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EXPERT CONSENSUS DECISION PATHWAY

2019 ACC Expert Consensus Decision Pathway on Risk Assessment, Management, and Clinical Trajectory of Patients Hospitalized With Heart Failure

A Report of the American College of Cardiology Solution Set Oversight Committee

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PREFACE

The American College of Cardiology (ACC) has a long history of developing documents (e.g., decision pathways, health policy statements, appropriate use criteria) to provide members with guidance on both clinical and nonclinical topics relevant to cardiovascular care. In most circumstances, these documents have been created to complement clinical practice guidelines and to inform clinicians about areas where evidence may be new and evolving or where sufficient data may be more limited. In spite of this, numerous care gaps continue to exist, highlighting the need for more streamlined and efficient processes to implement best practices in service to improved patient care.

Central to the ACC's strategic plan is the generation of "actionable knowledge"—a concept that places emphasis on making clinical information easier to consume, share, integrate, and update. To this end, the ACC has evolved from developing isolated documents to the development of integrated "solution sets." Solution sets are groups of

closely related activities, policy, mobile applications, decision support, and other tools necessary to transform care and/or improve heart health. Solution sets address key questions facing care teams and attempt to provide practical guidance to be applied at the point of care. They use both established and emerging methods to disseminate information for cardiovascular conditions and their related management. The success of the solutions sets rests firmly on their ability to have a measurable impact on the delivery of care. Because solutions sets reflect current evidence and ongoing gaps in care, the associated tools will be refined over time to best match member needs.

Expert consensus decision pathways (ECDPs) represent a key component of solution sets. The methodology for ECDPs is grounded in assembling a group of clinical experts to develop content that addresses key questions facing our members across a range of high-value clinical topics. This content is used to inform the development of various tools that accelerate real time use of clinical policy at the point of care. They are not intended to provide a single correct answer; rather, they encourage clinicians to ask questions and consider important factors as they define a treatment plan for their patients. Whenever appropriate, ECDPs seek to provide unified articulation of clinical practice guidelines, appropriate use criteria, and other related ACC clinical policy. In some cases, covered topics will be addressed in subsequent clinical practice guidelines as the evidence base evolves. In other cases, these will serve as standalone policy.

Ty J. Gluckman, MD, FACC
Chair, ACC Solution Set Oversight Committee

1. INTRODUCTION

Heart failure (HF) affects nearly 6.2 million Americans and is the primary diagnosis for hospital discharge in about 1 million and a secondary diagnosis in about 2 million hospitalizations annually (1). By 2030, more than 8 million people in the United States (1 in every 33) will have HF (2). HF makes up 1% to 2% of the total healthcare budget in the United States (3), with inpatient admissions accounting for more than one-half of this expenditure (4,5). Inpatient mortality ranges from 4% to 12% and may increase to 20% to 25% in high-risk subgroups (6–11). Readmissions and events are common, and the age-adjusted risk for all-cause mortality is tripled compared with non-HF patients (3,6,12).

While the typical hospital course includes rapid improvement in signs and symptoms and discharge after 4 to 5 days, episodes of worsening HF nevertheless mark a fundamental change in the HF trajectory; patients admitted with HF have a 20% to 30% risk of death within a year. Goals of hospitalization thus include not only clinical response, but also the assessment and optimization of therapy to address the long-term trajectory after discharge.

This ECDP focuses on patients hospitalized with HF and complements existing tools for outpatient management. We have construed our task broadly to comprise assessment extending from the original emergency department (ED) visit through the first post-discharge visit. The primary purpose is to optimize patient care and improve outcomes, rather than to focus on reducing length of stay and readmission, although practice improvement may enhance effective resource allocation. The evaluation and management processes are continuous rather than discrete, although different considerations come into play at various points along the path. Finally, optimal flow and exchange of information throughout the hospitalization and care afterward is crucial to achieve the best outcomes.

This document focuses on assessments and goals of therapy. Specific therapies are discussed extensively in other guideline and consensus documents in the United States and Europe from American College of Cardiology Foundation (ACCF), American Heart Association (AHA), Heart Failure Society of America (HFSA), and European Society of Cardiology-Heart Failure Association (13–19). Our aim is to help clinicians consider the short- and long-term outlook for their patients with HF, to institute therapies to reduce symptoms and optimize outcomes, to ensure that those plans are conveyed clearly to caregivers after discharge, and to engage patients to share in decisions and become active participants in their care.

