensus could be reached across the guideline panel and reviewers.

HFSA Guideline Approach to Strength of Recommendation

Determining Strength. Although level of evidence is important, the strength given to specific recommendations is critical. The process used to determine the strength of individual recommendations is complex. The goal of guideline development is to achieve the best recommendations for evaluation and management, considering not only efficacy, but the cost, convenience, side effect profile, and safety of various therapeutic approaches. The HFSA guideline committee often determined the strength of a recommendation by the “totality of evidence,” which is a synthesis of all types of available data, pro and con, about a particular therapeutic option.

Totality of Evidence. Totality of evidence includes not only results of clinical trials, but also expert opinion and findings from epidemiologic and basic science studies. Agreement among various types of evidence, especially from different methodologies, increases the likelihood that a particular therapy is valuable. Although many equate evidence-based medicine with the results of a few individual clinical trials, the best judgment seems to be derived from a careful analysis of all available trial data combined with integration of results from the basic laboratory and the findings of epidemiologic studies.

Scale of Strength. The HFSA guideline employs the categorization for strength of recommendation outlined in Table 1.3. There are several degrees of favorable recommendations and a single category for therapies felt to be not effective. The phrase “is recommended” should be taken to mean that the recommended therapy or management process should be followed as often as possible in individual patients. Exceptions are carefully delineated. “Should be considered” means that a majority of patients should receive the intervention, with some discretion

Table 1.3. HFSA System for Classifying the Strength of Recommendations

“Is recommended”	Part of routine care
“Should be considered”	Exceptions to therapy should be minimized
	Majority of patients should receive the intervention
“May be considered”	Some discretion in application to individual patients should be allowed
	Individualization of therapy is indicated
“Is not recommended”	Therapeutic intervention should not be used

involving individual patients. “May be considered” means that individualization of therapy is indicated (Table 1.3). When the available evidence is considered to be insufficient or too premature, or consensus fails, issues are labeled unresolved and included as appropriate at the end of the relevant section.

Process of Guideline Development

Key steps in the development of this guideline are listed in Table 1.4. Having determined the broad scope of the current guideline, subcommittees of the guideline committee were formed for each section of the guideline. Literature searches with relevant key words and phrases for each guideline section were provided to members of the subcommittees and the full Guideline Committee. Members of each subcommittee were asked to review the search and identify any additional relevant medical evidence for each assigned section. Changes in recommendation and background were carried out by each subcommittee with conference calls directed by the Guideline Committee chair. Each section was presented for comments and consensus approval to the Guideline Committee. Once subsections were complete, the Executive Council reviewed and commented on each section and these comments were returned to the Guideline Committee for changes and once complete, for final approval by the Executive Council. Appendix A provides a grid showing changes to the 2006 guideline.

Consensus. The development of a guideline involves the selection of individuals with expertise and experience to drive the process of formulating specific recommendations and producing a written document. The role of these experts goes well beyond the formulation of recommendations supported by expert opinion.

Experts involved in the guideline process must function as a collective, not as isolated individuals. Expert opinion is not always unanimous. Interpretations of data vary. Disagreements arise over the generalizability and applicability of trial results to various patient subgroups. Experts are

influenced by their own experiences with particular therapies, but still generally agree on the clinical value of trial data. Discomfort with the results of trials reported as positive or negative generally focus on factors that potentially compromise the evidence. Unfortunately, there are no absolute rules for downgrading or upgrading trial results or for deciding whether the limitations of the trial are sufficient to negate what has been regarded as a traditionally positive or negative statistical result.

The HFSA guideline committee sought resolution of difficult cases through consensus building. An open, dynamic discussion meant that no single voice was allowed to dominate. Written documents were essential to this process, because they provided the opportunity for feedback from all members of the group. On occasion, consensus of opinion was sufficient to override positive or negative results of almost any form of evidence. The HFSA process had a strong commitment to recommendations based on objective evidence rigorously reviewed by a panel of experts.

Issues that caused particular difficulty for the HFSA guideline process usually were some of the more important ones faced by the committee, because they mirrored those that are often most challenging to clinicians in day-to-day practice. The foundation of the HFSA guideline process was the belief that the careful judgment of recognized opinion leaders in these controversial areas is more likely to be correct than ad hoc decisions made “on the spot” by physicians in practice.

The involvement of many groups in the development of this guideline helped avoid the introduction of real or perceived bias, which can be personal, practice-based, or based on financial interest. Committee members and reviewers from the Executive Council received no direct financial support from the HFSA or any other source for the development of the guideline. Support was provided by the HFSA administrative staff, but the writing of the document was performed on a volunteer basis primarily by the Committee. Information concerning financial relationships that might represent conflicts of interest was collected annually from all members of the Guideline Committee and the Executive Council. Current relationships are shown in Appendix C.

Table 1.4. Steps in the Development of the 2010 HFSA Practice Guideline

Determine the scope of the practice guideline
Form subcommittees with expertise for each guideline section
Perform literature search relevant to each guideline section and distribute to subcommittee and committee members
Solicit additional relevant information from subcommittee and committee members for each subsection
Formulate new recommendations and revise previous recommendations assigning Strength of Recommendation and Strength of Evidence
Form consensus of subcommittee for each section by conference call
Assign writing of additional or revised background by subcommittee
Full committee review of each section with revisions by subcommittee
Review of each completed section by Executive Council with revisions made by full committee and returned to Executive Council for final approval.
Disseminate document
Update document as changes are necessary

Dissemination and Continuity. The value of a practice guideline is significantly influenced by the scope of its dissemination. The first and second HFSA guidelines were available on the Internet, and thousands of copies were downloaded. The current document will be accessible on the Internet both for file transfer and as a hypertext source of detailed knowledge concerning HF.

An important final consideration is the continuity of the guideline development process. The intent is to create a “living document” that will be updated and amended as necessary to ensure continuing relevance. The rapid development of new knowledge in HF from basic and clinical research and the continuing evolution of pharmacologic and device therapy for this condition provides a strong mandate

for timely updates. The HFSA intends to undertake targeted reviews and updates in areas where new research has implications for practice. Section 17: The Genetic Evaluation of Cardiomyopathy is an example of this policy.

Summary

Practice guidelines have become a major part of the clinical landscape and seem likely to become more rather than less pervasive. Some may perceive guidelines as another mechanism for process management or as another instrument for cost control. But there is a more patient-centered rationale for their development, especially for a common, potentially debilitating, and often fatal syndrome such as HF. Despite advances in clinical trial methodology and the extensive use of studies to evaluate therapeutics and the care process, essential elements of the management process remain undefined for many clinical problems. HF is no exception. Traditionally, management guidelines were determined on an ad hoc basis by physicians and other health care providers in the field. The development and utilization of practice guidelines

has emerged as an alternative strategy. The methodology of guideline development needs improvement, but when these documents are properly conceived and formulated, their importance to patient care seems evident. This HFSA guideline on HF is designed as a “living document,” which will continue to serve as a resource for helping patients with HF.

References

1. Adams K, Baughman K, Dec W, Elkayam U, Forker A, Gheorghiade M, et al. Heart Failure Society of America (HFSA) practice guidelines. HFSA guidelines for management of patients with heart failure caused by left ventricular systolic dysfunction—pharmacological approaches. *J Card Fail* 1999;5:357–82.
2. Adams K, Lindenfeld J, Arnold J, Baker D, Barnard D, Baughman K, et al. HFSA 2006 Comprehensive Heart Failure Practice Guideline. *J Card Fail* 2006;12:e1–e122.
3. Hershberger RE, Lindenfeld J, Mestroni L, Seidman CE, Taylor MR, Towbin JA. Genetic evaluation of cardiomyopathy—a Heart Failure Society of America practice guideline. *J Card Fail* 2009;15:83–97.

Appendix A. Comparison of the 2006 and 2010 HFSA Guideline

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
Section 3: Prevention of Ventricular Remodeling, Cardiac Dysfunction, and Heart Failure			
3.1	A careful and thorough clinical assessment, with appropriate investigation for known or potential risk factors, is recommended in an effort to prevent development of LV remodeling, cardiac dysfunction, and HF. These risk factors include, but are not limited to, hypertension, hyperlipidemia, atherosclerosis, diabetes mellitus, valvular disease, obesity, physical inactivity, excessive alcohol intake, and smoking. (Strength of Evidence = A)	A careful and thorough clinical assessment, with appropriate investigation for known or potential risk factors, is recommended in an effort to prevent development of LV remodeling, cardiac dysfunction, and HF. These risk factors include, but are not limited to, hypertension, hyperlipidemia, atherosclerosis, diabetes mellitus, valvular disease, obesity, physical inactivity, excessive alcohol intake, dietary choices, and smoking. (Strength of Evidence = A)	Addition of dietary choices to list of risk factors
3.2	No changes		
3.3	No changes		
3.4	No changes		
Section 4: Evaluation of Patients for Ventricular Dysfunction and Heart Failure			
4.1	Evaluation with a routine history, physical examination, chest x-ray, and electrocardiogram (ECG) is recommended in patients with the medical conditions or test findings listed in Table 4.1. (Strength of Evidence = B)	Evaluation for clinical manifestations of HF with a routine history and physical examination is recommended in patients with the medical conditions or test findings listed in Table 4.1. (Strength of Evidence = B)	Modification of wording and deletion of chest x-ray and ECG (retained in Table 4.1)
4.2	Assessment of Cardiac Structure and Function. Echocardiography with Doppler is recommended to determine LV size and function in patients without signs or symptoms suggestive of HF who have the risk factors listed in Table 4.2. (Strength of Evidence = B)	Assessment of Cardiac Structure and Function. Echocardiography with Doppler is recommended to determine cardiac structure and function in asymptomatic patients with the disorders or findings listed in Table 4.2. (Strength of Evidence = B)	Modification of wording and terminology
4.3	Determination of plasma B-type natriuretic peptide (BNP) or N-terminal pro-BNP concentration is not recommended as a routine part of the evaluation for structural heart disease in patients at risk but without signs or symptoms of HF. (Strength of Evidence = B)	Routine determination of plasma BNP or NT-proBNP concentration as part of a screening evaluation for structural heart disease in asymptomatic patients is not recommended. (Strength of Evidence = B)	Modification of wording and terminology
4.4	Symptoms Consistent with HF. The symptoms listed in Table 4.3 suggest the diagnosis of HF. It is recommended that each of these symptoms be solicited and graded in all patients in whom the diagnosis of HF is being considered. (Strength of Evidence = B)	Symptoms Consistent with HF. The symptoms listed in Table 4.3 suggest the diagnosis of HF. It is recommended that each of these symptoms be elicited in all patients in whom the diagnosis of HF is being considered. (Strength of Evidence = B)	Modification of wording and addition of depression to Table 4.3
4.5	Physical Examination. It is recommended that patients suspected of having HF undergo careful physical examination with determination of vital signs and be carefully evaluated for signs and symptoms shown in Table 4.4. (Strength of Evidence = C)	Physical Examination. It is recommended that patients suspected of having HF undergo careful physical examination with determination of vital signs and careful evaluation for signs shown in Table 4.4. (Strength of Evidence = B)	Modification of wording and change in Strength of Evidence from C to B and addition of reduced cardiac output and arrhythmia to cardiac abnormalities in Table 4.4
4.6	It is recommended that BNP or NT-proBNP levels be assessed in all patients suspected of having HF when the diagnosis is not certain. (Strength of Evidence = B)	It is recommended that BNP or NT-proBNP levels be assessed in all patients suspected of having HF, especially when the diagnosis is not certain. (Strength of Evidence = A)	Modification of wording and change in Strength of Evidence from B to A

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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
4.7	The differential diagnoses in Table 4.5 should be considered as alternative explanations for signs and symptoms consistent with HF. (Strength of Evidence = C)	Differential Diagnosis. The differential diagnoses in Table 4.5 should be considered as alternative explanations for signs and symptoms consistent with HF. (Strength of Evidence = B)	Modification of wording and change in Strength of Evidence from C to B and addition of chronic kidney disease and thyroid abnormalities to Table 4.5
4.8	No changes		
4.9	Symptoms. In addition to symptoms characteristic of HF, the following symptoms should be considered in the diagnosis of HF: <ul style="list-style-type: none"> • Angina • Symptoms of possible cerebral hypoperfusion, including syncope, pre-syncope, or lightheadedness • Symptoms suggestive of embolic events • Symptoms suggestive of sleep-disordered breathing (Strength of Evidence = C)	Symptoms. In addition to symptoms characteristic of HF (dyspnea, fatigue, decreased exercise tolerance, fluid retention), evaluation of the following symptoms should be considered in the diagnosis of HF: <ul style="list-style-type: none"> • Angina • Symptoms suggestive of embolic events • Symptoms suggestive of sleep-disordered breathing • Symptoms suggestive of arrhythmias, including palpitations • Symptoms of possible cerebral hypoperfusion, including syncope, presyncope, or lightheadedness (Strength of Evidence = B)	Clarification of HF symptoms and addition of arrhythmia to list of symptoms and change in Strength of Evidence from C to B
4.10	No changes		
4.11	The degree of volume excess is a key consideration during treatment. It is recommended that it be routinely assessed by determining: <ul style="list-style-type: none"> • Presence of paroxysmal nocturnal dyspnea or orthopnea • Daily weights and vital signs with assessment for orthostatic changes • Presence and degree of rales, S3 gallop, jugular venous pressure elevation, positive hepatojugular reflux, edema, and ascites (Strength of Evidence = B)	Volume Status. The degree of volume excess is a key consideration during treatment. It is recommended that it be routinely assessed by determining: <ul style="list-style-type: none"> • Presence of paroxysmal nocturnal dyspnea or orthopnea • Presence of dyspnea on exertion • Daily weights and vital signs with assessment for orthostatic changes • Presence and degree of rales, S3 gallop, jugular venous pressure elevation, hepatic enlargement and tenderness, positive hepatojugular reflux, edema, and ascites (Strength of Evidence = B)	Addition of presence of dyspnea on exertion and hepatic enlargement/tenderness to list of assessments
4.12	It is recommended that the following laboratory tests be obtained routinely in patients being evaluated for HF: serum electrolytes, blood urea nitrogen, creatinine, glucose, calcium, magnesium, lipid profile (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides), complete blood count, serum albumin, liver function tests, urinalysis, and thyroid function. (Strength of Evidence = B)	Standard Laboratory Tests. It is recommended that the following laboratory tests be obtained routinely in patients being evaluated for HF: serum electrolytes, blood urea nitrogen, creatinine, glucose, calcium, magnesium, fasting lipid profile (low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, triglycerides), complete blood count, serum albumin, uric acid, liver function tests, urinalysis, and thyroid function. (Strength of Evidence = B)	Addition of uric acid to list of standard laboratory tests
4.13	It is recommended that all patients with HF have an ECG performed to: <ul style="list-style-type: none"> • Assess cardiac rhythm and conduction • Detect LV hypertrophy • Evaluate QRS duration, especially when ejection fraction (EF) <35% • Detect evidence of myocardial infarction or ischemia (Strength of Evidence = B)	Electrocardiogram (ECG). It is recommended that all patients with HF have an ECG performed to: <ul style="list-style-type: none"> • Assess cardiac rhythm and conduction (in some cases, using Holter monitoring or event monitors) • Assess electrical dyssynchrony (wide QRS or bundle branch block), especially when left ventricular ejection fraction (LVEF) <35% • Detect LV hypertrophy or other chamber enlargement • Detect evidence of MI or ischemia • Assess QTc interval, especially with drugs that prolong QT intervals (Strength of Evidence = B)	Addition of electrical dyssynchrony and QTc interval to list of ECG assessments

4.14	It is recommended that all patients with HF have a posteroanterior and lateral chest X-ray examination for determination of heart size, evidence of fluid overload, and detection of pulmonary and other diseases. (Strength of Evidence = B)	Chest X-Ray. It is recommended that all patients with HF have a postero-anterior and lateral chest X-ray examination for determination of heart size, evidence of fluid overload, detection of pulmonary and other diseases, and appropriate placement of implanted cardiac devices. (Strength of Evidence = B)	Addition of placement of implanted cardiac devices to list of chest x-rays assessments
4.15	Additional Laboratory Tests. It is recommended that patients with no apparent etiology of HF or no specific clinical features suggesting unusual etiologies undergo additional directed blood and laboratory studies to determine the cause of HF. (Strength of Evidence = C)	Additional Laboratory Tests. It is recommended that patients with no apparent etiology of HF or no specific clinical features suggesting unusual etiologies undergo additional directed blood and laboratory studies to determine the cause of HF. (Strength of Evidence = B)	Change in Strength of Evidence from C to B
4.16		Evaluation of myocardial ischemia is recommended in those who develop new-onset LV systolic dysfunction especially in the setting of suspected myocardial ischemia or worsening symptoms with pre-existing CAD. The choice of testing modality should depend on the clinical suspicion and underlying cardiac risk factors. Coronary angiography should be considered when pre-test probability of underlying ischemic cardiomyopathy is high and an invasive coronary intervention may be considered. (See Section 13 for specific clinical situations and Strength of Evidence)	New recommendation
4.17 (previous 4.16)	Exercise testing is not recommended as part of routine evaluation in patients with HF. Specific circumstances in which maximal exercise testing with measurement of expired gases should be considered include: <ul style="list-style-type: none"> • Assessing disparity between symptomatic limitation and objective indicators of disease severity • Distinguishing non-HF-related causes of functional limitation, specifically cardiac versus pulmonary • Considering candidacy for cardiac transplantation or mechanical intervention • Determining the prescription for cardiac rehabilitation • Addressing specific employment capabilities Exercise testing for inducible abnormality in myocardial perfusion or wall motion abnormality should be considered to screen for the presence of coronary artery disease with inducible ischemia. (Strength of Evidence = C)	Exercise testing for functional capacity is not recommended as part of routine evaluation in patients with HF. Specific circumstances in which maximal exercise testing with measurement of expired gases should be considered include: <ul style="list-style-type: none"> • Assessing disparity between symptomatic limitation and objective indicators of disease severity • Distinguishing non HF-related causes of functional limitation, specifically cardiac versus pulmonary • Considering candidacy for cardiac transplantation or mechanical circulatory support • Determining the prescription for cardiac rehabilitation • Addressing specific employment capabilities (Strength of Evidence = C)	Modification of wording and deletion of recommendation for exercise testing for inducible abnormality in myocardial perfusion or wall motion abnormality
4.18 (previous 4.17)	No changes		
4.19 (previous 4.18)	It is recommended that clinical evaluation at each followup visit include the assessments listed in Table 4.9. (Strength of Evidence = B) These assessments should include the same symptoms and signs assessed during the initial evaluation. (Strength of Evidence = C)	It is recommended that clinical evaluation at each follow-up visit include determination of the elements listed in Table 4.9. (Strength of Evidence = B). These assessments should include the same symptoms and signs assessed during the initial evaluation. (Strength of Evidence = B)	Change (in second part of recommendation) Strength of Evidence from C to B

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	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
4.20 (previous 4.19)	<p>Routine reevaluation of cardiac function by noninvasive or invasive methods is not recommended. Repeat measurements of ventricular volume and EF should be considered under limited circumstances:</p> <ul style="list-style-type: none"> • After at least 3 months of medical therapy when prophylactic ICD placement is being considered to confirm that EF criteria are still met. (Strength of Evidence = B) • In patients who show substantial clinical improvement (for example, in response to b-blocker treatment). Such change may denote improved prognosis, although it does not in itself mandate alteration or discontinuation of specific treatments. (Strength of Evidence = C) <p>Repeat determination of EF is usually unnecessary in patients with previously documented LV dilation and low EF who manifest worsening signs or symptoms of HF. Repeat measurement should be considered when it is likely to prompt a change in patient management, such as cardiac transplantation. (Strength of Evidence = C)</p>	<p>In the absence of deteriorating clinical presentation, repeat measurements of ventricular volume and LVEF should be considered in these limited circumstances:</p> <ul style="list-style-type: none"> • When a prophylactic implantable cardioverter defibrillator (ICD) or CRT device and defibrillator (CRT-D) placement is being considered in order to determine that LVEF criteria for device placement are still met after medical therapy (Strength of Evidence = B) • When patients show substantial clinical improvement (for example, in response to beta blocker treatment or following pregnancy in patients with peripartum cardiomyopathy). Such change may denote improved prognosis, although it does not in itself mandate alteration or discontinuation of specific treatments (see Section 7). (Strength of Evidence = C) • In alcohol and cardiotoxic substance abusers who have discontinued the abused substance. (Strength of Evidence = C) • In patients receiving cardiotoxic chemotherapy. (Strength of Evidence = B) <p>Repeat determination of LVEF is usually unnecessary in patients with previously documented LV dilatation and low LVEF who manifest worsening signs or symptoms of HF, unless the information is needed to justify a change in patient management (such as surgery or device implantation). (Strength of Evidence = C)</p>	<p>Modifications of recommendation throughout</p>
4.21 (previous 4.20)	<p>It is recommended that reevaluation of electrolytes and renal function occur at least every 6 months in clinically stable patients and more frequently after changes in therapy or with evidence of change in volume status. More frequent assessment of electrolytes and renal function is recommended in patients with severe HF, those receiving high doses of diuretics, and those who are clinically unstable. (Strength of Evidence = C) (See Section 7 for recommendations regarding patients on angiotensin receptor blockers.)</p>	<p>It is recommended that reevaluation of electrolytes and renal function occur at least every 6 months in clinically stable patients and more frequently following changes in therapy or with evidence of change in volume status. More frequent assessment of electrolytes and renal function is recommended in patients with severe HF, those receiving high doses of diuretics, those on aldosterone antagonists, and those who are clinically unstable. (Strength of Evidence = C) (See Section 7 for recommendations regarding patients on angiotensin receptor blockers.)</p>	<p>Addition of aldosterone antagonists to list of patients in whom more frequent assessment of electrolytes and renal function is recommended.</p>
Section 5: Management of Asymptomatic Patients with Reduced LVEF			
5.1	<p>It is recommended that all patients with ALVD exercise regularly according to a physician-directed prescription to avoid general deconditioning; to improve weight, blood pressure, and diabetes control; and to reduce cardiovascular risk. (Strength of Evidence = C)</p>	<p>It is recommended that all patients with ALVD exercise regularly according to a physician-directed prescription to avoid general deconditioning; to optimize weight, blood pressure, and diabetes control; and to reduce cardiovascular risk. (Strength of Evidence = C)</p>	<p>Minor wording modification</p>
5.2	No changes		
5.3	<p>It is recommended that alcohol consumption be discouraged in patients with ALVD. Abstinence is recommended if there is a current habit or history of excessive alcohol intake. (Strength of Evidence = C)</p>	<p>Alcohol abstinence is recommended if there is current or previous history of excessive alcohol intake. (Strength of Evidence = C)</p>	<p>Deleted phrase discouraging alcohol use in ALVD. Other minor wording modifications.</p>
5.4	<p>It is recommended that all patients with ALVD with hypertension have aggressive blood pressure control. (Strength of Evidence = B)</p>	<p>It is recommended that all patients with ALVD with hypertension achieve optimal blood pressure control. (Strength of Evidence = B)</p>	<p>Aggressive blood pressure control changed to optimal blood pressure control</p>
5.5	No changes		