The document is structured into 5 nodes: Admission, Trajectory Check, transition to Oral Therapies, Discharge, and First Follow-Up Visit (**Figure 1**). Although these follow sequentially during an admission, their timing is flexible, and they clearly flow into each other. The trajectory check is a recurring theme rather than a specific event—the aim is to provide structure to the process of assessing the clinical course and planning future therapy. In addition, information collected at each point would ideally be accessible not only in the hospital, but also in outpatient settings and as a reference point for evaluation of recurrent presentations.

The document is designed to facilitate the creation of clinical tools to help improve outcomes. Some tools should facilitate collection and synthesis of patient information. Other tools support decisions among potential therapies. The most important measures of these tools will be how useful they are for providing patient care and how often they are used, but it is hoped that

they will also improve efficiencies of care and resource utilization as have the previous pivotal guidelines (17,18).

Risk assessment involves collection of information, but is most useful when that information is translated into strategies to address risk factors and to minimize risk going forward. This process may start during a hospitalization, but it should not end there. The document addresses collection and dissemination of information in several areas:

- Explicit goals for therapy and assessment of the degree to which they have been achieved
- Patient-specific comorbidities
- Barriers to care
- Therapies that have been titrated in-hospital and those planned for titration after discharge

The key to these processes is to make data acquisition as easy as possible during hospitalization, make therapeutic options as transparent as possible, and present information in a format that makes it readily accessible to members of the interprofessional team throughout the healthcare system (20,21).

The Writing Committee dedicates this document to the memory of Dr. Mihai Gheorghiade, who awakened them to HF hospitalization as an opportunity for thoughtful assessment and intervention to change the course of HF.

2. METHODS

The invited writing group participants represent the varied clinicians involved in the care of the patient with acute HF. A review of outstanding questions was facilitated. Subsequent writing assignments were configured according to areas of expertise. Teleconferences were used to edit contributed content. Conference calls of the writing committee were confidential and were attended only by committee members and ACC staff.

The work of the writing committee was supported exclusively by the ACC Foundation without commercial support. Writing committee members volunteered their time to this effort. All members of the writing committee, as well as those selected to serve as peer reviewers of this document, were required to disclose relationships with industry (RWI) and other entities (See Appendixes 1 and 2, respectively). The Chair is without any RWI and is responsible for the content of this document. In keeping with ACC policy, the majority of the writing committee were without relevant RWI. The formal peer review process was completed consistent with ACC policy, and included a public comment period to obtain further feedback. Following reconciliation of all comments, this document was approved for publication by the Clinical Policy Approval Committee.

3. ASSUMPTIONS AND DEFINITIONS

1. The committee decided not to distinguish HF on the basis of ejection fraction (EF) except where specifically noted. Although the evidence base for therapeutic interventions differs, the goals of decongestion and the importance of consideration of comorbidities and factors that influence adherence are common to patients admitted with reduced ejection fraction (HFrEF) or preserved ejection fraction (HFpEF). Management of patients with midrange ejection fraction (HFmrEF) shares similarities with management of both HFrEF and HFpEF (22–24).
2. The expert consensus writing committee endorses the evidence-based approaches to HF therapy and management enumerated in the 2013 ACCF/AHA Guideline for the Management of Heart Failure and the 2016 and 2017 ACC/AHA/HFSA focused updates (15,25,26).
3. These algorithms assume that a broad multidisciplinary approach is ideal, with input anticipated from experienced physician and nurse specialists, as well as other disciplines such as pharmacy, social work, psychiatry, physical therapy, and nutrition.
4. Therapeutic decisions should be governed by clinical judgment in accordance with patient preferences.
5. These algorithms are based on the best available data, but given the relatively limited current data concerning a number of aspects of the HF hospitalization, they will require revision as new data emerge.

3.1. Definitions

GDMT: Guideline-directed medical therapy

Optimal therapy: Treatment provided at either the target or the highest tolerated dose for a given patient.