5.6	ARBs are recommended for asymptomatic patients with reduced LVEF who are intolerant of ACE inhibitors because of cough or angioedema. (Strength of Evidence = C) Routine use of the combination of ACE inhibitors and ARBs for prevention of HF is not recommended in this population. (Strength of Evidence = C)	ARBs are recommended for asymptomatic patients with reduced LVEF who are intolerant of ACE inhibitors from cough or angioedema. (Strength of Evidence = C) Routine use of the combination of ACE inhibitors and ARBs for prevention of HF is not recommended in this population. (Strength of Evidence = C)	Minor wording modification
5.7	It is recommended that beta blocker therapy be administered to asymptomatic patients with reduced LVEF. (Post MI, Strength of Evidence = B; non-Post MI, Strength of Evidence = C)	Beta blocker therapy should be considered in asymptomatic patients with reduced LVEF. (post-MI, Strength of Evidence = B; non post-MI, Strength of Evidence = C)	Changed from “is recommended” to “should be considered”
Section 6: Nonpharmacologic Management and Health Care Maintenance in Patients with Chronic Heart Failure			
6.1	Dietary instruction regarding sodium intake is recommended in all patients with HF. Patients with HF and diabetes, dyslipidemia, or obesity should be given specific instructions regarding carbohydrate or caloric constraints. (Strength of Evidence = B)	Dietary instruction regarding sodium intake is recommended in all patients with HF. Patients with HF and diabetes, dyslipidemia, or severe obesity should be given specific dietary instructions. (Strength of Evidence = B)	Minor wording modification
6.2	No changes		
6.3	No changes		
6.4	It is recommended that specific attention be paid to nutritional management of patients with advanced HF and unintentional weight loss or muscle wasting (cardiac cachexia). Measurement of nitrogen balance, caloric intake, and prealbumin may be useful in determining appropriate nutritional supplementation. Caloric supplementation is recommended. Anabolic steroids are not recommended for such patients. (Strength of Evidence = C)	It is recommended that specific attention be paid to nutritional management of patients with advanced HF and unintentional weight loss or muscle wasting (cardiac cachexia). Measurement of nitrogen balance, caloric intake, and prealbumin may be useful in determining appropriate nutritional supplementation. Caloric supplementation is recommended. Anabolic steroids are not recommended for cachexic patients. (Strength of Evidence = C)	Minor wording modification
6.5	No changes		
6.6	Documentation of the type and dose of nutraceutical products used by patients with HF is recommended. (Strength of Evidence = C) Nutraceutical use is not recommended for relief of symptomatic HF or for the secondary prevention of cardiovascular events. Patients should be instructed to avoid using natural or synthetic products containing ephedra (ma huang), ephedrine, or its metabolites because of an increase risk of mortality and morbidity. Products should be avoided that may have significant drug interactions with digoxin, vasodilators, beta blockers, antiarrhythmic drugs, and anticoagulants. (Strength of Evidence = B)	Documentation of the type and dose of naturoceutical products used by patients with HF is recommended. (Strength of Evidence = C) Naturoceutical use is not recommended for relief of symptomatic HF or for the secondary prevention of cardiovascular events. Patients should be instructed to avoid using natural or synthetic products containing ephedra (ma huang), ephedrine, or its metabolites because of an increased risk of mortality and morbidity. Products should be avoided that may have significant drug interactions with digoxin, vasodilators, beta blockers, antiarrhythmic drugs, and anticoagulants. (Strength of Evidence = B)	Modification of terminology (nutraceutical to naturoceutical)
6.7	No changes		
6.8	No changes		
6.9	No changes		
6.10	It is recommended that screening for endogenous or prolonged reactive depression in patients with HF be conducted after diagnosis and at periodic intervals as clinically indicated. For pharmacologic treatment, selective serotonin receptor uptake inhibitors are preferred over tricyclic antidepressants, because the latter have the potential to cause ventricular arrhythmias, but the potential for drug interactions should be considered. (Strength of Evidence = B)	It is recommended that screening for endogenous or prolonged reactive depression in patients with HF be conducted following diagnosis and at periodic intervals as clinically indicated. For pharmacologic treatment, selective serotonin reuptake inhibitors are preferred over tricyclic antidepressants, because the latter have the potential to cause ventricular arrhythmias, but the potential for drug interactions should be considered. (Strength of Evidence = B)	Minor wording modification

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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
6.11	No changes		
6.12	No changes		
6.13	No changes		
6.14	No changes		
6.15	Endocarditis prophylaxis is not recommended based on the diagnosis of HF alone. Prophylaxis for dental and other procedures should be given according to standard clinical indications. (Strength of Evidence = C)	Endocarditis prophylaxis is not recommended based on the diagnosis of HF alone. Consistent with the AHA recommendation, 'prophylaxis should be given for only specific cardiac conditions, associated with the highest risk of adverse outcome from endocarditis.' These conditions include: 'prosthetic cardiac valves; previous infective endocarditis; congenital heart disease (CHD)' such as: 'unrepaired cyanotic CHD, including palliative shunts and conduits; completely repaired congenital heart defect with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first six months after the procedure; repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibit endothelialization); cardiac transplantation recipients who develop cardiac valvulopathy.' (Strength of Evidence = C)	Addition of criteria for endocarditis prophylaxis
6.16	No changes		
6.17	No changes		
6.18	No changes		
6.19		It is recommended that patients with HF undergo exercise testing to determine suitability for exercise training (patient does not develop significant ischemia or arrhythmias). (Strength of Evidence = B) If deemed safe, exercise training should be considered for patients with HF in order to facilitate understanding of exercise expectations (heart rate ranges and appropriate levels of exercise training), to increase exercise duration and intensity in a supervised setting, and to promote adherence to a general exercise goal of 30 minutes of moderate activity/exercise, 5 days per week with warm up and cool down exercises. (Strength of Evidence = B)	New recommendation
Section 7: Heart Failure in Patients with Reduced Ejection Fraction			
7.1	No changes		
7.2	It is recommended that other therapy be substituted for ACE inhibitors in the following circumstances: <ul style="list-style-type: none"> • In patients who cannot tolerate ACE inhibitors because of cough, ARBs are recommended. (Strength of Evidence = A) • The combination of hydralazine and an oral nitrate may be considered in such patients not tolerating ARB therapy. (Strength of Evidence = C) • Patients intolerant to ACE inhibitors because of hyperkalemia or renal insufficiency are likely to experience the same side effects with ARBs. In these cases, the combination of hydralazine and an oral nitrate should be considered. (Strength of Evidence = C) 	It is recommended that other therapy be substituted for ACE inhibitors in the following circumstances: <ul style="list-style-type: none"> • In patients who cannot tolerate ACE inhibitors due to cough, ARBs are recommended. (Strength of Evidence = A) • The combination of hydralazine and an oral nitrate may be considered in such patients not tolerating ARB therapy. (Strength of Evidence = C) • Patients intolerant to ACE inhibitors from hyperkalemia or renal insufficiency are likely to experience the same side effects with ARBs. In these cases, the combination of hydralazine and an oral nitrate should be considered. (Strength of Evidence = C) 	Minor wording modification

7.3 (previous 7.10)	No changes		
7.4 (previous 7.12)	ARBs should be considered in patients experiencing angioedema while on ACE inhibitors based on their underlying risk and with recognition that angioedema has been reported infrequently with these agents. (Strength of Evidence = B) The combination of hydralazine and oral nitrates may be considered in this setting for patients who do not tolerate ARB therapy. (Strength of Evidence = C)	ARBs should be considered in patients experiencing angioedema while on ACE inhibitors based on their underlying risk and with recognition that angioedema has been reported infrequently with ARBs. (Strength of Evidence = B) The combination of hydralazine and oral nitrates may be considered in this setting for patients who do not tolerate ARB therapy. (Strength of Evidence = C)	Minor wording modifications
7.5 (previous 7.11)	Individual ARBs may be considered as initial therapy rather than ACE inhibitors for patients with the following conditions: <ul style="list-style-type: none"> • HF post MI (Strength of Evidence = A) • Chronic HF and systolic dysfunction (Strength of Evidence = B) 	Individual ARBs may be considered as initial therapy rather than ACE inhibitors for patients with the following conditions: <ul style="list-style-type: none"> • HF Post-MI (Strength of Evidence = A) • Chronic HF and reduced LVEF (Strength of Evidence = B) 	Terminology modification (changed “systolic dysfunction” to “reduced LVEF”)
7.6 (previous 7.3)	No changes		
7.7 (previous 7.4)	No changes		
7.8 (previous 7.5)	Beta blocker therapy is recommended for patients with a recent decompensation of HF after optimization of volume status and successful discontinuation of intravenous diuretics and vasoactive agents, including inotropic support. Whenever possible, beta blocker therapy should be initiated in the hospital setting at a low dose before discharge in stable patients. (Strength of Evidence = B)	Beta blocker therapy is recommended for patients with a recent decompensation of HF after optimization of volume status and successful discontinuation of intravenous diuretics and vasoactive agents, including inotropic support. Whenever possible, beta blocker therapy should be initiated in the hospital setting at a low dose prior to discharge in stable patients. (Strength of Evidence = B)	Minor wording modifications
7.9 (previous 7.6)	Beta blocker therapy is recommended in the great majority of patients with LV systolic dysfunction, even if there is concomitant diabetes, chronic obstructive lung disease, or peripheral vascular disease. Beta blocker therapy should be used with caution in patients with diabetes with recurrent hypoglycemia, asthma, or resting limb ischemia. Considerable caution should be used if beta blockers are initiated in patients with marked bradycardia (<55 beats/min) or marked hypotension (systolic blood pressure <80 mm Hg). Beta blockers are not recommended in patients with asthma with active bronchospasm. (Strength of Evidence = C)	Beta blocker therapy is recommended in the great majority of patients with HF and reduced LVEF, even if there is concomitant diabetes, chronic obstructive lung disease, or peripheral vascular disease. Beta blocker therapy should be used with caution in patients with diabetes with recurrent hypoglycemia, with asthma, or with resting limb ischemia. Considerable caution should be used if beta blockers are initiated in patients with marked bradycardia (<55 beats/min) or marked hypotension (systolic blood pressure <80 mm Hg). Beta blockers are not recommended in patients with asthma with active bronchospasm. (Strength of Evidence = C)	Modification of terminology (“LV systolic dysfunction” changed to “reduced LVEF”)
7.10 (previous 7.7)	It is recommended that b-blockade be initiated at low doses and uptitrated gradually, typically no sooner than at 2-week intervals. Doses found to be effective in HF trials generally are achieved in 8 to 12 weeks. Patients developing worsening HF symptoms or other side effects during titration may require a dosage adjustment of diuretic or concomitant vasoactive medications. If side effects resolve with medication adjustment, patients can subsequently be titrated to target or maximally tolerated doses. Some patients may require a more prolonged interval during uptitration, a temporary reduction in b-blocker dose, or, in rare cases, withdrawal of therapy. (Strength of Evidence = B)	It is recommended that beta blockade be initiated at low doses and uptitrated gradually, typically at 2-week intervals in patients with reduced LVEF, and after 3-10 day intervals in patients with reduced LVEF following newly diagnosed MI. (Strength of Evidence = B)	Deleted information related to beta blocker management

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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
7.11 (previous 7.8)	It is recommended that beta blocker therapy be continued in most patients experiencing a symptomatic exacerbation of HF during chronic maintenance treatment. (Strength of Evidence = C) A temporary reduction of dose in this setting may be considered. Abrupt discontinuation in patients with symptomatic exacerbation should be avoided. (Strength of Evidence = C) If discontinued or reduced, beta blockers should be reinstated or the dose should be gradually increased before the patient is discharged.	It is recommended that beta blocker therapy be continued in most patients experiencing a symptomatic exacerbation of HF during chronic maintenance treatment, unless they develop cardiogenic shock, refractory volume overload, or symptomatic bradycardia (Strength of Evidence = C) A temporary reduction of dose (generally by one-half) in this setting may be considered. Abrupt discontinuation in patients with symptomatic exacerbation should be avoided, unless the situation is life-threatening. (Strength of Evidence = C) If discontinued or reduced, beta blockers should be reinstated before the patient is discharged. In general, doses should be uptitrated to the previous well-tolerated dose as soon as safely possible (Strength of Evidence =B)	Addition of criteria for beta blocker discontinuation and reinstatement
7.12 (previous 7.13)	The routine administration of an ARB is not recommended in addition to ACE inhibitor and beta blocker therapy in patients with recent acute MI and LV dysfunction. (Strength of Evidence = A)	The routine administration of an ARB is not recommended in addition to ACE inhibitor and beta blocker therapy in patients with a recent acute MI and reduced LVEF. (Strength of Evidence = A)	Modification of terminology (“LV dysfunction” changed to “reduced LVEF”)
7.13		The addition of an ARB should be considered in patients with HF due to reduced LVEF who have persistent symptoms or progressive worsening despite optimized therapy with an ACE inhibitor and beta blocker. (Strength of Evidence = A)	New recommendation
7.14	Administration of an aldosterone antagonist is recommended for patients with NYHA class IV or class III, previously class IV, HF from LV systolic dysfunction (LVEF \leq 35%) while receiving standard therapy, including diuretics. (Strength of Evidence = A)	Administration of an aldosterone antagonist is recommended for patients with NYHA class IV (or class III, previously class IV) HF from reduced LVEF (<35%) while receiving standard therapy, including diuretics. (Strength of Evidence = A)	Modification of terminology (“LV systolic dysfunction” changed to “reduced LVEF”)
7.15	Administration of an aldosterone antagonist should be considered in patients after an acute MI, with clinical HF signs and symptoms and an LVEF <40%. Patients should be on standard therapy, including an ACE inhibitor (or ARB) and a b-blocker. (Strength of Evidence = A)	Administration of an aldosterone antagonist should be considered in patients following an acute MI, with clinical HF signs and symptoms or history of diabetes mellitus, and an LVEF <40%. Patients should be on standard therapy, including an ACE inhibitor (or ARB) and a beta blocker. (Strength of Evidence = A)	Addition of history of diabetes mellitus to criteria for therapy
7.16	No changes		
7.17	No changes		
7.18	No changes		
7.19	A combination of hydralazine and isosorbide dinitrate is recommended as part of standard therapy in addition to beta blockers and ACE inhibitors for African Americans with LV systolic dysfunction. • NYHA III or IV HF (Strength of Evidence = A) • NYHA II HF (Strength of Evidence = B) (See Section 15 Special Populations)	A combination of hydralazine and isosorbide dinitrate is recommended as part of standard therapy in addition to beta blockers and ACE inhibitors for African Americans with HF and reduced LVEF. • NYHA III or IV HF (Strength of Evidence = A) • NYHA II HF (Strength of Evidence = B) (See Section 15: Special Populations)	Modification of terminology (“LV systolic dysfunction” changed to “reduced LVEF”)
7.20	A combination of hydralazine and isosorbide dinitrate may be considered in non-African American patients with LV systolic dysfunction who remain symptomatic despite optimized standard therapy. (Strength of Evidence = C)	A combination of hydralazine and isosorbide dinitrate may be considered in non-African-American patients with HF and reduced LVEF who remain symptomatic despite optimized standard therapy. (Strength of Evidence = C)	Modification of terminology (“LV systolic dysfunction” changed to “reduced LVEF”)

7.21	<p>Additional pharmacologic therapy should be considered in patients with HF due to systolic dysfunction who have persistent symptoms or progressive worsening despite optimized therapy with an ACE inhibitor and beta blocker. The choice of specific agent will be influenced by clinical considerations, including renal function status, chronic serum potassium concentration, blood pressure, and volume status. The triple combination of an ACE inhibitor, an ARB, and an aldosterone antagonist is not recommended because of the high risk of hyperkalemia. (Strength of Evidence = C)</p> <ul style="list-style-type: none"> ● Addition of an ARB. (Strength of Evidence = A) ● Addition of an aldosterone antagonist: <ul style="list-style-type: none"> ○ For severe HF (Strength of Evidence = A) ○ For moderate HF (Strength of Evidence = C) ● Addition of the combination of hydralazine/isosorbide dinitrate: <ul style="list-style-type: none"> ○ For African Americans (Strength of Evidence = A) ○ For others (Strength of Evidence = C) 	<p>Additional pharmacologic therapy should be considered in patients with HF and reduced LVEF who have persistent symptoms or progressive worsening despite optimized therapy with an ACE inhibitor and beta blocker. The choice of specific agent will be influenced by clinical considerations, including renal function status, chronic serum potassium concentration, blood pressure, and volume status. The triple combination of an ACE inhibitor, an ARB, and an aldosterone antagonist is not recommended because of the high risk of hyperkalemia. (Strength of Evidence = C)</p> <ul style="list-style-type: none"> ● Addition of an ARB. (Strength of Evidence = A) ● Addition of an aldosterone antagonist: <ul style="list-style-type: none"> ○ for severe HF (Strength of Evidence = A) ○ for moderate HF (Strength of Evidence = C) ○ for post-MI HF (Strength of Evidence = A) ● Addition of the combination of hydralazine/isosorbide dinitrate: <ul style="list-style-type: none"> ○ for African Americans (Strength of Evidence = A) ○ for others (Strength of Evidence = C) 	<p>Modification of terminology (“systolic dysfunction” changed to “reduced LVEF”); addition of post-MI HF under aldosterone antagonists</p>
7.22	<p>Additional pharmacological therapy should be considered in patients with HF due to systolic dysfunction who are unable to tolerate a beta blocker and have persistent symptoms or progressive worsening despite optimized therapy with an ACE inhibitor. The choice of specific agent will be influenced by clinical considerations, including renal function status, chronic serum potassium concentration, blood pressure and volume status. The triple combination of an ACE inhibitor, an ARB, and an aldosterone antagonist is not recommended due to the high risk of hyperkalemia. (Strength of Evidence = C)</p> <ul style="list-style-type: none"> ● Addition of an ARB. (Strength of Evidence = C) ● Addition of an aldosterone antagonist: <ul style="list-style-type: none"> ○ for severe HF (Strength of Evidence = C) ○ for moderate HF (Strength of Evidence = C) ● Addition of the combination of hydralazine/isosorbide dinitrate: <ul style="list-style-type: none"> ○ For African-Americans (Strength of Evidence = C) ○ for others (Strength of Evidence = C) 	<p>Additional pharmacological therapy should be considered in patients with HF and reduced LVEF <i>who are unable to tolerate a beta blocker</i> and have persistent symptoms or progressive worsening despite optimized therapy with an ACE inhibitor. The choice of specific agent will be influenced by clinical considerations, including renal function status, chronic serum potassium concentration, blood pressure and volume status. The triple combination of an ACE inhibitor, an ARB, and an aldosterone antagonist is not recommended due to the high risk of hyperkalemia. (Strength of Evidence = C)</p> <ul style="list-style-type: none"> ● Addition of an ARB. (Strength of Evidence = C) ● Addition of an aldosterone antagonist: <ul style="list-style-type: none"> ○ for severe HF (Strength of Evidence = C) ○ for moderate HF (Strength of Evidence = C) ● Addition of the combination of hydralazine/isosorbide dinitrate: <ul style="list-style-type: none"> ○ for African Americans (Strength of Evidence = C) ○ for others (Strength of Evidence = C) 	<p>Modification of terminology (“systolic dysfunction” changed to “reduced LVEF”)</p>
7.23	No changes		
7.24	<p>The initial dose of diuretic may be increased as necessary to relieve congestion. Restoration of normal volume status may require multiple adjustments over many days and occasionally weeks in patients with severe fluid overload evidenced by massive edema or ascites. After a diuretic effect is achieved with short acting loop diuretics, increasing administration frequency to twice or even 3 times per day will provide more diuresis with less physiologic perturbation than larger single doses. (Strength of Evidence = B)</p> <p>Oral torsemide may be considered in patients in whom poor absorption of oral medication or erratic diuretic effect may be present, particularly those with right-sided HF and refractory fluid retention despite high doses of other loop diuretics. (Strength of Evidence = C)</p> <p>Intravenous administration of diuretics may be necessary to relieve congestion. (Strength of Evidence = A)</p> <p>Diuretic refractoriness may represent patient noncompliance, a direct effect of diuretic use on the kidney, or progression of underlying cardiac dysfunction.</p>	<p>The initial dose of diuretic may be increased as necessary to relieve congestion. Restoration of normal volume status may require multiple adjustments over many days and occasionally weeks in patients with severe fluid overload evidenced by massive edema or ascites. After a diuretic effect is achieved with short-acting loop diuretics, increasing administration frequency to twice or even 3 times per day will provide more diuresis with less physiologic perturbation than larger single doses. (Strength of Evidence = B)</p> <p>Oral torsemide may be considered in patients in whom poor absorption of oral medication or erratic diuretic effect may be present, particularly those with right-sided HF and refractory fluid retention despite high doses of other loop diuretics. (Strength of Evidence = C)</p> <p>Intravenous administration of diuretics may be necessary to relieve congestion. (Strength of Evidence = A)</p> <p>Diuretic refractoriness may represent patient nonadherence, a direct effect of diuretic use on the kidney, or progression of underlying cardiac dysfunction.</p>	<p>Modification of terminology (“noncompliance” changed to “nonadherence”)</p>

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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
7.25	No changes		
7.26	Careful observation for the development of side effects, including electrolyte abnormalities, symptomatic hypotension, and renal dysfunction, is recommended in patients treated with diuretics, especially when used at high doses and in combination. Patients should undergo routine laboratory studies and clinical examination as dictated by their clinical response. (Strength of Evidence = B)	Careful observation for the development of side effects, including electrolyte abnormalities, symptomatic hypotension, renal dysfunction, or worsening renal function, is recommended in patients treated with diuretics, especially when used at high doses and in combination. Patients should undergo routine laboratory studies and clinical examination as dictated by their clinical response. (Strength of Evidence = B)	Addition of worsening renal function to list of potential side effects
7.27	No changes		
7.28	No changes		
7.29	Digoxin should be considered for patients with LV systolic dysfunction (LVEF \leq 40%) who have signs or symptoms of HF while receiving standard therapy, including ACE inhibitors and beta blockers: NYHA class II-III (Strength of Evidence = A) NYHA class IV (Strength of Evidence = B)	Digoxin may be considered to improve symptoms in patients with reduced LVEF (LVEF \leq 40%) who have signs or symptoms of HF while receiving standard therapy, including ACE inhibitors and beta blockers: <ul style="list-style-type: none"> • NYHA class II-III (Strength of Evidence = B) • NYHA class IV (Strength of Evidence = C) 	Modification from “should be considered” to “may be considered”, and change in Strength of Evidence
7.30	It is recommended that the dose of digoxin, which should be based on lean body mass, renal function and concomitant medications, should be 0.125 mg daily in the majority of patients and the serum digoxin level should be $<$ 1.0 ng/mL. (Strength of Evidence = C)	It is recommended that the dose of digoxin, which should be based on lean body mass, renal function, and concomitant medications, should be 0.125 mg daily in the majority of patients and the serum digoxin level should be $<$ 1.0 ng/mL, generally 0.7-0.9 ng/mL. (Strength of Evidence = B)	Addition of a lower serum concentration range (0.7-0.9 ng/ml), and change in strength of evidence from C to B
7.31	Adequate control of the ventricular response to atrial fibrillation in patients with HF is recommended. (Level of Evidence = B)	Digoxin should be considered for achieving adequate control of the ventricular response to atrial fibrillation in patients with HF. (Strength of Evidence = B)	Modification from “is recommended” to “should be considered”
7.32	No changes		
7.33	Treatment with warfarin (goal INR 2.0–3.0) is recommended for all patients with HF and chronic or documented paroxysmal atrial fibrillation (Strength of Evidence = A) or a history of systemic or pulmonary emboli, including stroke or transient ischemic attack, (Strength of Evidence = C) unless contraindicated.	Treatment with warfarin (goal international normalized ratio [INR] 2.0-3.0) is recommended for all patients with HF and chronic or documented paroxysmal, persistent, or long-standing atrial fibrillation (Strength of Evidence = A) or a history of systemic or pulmonary emboli, including stroke or transient ischemic attack (Strength of Evidence = C), unless contraindicated.	Addition of persistent or long-standing atrial fibrillation
7.34	No changes		
Previous 7.35			Deleted from current guideline
7.35 (previous 7.36)	Long-term treatment with an antithrombotic agent is recommended for patients with HF from ischemic cardiomyopathy, whether or not they are receiving ACE inhibitors. (Strength of Evidence = B) Aspirin is recommended in most patients for whom anticoagulation is not specifically indicated because of its proven efficacy in non-HF patients with ischemic heart disease, its convenience, and lower cost. Lower doses of aspirin (75 or 81 mg) may be preferable because data from 2 trials suggest more frequent worsening of HF at higher doses. (Strength of Evidence = C) Warfarin (goal INR 2.0–3.5) and clopidogrel (75 mg) have also prevented vascular events in post MI patients and may be considered as alternatives to aspirin. (Strength of Evidence = B)	Long-term treatment with an antiplatelet agent, generally aspirin in doses of 75 to 81 mg, is recommended for patients with HF due to ischemic cardiomyopathy, whether or not they are receiving ACE inhibitors. (Strength of Evidence = B) Warfarin (goal INR 2.0-3.0) and clopidogrel (75 mg) also have prevented vascular events in post-MI patients and may be considered as alternatives to aspirin. (Strength of Evidence = B)	Modification of terminology from “antithrombotic” to “antiplatelet”; addition of recommended doses for aspirin. INR range changed to 2.0-3.0

7.36 (previous 7.37)	Routine use of aspirin is not recommended in patients with HF not from ischemic cardiomyopathy and without other evidence of atherosclerotic vascular disease. (Strength of Evidence = C)	Routine use of aspirin is not recommended in patients with HF without atherosclerotic vascular disease. (Strength of Evidence = C)	Modification of terminology
Previous 7.38			Deleted from current guideline; addressed in recommendation 7.35
7.37 (previous 7.39)	No changes		
7.38 (previous 7.40)	In patients with HF and an implantable cardioverter defibrillator (ICD), amiodarone may be considered to reduce the frequency of repetitive discharges. (Strength of Evidence = C)	In patients with HF and an ICD, amiodarone may be considered to reduce the frequency of recurrent symptomatic arrhythmias causing ICD shocks. (Strength of Evidence = C)	Modification of wording
7.39 (previous 7.41)	It is recommended that patients taking amiodarone therapy and digoxin or warfarin generally have their maintenance doses of many commonly used agents, such as digoxin, warfarin, and statins, reduced when amiodarone is initiated and then carefully monitored for the possibility of adverse drug interactions. Adjustment in doses of these drugs and laboratory assessment of drug activity or serum concentration after initiation of amiodarone is recommended. (Strength of Evidence = A)	It is recommended that when amiodarone therapy is initiated, the potential for interactions with other drugs be reviewed. The maintenance doses of digoxin, warfarin, and some statins should be reduced when amiodarone is initiated and then carefully monitored. Adjustment in doses of these drugs and laboratory assessment of drug activity or serum concentration after initiation of amiodarone is recommended. (Strength of Evidence = A)	Modification of wording
7.40		Routine use of amiodarone therapy for asymptomatic arrhythmias that are not felt to contribute to HF or ventricular dysfunction is not recommended. (Strength of Evidence = B)	New recommendation
7.41		n-3 polyunsaturated fatty acids (PUFA) may be considered to reduce mortality in HF patients with NYHA class II-IV symptoms and reduced LVEF. (Strength of Evidence = B)	New recommendation

Section 8: Disease Management, Advance Directives, and End-of-Life Care in Heart Failure

8.1	It is recommended that patients with HF and their family members or caregivers receive individualized education and counseling that emphasizes self-care. This education and counseling should be delivered by providers using a team approach in which nurses with expertise in HF management provide the majority of education and counseling, supplemented by physician input and, when available and needed, input from dietitians, pharmacists, and other health care providers. All HF patients benefit from education and counseling, but patients in NYHA functional class III or IV need the most intensive education, whereas patients in NYHA I or II need less intensive education. (Strength of Evidence = B) Teaching is not sufficient without skill building and specification of critical target behaviors. Essential elements of patient education to promote self-care with associated skills are shown in Table 8.1. (Strength of Evidence = B)	It is recommended that patients with HF and their family members or caregivers receive individualized education and counseling that emphasizes self-care. This education and counseling should be delivered by providers using a team approach in which nurses with expertise in HF management provide the majority of education and counseling, supplemented by physician input and, when available and needed, input from dietitians, pharmacists, and other health care providers. (Strength of Evidence = B) Teaching is not sufficient without skill building and specification of critical target behaviors. It is recommended that essential elements of patient education (with associated skills) are utilized to promote self-care as shown in Table 8.1. (Strength of Evidence = B)	Deletion of NYHA specific portion of the recommendation; modification of wording
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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
8.2	It is recommended that patients' literacy, cognitive status, psychologic state, culture, and access to social and financial resources be taken into account for optimal education and counseling. Because cognitive impairment and depression are common in HF and can seriously interfere with learning, patients should be screened for these. Appropriate interventions, such as supportive counseling and pharmacotherapy, are recommended for those patients found to be depressed. Patients found to be cognitively impaired need additional support to manage their HF. (Strength of Evidence = C)	It is recommended that patients' literacy, cognitive status, psychological state, culture, and access to social and financial resources be taken into account for optimal education and counseling. Because cognitive impairment and depression are common in HF and can seriously interfere with learning, patients should be screened for these. Patients found to be cognitively impaired need additional support to manage their HF. (Strength of Evidence = B)	Deletion of description of interventions; modification of Strength of Evidence from C to B
8.3	No changes		
8.4	It is recommended that the frequency and intensity of patient education and counseling vary according to the stage of illness. Patients in advanced HF or with persistent difficulty adhering to the recommended regimen require the most education and counseling. Patients should be offered a variety of options for learning about HF according to their individual preferences: videotape, one-on-one or group discussion, reading materials, translators, telephone calls, mailed information, internet, visits. Repeated exposure to material is essential because a single session is never sufficient. (Strength of Evidence = B)	It is recommended that the frequency and intensity of patient education and counseling vary according to the stage of the illness. Patients in advanced HF or persistent difficulty adhering to the recommended regimen require the most education and counseling. Patients should be offered a variety of options for learning about HF according to their individual preferences: videotape, one-on-one or group discussion, reading materials, translators, telephone calls, mailed information, internet, visits. Repeated exposure to material is recommended because a single session is never sufficient. (Strength of Evidence = B)	Modification of wording
8.5	No changes		
8.6	No changes		
8.7	Patients recently hospitalized for HF and other patients at high risk should be considered for referral to a comprehensive HF disease management program that delivers individualized care. High-risk patients include those with renal insufficiency, low output state, diabetes, chronic obstructive pulmonary disease, persistent NYHA class III or IV symptoms, frequent hospitalization for any cause, multiple active comorbidities, or a history of depression, cognitive impairment, or persistent nonadherence to therapeutic regimens. (Strength of Evidence = A)	Patients recently hospitalized for HF and other patients at high risk for HF decompensation should be considered for comprehensive HF disease management. High-risk patients include those with renal insufficiency, low output state, diabetes, chronic obstructive pulmonary disease, persistent NYHA class III or IV symptoms, frequent hospitalization for any cause, multiple active comorbidities, or a history of depression, cognitive impairment, inadequate social support, poor health literacy, or persistent nonadherence to therapeutic regimens. (Strength of Evidence = A)	Addition of poor health literacy
8.8	No changes		
8.9	No changes		
8.10	No changes		
8.11	Patient and family or caregiver discussions about quality of life and prognosis are recommended as part of the disease management of HF. (Strength of Evidence = C)	It is recommended that patient and family or caregiver discussions about quality of life and prognosis be included in the disease management of HF. (Strength of Evidence = C)	Modification of wording

8.12	<p>It is recommended that the patient's status be optimized medically and psychologically before discussing the possibility that end-of-life care is indicated. The decision to declare a patient as an appropriate candidate for end-of-life care should be made by physicians experienced in the care of patients with HF. End-of-life management should be coordinated with the patient's primary care physician. As often as possible, discussions regarding end-of-life care should be initiated while the patient is still capable of participating in decision making. (Strength of Evidence = C)</p>	<p>It is recommended that</p> <ul style="list-style-type: none"> • Seriously ill patients with HF and their families be educated to understand that patients with HF are at high risk of death, even while aggressive efforts are made to prolong life. • Patients with HF be made aware that HF is potentially life-limiting, but that pharmacologic and device therapies and self-management can prolong life. In most cases, chronic HF pharmacologic and device therapies should be optimized as indicated before identifying that patients are near end-of-life. • Identification of end-of-life in a patient should be made in collaboration with clinicians experienced in the care of patients with HF when possible. • End-of-life management should be coordinated with the patient's primary care physician. • As often as possible, discussions regarding end-of-life care should be initiated while the patient is still capable of participating in decision-making. (Strength of Evidence = C) 	<p>Addition of criteria for end of life care</p>
8.13	<p>End-of-life care should be considered in patients who have advanced, persistent HF with symptoms at rest despite repeated attempts to optimize pharmacologic and nonpharmacologic therapy, as evidenced by one or more of the following:</p> <ul style="list-style-type: none"> • Frequent hospitalizations (3 or more per year) • Chronic poor quality of life with inability to accomplish activities of daily living • Need for intermittent or continuous intravenous support • Consideration of assist devices as destination therapy (Strength of Evidence = C) 	<p>End-of-life care should be considered in patients who have advanced, persistent HF with symptoms at rest despite repeated attempts to optimize pharmacologic, cardiac device, and other therapies, as evidenced by 1 or more of the following:</p> <ul style="list-style-type: none"> • HF hospitalization (Strength of Evidence = B) • Chronic poor quality of life with minimal or no ability to accomplish activities of daily living (Strength of Evidence = C) • Need for continuous intravenous inotropic therapy support (Strength of Evidence = B) 	<p>Addition of cardiac device to list of optimization therapies; modification of strength of evidence</p>
8.14	<p>It is recommended that end-of-life care strategies be individualized, include effective symptom management, and avoid unnecessary testing and interventions. (Strength of Evidence = C)</p>	<p>It is recommended that end-of-life care strategies be individualized and include core HF pharmacologic therapies, effective symptom management and comfort measures, while avoiding unnecessary testing. New life-prolonging interventions should be discussed with patients and care-givers with careful discussion of whether they are likely to improve symptoms. (Strength of Evidence = C)</p>	<p>Addition of information regarding end-of-life care strategies</p>
8.15	<p>It is recommended that, as part of end-of life-care, patients and their families/caregivers be given specific directions concerning their response to clinical events if they decide against resuscitation. Inactivation of an implantable defibrillation device should be discussed. (Strength of Evidence = C)</p>	<p>It is recommended that a specific discussion about resuscitation be held in the context of planning for overall care and for emergencies with all patients with HF. The possibility of SCD for patients with HF should be acknowledged. Specific plans to reduce SCD (for example with an ICD) or to allow natural death should be based on the individual patient's risks and preferences for an attempt at resuscitation with specific discussion of risks and benefits of inactivation the ICD. Preferences for attempts at resuscitation and plans for approach to care should be readdressed at turning points in the patient's course or if potentially life-prolonging interventions are considered. (Strength of Evidence = C)</p>	<p>Addition of information regarding resuscitation</p>
8.16		<p>It is recommended that, as part of end-of-life care, patients and their families/caregivers have a plan to manage a sudden decompensation, death, or progressive decline. Inactivation of an implantable defibrillation device should be discussed in the context of allowing natural death at end of life. A process for deactivating defibrillators should be clarified in all settings in which patients with HF receive care. (Strength of Evidence = C)</p>	<p>New recommendation</p>

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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
8.17	Patients with HF undergoing end-of-life care may be considered for hospice services that can be delivered in the home, a hospital setting, or a special hospice unit. (Strength of Evidence = C)	Patients with HF receiving end-of-life care should be considered for enrollment in hospice that can be delivered in the home, a nursing home, or a special hospice unit. (Strength of Evidence = C)	Modification from “may be considered” to “should be considered”
Previous 8.16 and 8.18			Deleted recommendations; portions of these recommendations have been incorporated into recommendations 8.15 and 8.16
Section 9: Electrophysiology Testing and the Use of Devices in Heart Failure			
9.1	It is recommended that the decision to undertake electrophysiologic intervention be made in light of functional status and prognosis based on severity of underlying HF and comorbid conditions. If LV dysfunction is a reason for recommending electrophysiologic intervention, LV function should be re-assessed, ideally after 3–6 months of optimal medical therapy. (Strength of Evidence = C)	It is recommended that the decision to undertake electrophysiologic intervention, including ICD implantation, be made in light of functional status and prognosis based on severity of underlying HF and comorbid conditions. If an ICD is considered due to LV dysfunction which is of recent onset, LV function should be reassessed, ideally after 3-6 months of optimal medical therapy. (Strength of Evidence = C)	Modification/clarification of wording
9.2	Immediate evaluation is recommended in patients with HF who present with syncope. In the absence of a clear identifiable noncardiac cause, patients should be referred for electrophysiologic evaluation. (Strength of Evidence = C)	Immediate evaluation is recommended in patients with HF who present with syncope. In the absence of a clear identifiable noncardiac cause, consultation with an EP specialist should be obtained. (Strength of Evidence = C)	Modification/clarification of wording
9.3	No changes		
9.4	In patients with or without concomitant coronary artery disease (including a prior MI > 1 month ago): a) Prophylactic ICD placement should be considered (LVEF ≤30%) and may be considered (LVEF 31–35%) for those with mild to moderate HF symptoms (NYHA II-III). (Strength of Evidence = A) See Recommendation 9.1 for additional criteria. b) Concomitant ICD placement should be considered in patients undergoing implantation of a biventricular pacing device according to the criteria in Recommendations 9.7–9.8. (Strength of Evidence = B) See Recommendation 9.1 for additional criteria.	a. Prophylactic ICD placement should be considered in patients with an LVEF ≤35% and mild to moderate HF symptoms: • Ischemic etiology (Strength of Evidence = A) • Non-ischemic etiology (Strength of Evidence = B) See Recommendation 9.1 for additional criteria. b. In patients who are undergoing implantation of a biventricular pacing device according to the criteria in recommendations 9.7-9.8, use of a device that provides defibrillation should be considered. (Strength of Evidence = B) See Recommendation 9.1 for additional criteria.	Revision of LVEF criteria and strength of evidence based on etiology
9.5	ICD placement is not recommended in chronic, severe refractory HF when there is no reasonable expectation for improvement. (Strength of Evidence = C)	ICD placement is not recommended in chronic, severe refractory HF when there is no reasonable expectation for improvement or in patients with a life expectancy of less than 1 year. (Strength of Evidence = C)	Addition of life expectancy criterion to recommendation
9.6	ICD implantation is recommended for survivors of cardiac arrest from ventricular fibrillation or hemodynamically unstable sustained ventricular tachycardia without evidence of acute MI or if the event occurs more than 48 hours after the onset of infarction in the absence of a recurrent ischemic event. (Strength of Evidence = A)	ICD implantation is recommended for survivors of cardiac arrest from ventricular fibrillation or hemodynamically unstable sustained VT that is not due to a transient, potentially reversible cause, such as acute MI. (Strength of Evidence = A)	Revision of MI criteria
9.7	Biventricular pacing therapy should be considered for patients with sinus rhythm, a widened QRS interval (≥120 ms) and severe LV systolic dysfunction (LVEF ≤35% with LV dilatation >5.5 cm) who have persistent, moderate to severe HF (NYHA III) despite optimal medical therapy. (Strength of Evidence = A)	Biventricular pacing therapy is recommended for patients in sinus rhythm with a widened QRS interval (≥120 ms) and severe LV systolic dysfunction LVEF (≤ 35%) who have persistent, moderate to severe HF (NYHA III) despite optimal medical therapy. (Strength of Evidence = A)	Modification from “should be considered” to “is recommended”; removal of LV dimension criterion

9.8		Biventricular pacing therapy may be considered for patients with atrial fibrillation with a widened QRS interval (≥ 120 ms) and severe LV systolic dysfunction LVEF $\leq 35\%$ who have persistent, moderate to severe HF (NYHA III) despite optimal medical therapy. (Strength of Evidence = B)	New recommendation
9.9 (Previous 9.8)	Selected ambulatory NYHA IV patients may be considered for biventricular pacing therapy. (Strength of Evidence = B)	Selected ambulatory NYHA IV patients in sinus rhythm with QRS ≥ 120 ms and LV systolic dysfunction may be considered for biventricular pacing therapy. (Strength of Evidence = B)	Additional criteria for patient selection
9.10 (previous 9.9)	Biventricular pacing therapy is not recommended in patients who are asymptomatic or have mild HF symptoms. (Strength of Evidence = C)	Biventricular pacing therapy may be considered in patients with reduced LVEF and QRS ≥ 150 ms who have NYHA I or II HF symptoms. (Strength of Evidence = B)	Modification from “is not recommended” to “may be considered”; modification of strength of evidence from C to B; additional criteria for patient selection
9.11		In patients with reduced LVEF who require chronic pacing and in whom frequent ventricular pacing is expected, biventricular pacing may be considered. (Strength of Evidence = C)	New recommendation
9.12 (previous 9.10)	No changes		
Section 10: Surgical Approaches to the Treatment of Heart Failure			
10.1	No changes		
10.2	No changes		
10.3	No changes		
10.4	No changes		
10.5	No changes		
10.6	No changes		
10.7		Patients with refractory HF and hemodynamic instability, and/or compromised end-organ function, with relative contraindications to cardiac transplantation or permanent mechanical circulatory assistance expected to improve with time or restoration of an improved hemodynamic profile should be considered for urgent mechanical circulatory support as a “bridge to decision.” These patients should be referred to a center with expertise in the management of patients with advanced HF. (Strength of Evidence = C)	New recommendation
Section 11: Evaluation and Management of Patients with Heart Failure and Preserved LVEF			
11.1	Careful attention to differential diagnosis is recommended in patients with HF and preserved LVEF to distinguish among a variety of cardiac disorders, because treatments may differ. These various entities may be distinguished based on echocardiography, electrocardiography, and stress imaging (via exercise or pharmacologic means using myocardial perfusion or echocardiographic imaging). See algorithm in Figure 11.1 for a detailed approach to differential diagnosis. (Strength of Evidence = C)	Careful attention to differential diagnosis is recommended in patients with HF and preserved LVEF to distinguish among a variety of cardiac disorders, because treatments may differ. These various entities may be distinguished based on echocardiography, electrocardiography, and stress imaging (via exercise or pharmacologic means, using myocardial perfusion or echocardiographic imaging) and cardiac catheterization. See Figures 11.1, 11.2, and 11.3 for guidance to a differential diagnosis. (Strength of Evidence = C)	Addition of cardiac catheterization to list of diagnostic tools, modification of Figure 11.3 and addition of Figures 11.1 and 11.2.

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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
11.2	Evaluation for the possibility of ischemic heart disease and inducible myocardial ischemia is recommended in patients with HF and preserved LVEF. (Strength of Evidence = C)	Evaluation for ischemic heart disease and inducible myocardial ischemia is recommended in patients with HF and preserved LVEF (see Section 13). (Strength of Evidence = C)	Minor wording modifications
11.3	Aggressive blood pressure management is recommended in patients with HF and preserved LVEF (Section 14, Recommendation 14.15). (Strength of Evidence = C)	Blood pressure monitoring is recommended in patients with HF and preserved LVEF (Section 14, Recommendation 14.1). (Strength of Evidence = C)	Modification of terminology (“aggressive blood pressure management” changed to “blood pressure monitoring”)
11.4	No changes		
11.5	No changes		
11.6	ARBs or ACE inhibitors should be considered in patients with HF and preserved LVEF. (Strength of evidence = B) <ul style="list-style-type: none"> • ARBs (Strength of Evidence = B) • ACE inhibitors (Strength of Evidence = C) 	In the absence of other specific indications for these drugs, ARBs or ACE inhibitors may be considered in patients with HF and preserved LVEF. <ul style="list-style-type: none"> • ARBs (Strength of Evidence = C) • ACE inhibitors (Strength of Evidence = C) 	Modification from “should be considered” to “may be considered”; modification of strength of evidence for ARBs from B to C
11.7	No changes		
11.8	No changes		
11.9	Calcium channel blockers should be considered in patients with: <ul style="list-style-type: none"> • Atrial fibrillation requiring control of ventricular rate in whom b-blockers have proven inadequate for this purpose because of intolerance. In these patients, diltiazem or verapamil should be considered. (Strength of Evidence = C) • Symptom-limiting angina. (Strength of Evidence = A) • Hypertension. Amlodipine should be considered. (Strength of Evidence = C) 	Calcium channel blockers should be considered in patients with HF and preserved LVEF and: <ul style="list-style-type: none"> • Atrial fibrillation requiring control of ventricular rate and intolerance to beta blockers. In these patients, diltiazem or verapamil should be considered. (Strength of Evidence = C) • Symptom-limiting angina. (Strength of Evidence = A) • Hypertension. (Strength of Evidence = C) 	Modification of wording regarding beta blocker intolerance
11.10	Measures to restore and maintain sinus rhythm should be considered in patients who have symptomatic atrial flutter-fibrillation, but this decision should be individualized. (Strength of Evidence = C)	Measures to restore and maintain sinus rhythm may be considered in patients who have symptomatic atrial flutter-fibrillation and preserved LVEF, but this decision should be individualized. (Strength of Evidence = C)	Modification from “should be considered” to “may be considered”
Section 12: Evaluation and Management of Patients with Acute Decompensated Heart Failure			
12.1	The diagnosis of decompensated HF should be based primarily on signs and symptoms. (Strength of Evidence = C) When the diagnosis is uncertain, determination of BNP or NT-proBNP concentration should be considered in patients being evaluated for dyspnea who have signs and symptoms compatible with HF. (Strength of Evidence = A) The natriuretic peptide concentration should not be interpreted in isolation, but in the context of all available clinical data bearing on the diagnosis of HF.	The diagnosis of ADHF should be based primarily on signs and symptoms. (Strength of Evidence = C) When the diagnosis is uncertain, determination of BNP or NT-proBNP concentration is recommended in patients being evaluated for dyspnea who have signs and symptoms compatible with HF. (Strength of Evidence = A) The natriuretic peptide concentration should not be interpreted in isolation, but in the context of all available clinical data bearing on the diagnosis of HF, and with the knowledge of cardiac and non-cardiac factors that can raise or lower natriuretic peptide levels.	Modification of BNP recommendation from “should be considered” to “is recommended”
12.2	No changes		

12.3	No changes		
12.4	No changes		
12.5	No changes		
12.6	It is recommended that diuretics be administered at doses needed to produce a rate of diuresis sufficient to achieve optimal volume status with relief of signs and symptoms of congestion (edema, elevated JVP, dyspnea), without inducing an excessively rapid reduction in intravascular volume, which may result in symptomatic hypotension and/or worsening renal function. (Strength of Evidence = C)	It is recommended that diuretics be administered at doses needed to produce a rate of diuresis sufficient to achieve optimal volume status with relief of signs and symptoms of congestion (edema, elevated JVP, dyspnea), without inducing an excessively rapid reduction in 1) intravascular volume, which may result in symptomatic hypotension and/or worsening renal function, or 2) serum electrolytes, which may precipitate arrhythmias or muscle cramps. (Strength of Evidence = C)	Addition of serum electrolytes
12.7	No changes		
12.8	Monitoring of daily weights, intake, and output is recommended to assess clinical efficacy of diuretic therapy. Routine use of a Foley catheter is not recommended for monitoring volume status. However, placement of a catheter is recommended when close monitoring of urine output is needed. (Strength of Evidence = C)	Monitoring of daily weights, intake, and output is recommended to assess clinical efficacy of diuretic therapy. Routine use of a Foley catheter is not recommended for monitoring volume status. However, placement of a catheter is recommended when close monitoring of urine output is needed or if a bladder outlet obstruction is suspected of contributing to worsening renal function. (Strength of Evidence = C)	Addition of criterion for catheter placement
12.9	Careful observation for development of a variety of side effects, including renal dysfunction, electrolyte abnormalities and symptomatic hypotension, is recommended in patients treated with diuretics, especially when used at high doses and in combination. Patients should undergo routine laboratory studies and clinical examination as dictated by their clinical response. (Strength of Evidence = C) Serum potassium and magnesium levels should be monitored at least daily and maintained in the normal range. More frequent monitoring may be necessary when diuresis is rapid. (Strength of Evidence = C) Overly rapid diuresis may be associated with severe muscle cramps, which should be treated with potassium replacement if indicated. (Strength of Evidence = C)	Careful observation for development of a variety of side effects, including renal dysfunction, electrolyte abnormalities, symptomatic hypotension, and gout is recommended in patients treated with diuretics, especially when used at high doses and in combination. Patients should undergo routine laboratory studies and clinical examination as dictated by their clinical response. (Strength of Evidence = C) It is recommended that serum potassium and magnesium levels should be monitored at least daily and maintained in the normal range. More frequent monitoring may be necessary when diuresis is rapid. (Strength of Evidence = C) Overly rapid diuresis may be associated with severe muscle cramps. If indicated, treatment with potassium replacement is recommended. (Strength of Evidence = C)	Addition of gout as side effect Wording modified
12.10	No changes		
12.11	When congestion fails to improve in response to diuretic therapy, the following options should be considered: <ul style="list-style-type: none"> • Sodium and fluid restriction, • Increased doses of loop diuretic, • Continuous infusion of a loop diuretic, or • Addition of a second type of diuretic orally (metolazone or spironolactone) or intravenously (chlorothiazide). • A fifth option, ultrafiltration, may be considered. (Strength of Evidence = C) 	When congestion fails to improve in response to diuretic therapy, the following options should be considered: <ul style="list-style-type: none"> • Re-evaluating presence/absence of congestion • Sodium and fluid restriction, • Increasing doses of loop diuretic, • Continuous infusion of a loop diuretic, or • Addition of a second type of diuretic orally (metolazone or spironolactone) or intravenously (chlorothiazide). Another option, ultrafiltration, may be considered. (Strength of Evidence = C)	Addition of re-evaluation of congestion
12.12	A low-sodium diet (2 g daily) is recommended, as is supplemental oxygen, as needed for hypoxemia. (Strength of Evidence = C) In patients with recurrent or refractory volume overload, stricter sodium restriction may be considered. (Strength of Evidence = C)	A low sodium diet (2 g daily) is recommended for most hospitalized patients. (Strength of Evidence = C) In patients with recurrent or refractory volume overload, stricter sodium restriction may be considered. (Strength of Evidence = C)	Deletion of supplemental oxygen (moved to recommendation 12.14)

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2006 Guideline Recommendation		2010 Guideline Recommendation	Comments
12.13	No changes		
12.14	Routine administration of supplemental oxygen in the absence of hypoxia is not recommended. (Strength of Evidence = C)	Routine administration of supplemental oxygen in the presence of hypoxia is recommended. (Strength of Evidence = C) Routine administration of supplemental oxygen in the absence of hypoxia is not recommended. (Strength of Evidence = C)	Addition of recommendation for oxygen in the presence of hypoxemia
12.15		Use of non-invasive positive pressure ventilation may be considered for severely dyspneic patients with clinical evidence of pulmonary edema. (Strength of Evidence = A)	New recommendation
12.16		Venous thromboembolism prophylaxis with low dose unfractionated heparin, low molecular weight heparin, or fondaparinux to prevent proximal deep venous thrombosis and pulmonary embolism is recommended for patients who are admitted to the hospital with ADHF and who are not already anticoagulated and have no contraindication to anticoagulation. (Strength of Evidence = B) Venous thromboembolism prophylaxis with a mechanical device (intermittent pneumatic compression devices or graded compression stockings) to prevent proximal deep venous thrombosis and pulmonary embolism should be considered for patients who are admitted to the hospital with ADHF and who are not already anticoagulated and who have a contraindication to anticoagulation. (Strength of Evidence = C)	New recommendation
12.17 (previous 12.15)	In the absence symptomatic hypotension, intravenous nitroglycerin, nitroprusside, or nesiritide may be considered as an addition to diuretic therapy for rapid improvement of congestive symptoms in patients admitted with ADHF. Frequent blood pressure monitoring is recommended with these agents. (Strength of Evidence = B). These agents should be decreased in dosage or discontinued if symptomatic hypotension develops. (Strength of Evidence = B) Reintroduction in increasing doses may be considered once symptomatic hypotension is resolved. (Strength of Evidence = C)	In the absence of symptomatic hypotension, intravenous nitroglycerin, nitroprusside or nesiritide may be considered as an addition to diuretic therapy for rapid improvement of congestive symptoms in patients admitted with ADHF. (Strength of Evidence = B) Frequent blood pressure monitoring is recommended with these agents. (Strength of Evidence = B) These agents should be decreased in dosage or discontinued if symptomatic hypotension or worsening renal function develops. (Strength of Evidence = B) Reintroduction in increasing doses may be considered once symptomatic hypotension is resolved. (Strength of Evidence = C)	Addition of worsening renal function as potential side effect
12.18 (previous 12.16)	No changes		
12.19 (previous 12.17)	Intravenous vasodilators (nitroprusside, nitroglycerin, or nesiritide) may be considered in patients with ADHF and advanced HF who have persistent severe HF despite aggressive treatment with diuretics and standard oral therapies. (Strength of Evidence = C)	Intravenous vasodilators (nitroprusside, nitroglycerin, or nesiritide) may be considered in patients with ADHF who have persistent severe HF despite aggressive treatment with diuretics and standard oral therapies. • Nitroprusside (Strength of Evidence = B) • Nitroglycerine, Nesiritide (Strength of Evidence = C)	Modification of strength of evidence for nitroprusside from C to B

12.20 (previous 12.18)	<p>Intravenous inotropes (milrinone or dobutamine) may be considered to relieve symptoms and improve end-organ function in patients with advanced HF characterized by LV dilation, reduced LVEF, and diminished peripheral perfusion or end-organ dysfunction (low output syndrome), particularly if these patients have marginal systolic blood pressure (<90 mm Hg), have symptomatic hypotension despite adequate filling pressure, or are unresponsive to, or intolerant of, intravenous vasodilators. (Strength of Evidence = C)</p> <p>These agents may be considered in similar patients with evidence of fluid overload if they respond poorly to intravenous diuretics or manifest diminished or worsening renal function. (Strength of Evidence = C)</p> <p>When adjunctive therapy is needed in other patients with ADHF, administration of vasodilators should be considered instead of intravenous inotropes (milrinone or dobutamine). (Strength of Evidence = B)</p> <p>Intravenous inotropes (milrinone or dobutamine) are not recommended unless left heart filling pressures are known to be elevated based on direct measurement or clear clinical signs. (Strength of Evidence = B)</p> <p>Administration of intravenous inotropes (milrinone or dobutamine) in the setting of ADHF should be accompanied by continuous or frequent blood pressure monitoring and continuous monitoring of cardiac rhythm. (Strength of Evidence = C)</p> <p>If symptomatic hypotension or worsening tachyarrhythmias develop during administration of these agents, discontinuation or dose reduction should be considered. (Strength of Evidence = C)</p>	<p>Intravenous inotropes (milrinone or dobutamine) may be considered to relieve symptoms and improve end-organ function in patients with advanced HF characterized by LV dilation, reduced LVEF, and diminished peripheral perfusion or end-organ dysfunction (low output syndrome), particularly if these patients have marginal systolic blood pressure (< 90 mm Hg), have symptomatic hypotension despite adequate filling pressure, or are unresponsive to, or intolerant of, intravenous vasodilators. (Strength of Evidence = C)</p> <p>These agents may be considered in similar patients with evidence of fluid overload if they respond poorly to intravenous diuretics or manifest diminished or worsening renal function. (Strength of Evidence = C)</p> <p>When adjunctive therapy is needed in other patients with ADHF, administration of vasodilators should be considered instead of intravenous inotropes (milrinone or dobutamine). (Strength of Evidence = C)</p> <p>Intravenous inotropes (milrinone or dobutamine) are not recommended unless left heart filling pressures are known to be elevated or cardiac index is severely impaired based on direct measurement or clear clinical signs. (Strength of Evidence = C)</p> <p>It is recommended that administration of intravenous inotropes (milrinone or dobutamine) in the setting of ADHF be accompanied by continuous or frequent blood pressure monitoring and continuous monitoring of cardiac rhythm. (Strength of Evidence = C)</p> <p>If symptomatic hypotension or worsening tachyarrhythmias develop during administration of these agents, discontinuation or dose reduction should be considered. (Strength of Evidence = C)</p>	<p>Modification of strength of evidence from B to C for portions of this recommendation</p> <p>Wording modified</p>
12.21 (previous 12.19)	No changes		
12.22 (previous 12.20)	<p>Invasive hemodynamic monitoring should be considered in a patient:</p> <ul style="list-style-type: none"> • Who is refractory to initial therapy, • Whose volume status and cardiac filling pressures are unclear, • Who has clinically significant hypotension (typically systolic blood pressure <80 mm Hg) or worsening renal function during therapy, or • In whom documentation of an adequate hemodynamic response to the inotropic agent is necessary when chronic outpatient infusion is being considered. <p>(Strength of Evidence = C)</p>	<p>Invasive hemodynamic monitoring should be considered in a patient:</p> <ul style="list-style-type: none"> • who is refractory to initial therapy, • whose volume status and cardiac filling pressures are unclear, • who has clinically significant hypotension (typically SBP < 80mm Hg) or worsening renal function during therapy, or • who is being considered for cardiac transplant and needs assessment of degree and reversibility of pulmonary hypertension, or • in whom documentation of an adequate hemodynamic response to the inotropic agent is necessary when chronic outpatient infusion is being considered. <p>(Strength of Evidence = C)</p>	<p>Addition of cardiac transplant as criterion for invasive hemodynamic monitoring</p>
12.23 (previous 12.21)	No changes		
12.24 (previous 12.22)	<p>It is recommended that every effort be made to use the hospital stay for assessment and improvement of patient compliance via patient and family education and social support services (Section 8). (Strength of Evidence = C)</p>	<p>It is recommended that every effort be made to use the hospital stay for assessment and improvement of patient adherence via patient and family education and social support services (see Section 8). (Strength of Evidence = B)</p>	<p>Modification of strength of evidence from C to B; change in terminology (“compliance” to “adherence”)</p>

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Appendix A. (continued)

	2006 Guideline Recommendation	2010 Guideline Recommendation	Comments
12.25 (previous 12.23)	No changes		
12.26 (previous 12.24)	<p>Discharge planning is recommended as part of the management of patients with ADHF. Discharge planning should address the following issues:</p> <ul style="list-style-type: none"> • Details regarding medication, dietary sodium restriction, and recommended activity level • Follow-up by phone or clinic visit early after discharge to reassess volume status • Medication and dietary compliance • Monitoring of body weight, electrolytes, and renal function • Consideration of referral for formal disease management (Strength of Evidence = C) 	<p>Discharge planning is recommended as part of the management of patients with ADHF. Discharge planning should address the following issues:</p> <ul style="list-style-type: none"> • Details regarding medication, dietary sodium restriction, and recommended activity level • Follow-up by phone or clinic visit early after discharge to reassess volume status • Medication and dietary compliance • Alcohol moderation and smoking cessation • Monitoring of body weight, electrolytes and renal function • Consideration of referral for formal disease management (Strength of Evidence = C) 	Addition of alcohol moderation and smoking cessation
Section 13: Evaluation and Therapy for Heart Failure in the Setting of Ischemic Heart Disease			
13.1	<p>Assessment for risk factors for CAD is recommended in all patients with chronic HF regardless of EF. (Strength of Evidence = A)</p> <p>The diagnostic approach for CAD should be individualized based on patient preference and comorbidities, eligibility and willingness to perform revascularization. (Strength of Evidence = C)</p>	Ongoing assessment for risk factors for CAD is recommended in all patients with chronic HF regardless of LVEF. (Strength of Evidence = A)	Moved diagnostic portion of recommendation to 13.2
13.2		It is recommended that the diagnostic approach for CAD be individualized based on patient preference and comorbidities, eligibility, symptoms suggestive of angina and willingness to undergo revascularization. (Strength of Evidence = C)	Previously part of 13.1
13.3 (previous 13.2)	It is recommended that patients with HF and angina undergo cardiac catheterization with coronary angiography to assess for potential revascularization. (Strength of Evidence = B)	It is recommended that patients with HF and symptoms suggestive of angina undergo cardiac catheterization with coronary angiography to assess for potential revascularization. (Strength of Evidence = B)	Modification of wording
13.4 (previous 13.3)	It is recommended that patients with HF, no angina, and known CAD should undergo noninvasive stress imaging and/or coronary angiography to assess severity of coronary disease and the presence of ischemia. (Strength of Evidence = C)	It is recommended that, at the initial diagnosis of HF and any time symptoms worsen without obvious cause, patients with HF, no angina, and known CAD should undergo risk assessment that may include noninvasive stress imaging and/or coronary angiography to assess severity of coronary disease and the presence of ischemia. (Strength of Evidence = C)	Clarification of type and timing of risk assessments
13.5 (previous 13.4)	No changes		
13.6 (previous 13.5)	No changes		

13.7 (previous 13.6)	Any of the following imaging tests may be used to identify inducible ischemia or viable but noncontractile myocardium: <ul style="list-style-type: none"> • Exercise or pharmacologic stress myocardial perfusion imaging • Exercise or pharmacologic stress echocardiography • Cardiac magnetic resonance imaging • Positron emission tomography scanning (Strength of Evidence = B) 	Any of the following imaging tests should be considered to identify inducible ischemia or viable myocardium: <ul style="list-style-type: none"> • Exercise or pharmacologic stress myocardial perfusion imaging • Exercise or pharmacologic stress echocardiography • Cardiac magnetic resonance imaging • Positron emission tomography scanning (Strength of Evidence = B) 	Modification of wording
13.8 (previous 13.7)	No changes		
13.9 (previous 13.8)	Antiplatelet therapy is recommended in patients with HF and CAD unless contraindicated. (Aspirin, Strength of Evidence = B; Clopidogrel, Strength of Evidence = C)	Antiplatelet therapy is recommended to reduce vascular events in patients with HF and CAD unless contraindicated. (aspirin, Strength of Evidence = A; clopidogrel, Strength of Evidence = B)	Addition of indication for antiplatelet therapy, and modification of strength of evidence
13.10 (previous 13.9)	ACE inhibitors are recommended in all patients with systolic dysfunction or preserved systolic function after an MI. (Strength of Evidence = A)	ACE inhibitors are recommended in all patients with either reduced or preserved LVEF after an MI. (Strength of Evidence = A)	Modification of terminology (“systolic dysfunction” changed to “reduced LVEF”)
13.11 (previous 13.10)	No changes		
13.12 (previous 13.11)	It is recommended that ACE-inhibitor and beta blocker therapy be initiated early (<48 hours) during hospitalization in hemodynamically stable post MI patients with LV dysfunction or HF. (Strength of Evidence = A)	It is recommended that ACE-inhibitor and beta blocker therapy be initiated early (<48 hours) during hospitalization in hemodynamically stable post-MI patients with reduced LVEF or HF. (Strength of Evidence = A)	Modification of terminology (“LV dysfunction” changed to “reduced LVEF”)
13.13 (previous 13.12)	No changes		
13.14 (previous 13.13)	Calcium channel blockers should be considered in patients with HF who have angina despite the optimal use of beta blockers and nitrates. Amlodipine and felodipine are the preferred calcium channel blockers in patients with angina and decreased systolic function. (Strength of Evidence = C)	Calcium channel blockers may be considered in patients with HF who have angina despite the optimal use of beta blockers and nitrates. Amlodipine and felodipine are the preferred calcium channel blockers in patients with angina and decreased systolic function. Based on available data, first generation calcium channel blockers (i.e. diltiazem, verapamil) should be avoided in patients with CAD, HF, and LVEF <40, unless necessary for heart rate control or other indications. (Strength of Evidence = C)	Addition of calcium channel blockers that should be avoided
13.15 (previous 13.14)	No changes		
13.16 (previous 13.15)	No changes		
Section 14: Managing Patients with Hypertension and Heart Failure			
14.1	It is recommended that blood pressure be aggressively treated to lower systolic and usually diastolic levels. Target resting levels should be <130/<80 mm Hg, if tolerated. (Strength of Evidence = C)	It is recommended that blood pressure be optimally treated to lower systolic and usually diastolic levels. More than 1 drug may be required. Target resting levels should be <130/<80 mm Hg, if tolerated. (Strength of Evidence = A)	Modification of wording and change in strength of evidence from C to A

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Appendix A. (continued)

2006 Guideline Recommendation		2010 Guideline Recommendation	Comments
Previous 14.2			Deleted
14.2 (previous 14.3)	No changes		
14.3 (previous 14.4)	No changes		
14.4 (previous 14.5)	If BP remains > 130/80 mm Hg then the addition of a diuretic is recommended, followed by a calcium antagonist or other antihypertensive drugs. (Strength of Evidence = C)	If blood pressure remains > 130/80 mm Hg then the addition of a thiazide diuretic is recommended, followed by a dihydropyridine calcium antagonist (eg, amlodipine or felodipine) or other antihypertensive drugs. (Strength of Evidence = C)	Modified to specify thiazide diuretic or dihydropyridine calcium channel antagonist
14.5 (previous 14.6)	No changes		
14.6 (previous 14.7)	If blood pressure remains > 130/80 mm Hg, a noncardiac-depressing calcium antagonist (eg, amlodipine) may be considered or other antihypertensive medication doses increased. (Strength of Evidence = C)	If blood pressure remains > 130/80 mm Hg, a dihydropyridine calcium antagonist (eg, amlodipine or felodipine) may be considered or other antihypertensive medication doses increased. (Strength of Evidence = C)	Modified to specify dihydropyridine
Section 15: Management of Heart Failure in Special Populations			
15.1	No changes		
15.2	No changes		
15.3	No changes		
15.4	No changes		
15.5	No changes		
15.6		ARBs are recommended for administration to symptomatic and asymptomatic women with an LVEF \leq 40% who are intolerant to ACE inhibitors for reasons other than hyperkalemia or renal insufficiency. (Strength of Evidence = A)	New recommendation
15.7		The combination of hydralazine/isosorbide dinitrate is recommended as standard therapy for African American women with moderate to severe HF symptoms who are on background neurohormonal inhibition. (Strength of Evidence = B)	New recommendation
15.8 (previous 15.6)	No changes		

15.9 No changes
(previous
15.7)

15.10 No changes
(previous
15.8)

15.11 No changes
(previous
15.9)

Section 16: Myocarditis: Current Treatment

16.1 No changes

16.2 No changes

Section 17: Genetic Evaluation of Cardiomyopathy

New section

Appendix C. Financial Disclosure

Name	Consulting Fees/Honoraria	Speaker's Bureau	Research Grants	Equity Interests/ Stock/Stock Options	Equity Interests	Royalty Income	Non-Royalty Payments	Other Financial Benefit	Salary	Intellectual Property Rights	Fellowship Support
Nancy M. Albert, R.N., Ph.D	Medtronic										
Inder S. Anand, M.D., Ph.D.	Amgen Pharmaceuticals, Boston Scientific, Corventis, CVRx, Merck, Medtronic, N30, Paracor	Novartis Pharmaceuticals	CVRx, Novartis Pharmaceuticals, Paracor						VA Medical Center		
J. Malcolm O. Arnold, M.D.	Abbott, Boehringer Ingelheim, GlaxoSmithKline, Merck-Frosst, Novartis, Pfizer										
John P. Boehmer, M.D.	Boston Scientific, Medtronic, St. Jude		Boston Scientific, CardioMEMS, Medtronic, Novartis, Paracor								
John C. Burnett, M.D.	Anexon, Nile Therapeutics, Otsuka		Anexon, Bayer, BioRad, Merck, Nile Therapeutics, Trevena			Anexon, Nile Therapeutics					
John Chin, M.D.	Gilead, Otsuka	Boston Scientific, Eli Lilly, Gilead, Novartis									
Jay N. Cohn, M.D.			GlaxoSmithKline	CPC, LLC	HDT, Inc.	MLHFQ, NitroMed					
Sean P. Collins, M.D., MSc	Abbott Point-of-Care, Astellas, Bayer, Corthera, The Medicines Company, Otsuka		Abbott Point-of-Care, BRAHMS Diagnostics, National Institutes of Health/NHLBI								
Justin A. Ezekowitz, MBBCh	Amgen, Bristol-Myers Squibb, Pfizer		Amgen, Bristol-Myers Squibb, Merck, Ortho-Biotech/Johnson & Johnson								
Thomas Force, M.D.		Merck Schering Plough/Merck						GlaxoSmithKline			
Bart Galle, Ph.D.	disclosures: none										

Michael M. Givertz, M.D.	Cardioxyl		Asahi Kasei							
Sarah J. Goodlin, M.D.	Servier		Boston Scientific CARE, St. Jude Medical Foundation							
Barry H. Greenberg, M.D.	Biogen Idec, CardioMEMS, Corthera, Cytokinetics, GlaxoSmithKline, Otsuka, Paracor, St. Jude, Zensun	Gilead, Merck, Novartis, sanofi-aventis								
Ray E. Hershberger, M.D.	disclosures: none									
Steven R. Houser, Ph.D.	disclosures: none									
Jonathan G. Howlett, M.D.	AstraZeneca, Merck, Novartis, Schering, Servier	AstraZeneca, Merck, Novartis, Schering, Servier	AstraZeneca, Medtronic, Merck, Novartis, Schering, Servier							
Sharon A. Hunt, M.D.	disclosures: none									
Mariell Jessup, M.D.	Medtronic	Boston Scientific								
Stuart D. Katz, M.D.	Amgen, Dura Heart Terumo, Merck, Paracor									
Marc Klapholz, M.D.	GlaxoSmithKline, Medtronic, Paracor, St. Jude, Schering	GlaxoSmithKline								
Marvin W. Kronenberg, M.D.				Cardiovascular Services of America						
JoAnn Lindenfeld, M.D.	Astellas, Boston Scientific, Forest, Medtronic, N30		Merck							
Douglas L. Mann, M.D.	ARMGO Pharmaceuticals, Medtronic, Miragen, Nile Therapeutics, PeriCor Therapeutics				Miragen					

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Appendix C. (continued)

Name	Consulting Fees/Honoraria	Speaker's Bureau	Research Grants	Equity Interests/ Stock/Stock Options	Equity Interests	Royalty Income	Non-Royalty Payments	Other Financial Benefit	Salary	Intellectual Property Rights	Fellowship Support
Barry M. Massie, M.D.	ARCA, Boehringer, Bristol-Myers Squibb, Corthera, Cytokinetics, Duke Clinical Research Institute, Merck, Nile Therapeutics, Novartis, sanofi-aventis, St. Jude, Takeda, Trevena	Merck									
Mandeep R. Mehra, M.D.	Geron, Johnson & Johnson, Medtronic, Pericor, Solvay, St. Jude		National Institutes of Health								
Luisa Mestroni, M.D.		M01 RR00051-1575						University of Colorado (employee)			
Alan B. Miller, M.D.	disclosures: none										
Debra K. Moser, R.N., DNSc.	disclosures: none										
Mariann R. Piano, R.N., Ph.D.	disclosures: none										
Richard J. Rodeheffer, M.D.	disclosures: none										
Joseph G. Rogers, M.D.	Forrest Pharmaceuticals, Thoratec	Boston Scientific, Medtronic									
Christine E. Seidman, M.D.			HHMI & NIH								
Randall C. Starling, M.D., MPH	BioControl, Medtronic, Novartis		Biotronik, Medtronic, Novartis, Thoratec	CardioMEMS							Medtronic
William G. Stevenson, M.D.	disclosures: none										

Wendy Gattis Stough, Pharm.D.	ARCA Discovery, Inc., Gilead Sciences, Inc., GlaxoSmithKline, Heart Failure Society of America, Medtronic, Otsuka, Scios										
W.H. Wilson Tang, M.D.	Medtronic, Merck & Company		Abbott Laboratories								
Matthew R.G. Taylor, M.D., Ph.D.		Genzyme Therapeutics	Muscular Dystrophy Association/March of Dimes/Genzyme Therapeutics								
John R. Teerlink, M.D.	Abbott Laboratories, BAS Medical/Corthera, Biogen Idec, Bristol-Myers Squibb, CardioDynamics, CardioMEMS, CoGeneSys, Cytokinetics, Geron, GlaxoSmithKline, Icon Medical Imaging, Indigo Pharma, Kowa Pharma, Luitpold Pharma, Merck, Momentum Research, Nile Therapeutics, Novartis, sanofi-aventis, Scios/Johnson & Johnson		Abbott Laboratories, BAS Medical/Corthera, Bristol-Myers Squibb, Cytokinetics, GlaxoSmithKline, Merck, National Institutes of Health, Novartis, sanofi-aventis	Cytokinetics							
Jeffery A. Towbin, M.D.	disclosure: none										
Mary N. Walsh, M.D.	ARCA, Boston Scientific, EMERGE, Medtronic, United Health Care										
Clyde W. Yancy, M.D.	disclosures: none										
Cheryl Yano	disclosures: none										
Michael R. Zile, M.D.	ABIM, BMS, CorAssist, CVRx, DC Devices, Gilead, Medtronic, Merck, N30, Novartis, OCD/J&J, sanofi-aventis, Up-To-Date		BMS, Boston, Scientific, CVRx, Department of VA, Gilead, Medtronic, Merck, National Heart, Lung and Blood Institute, Novartis, OCD/J&J, Pfizer, sanofi-aventis						Department of VA, MUSC, National Heart, Lung and Blood Institute	MUSC, OCD/J&J	