EF: Ejection fraction

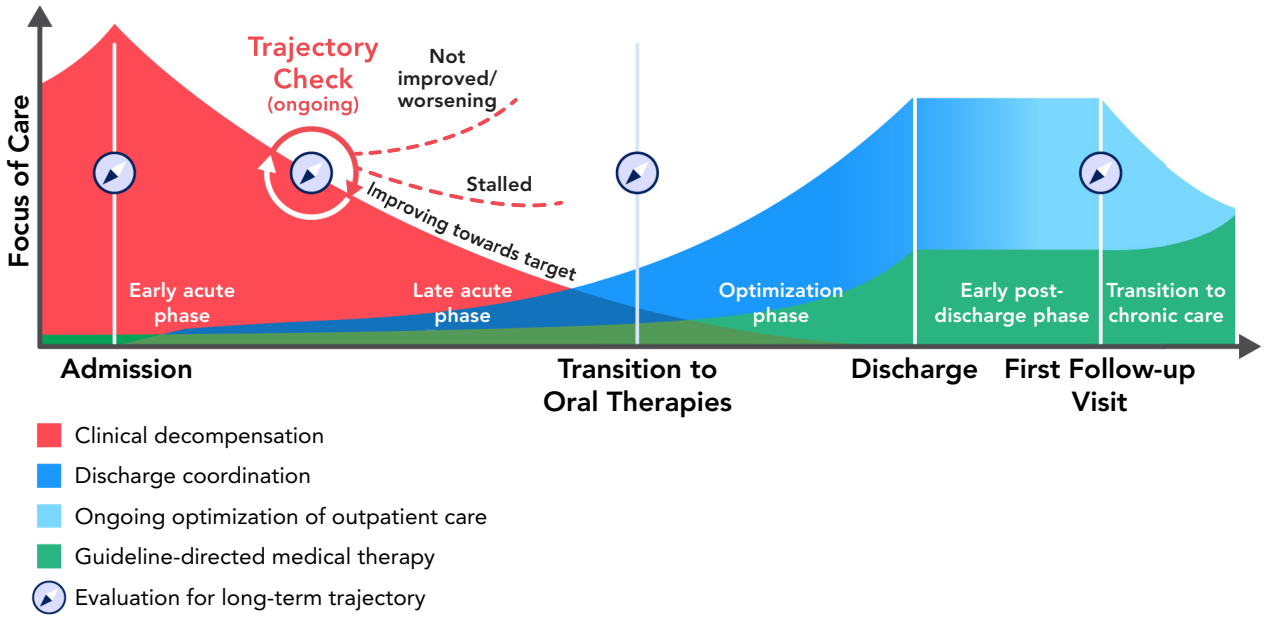
HFrEF: Heart failure with reduced left ventricular ejection fraction (EF $\leq$0.40)

HFpEF: Heart failure with preserved left ventricular ejection fraction (EF $\geq$0.50)

HFmrEF: Heart failure with midrange ejection fraction (EF $<$0.50 but $>$0.40)

4. PATHWAY SUMMARY GRAPHIC

FIGURE 1 Clinical Course of Heart Failure



Graphic depiction of course of heart failure admission, showing the degree of focus on clinical decompensation (red), discharge coordination (blue), ongoing coordination of outpatient care (light blue), and optimization of guideline-directed medical therapy (green), with ongoing assessment of the clinical course (circle with arrows), and key time points for review and revision of the long-term disease trajectory for the HF journey (compass signs).

5. DESCRIPTION AND RATIONALE

5.1. Key points

1. The pathway to improve outcomes after HF hospitalization begins with admission, continues through the process of decongestion and transition to oral therapies before the day of discharge, and connects through the first post-discharge follow-up.
2. Clinical trajectory of HF should be assessed continuously during admission. Three main in-hospital trajectories have been defined: *improving towards target, stalled after initial response,* or *not improved/worsening*. These translate into different management strategies throughout hospitalization and post-discharge.
3. Evaluation of the long-term course of HF should be part of the initial comprehensive assessment, reviewed on the day of transition to oral therapy, and re-assessed at the first follow-up visit for persistent or new indications of high risk leading to consideration of advanced therapies or revision of goals of care.
4. Key risk factors modifiable during hospitalization include the degree of congestion as assessed by clinical signs and natriuretic peptides and the lack of appropriate guideline-directed medical therapies. Improvement in these factors is associated with improved prognosis, but failure to improve, including failure to tolerate guideline-directed medical therapy (GDMT) for HF, is associated with a much worse prognosis.
5. Common comorbidities, including diabetes; anemia; and kidney, lung, and liver disease, should be assessed during initial evaluation and addressed throughout hospitalization and discharge planning.
6. The day of transition from intravenous to oral diuretic therapy should trigger multiple considerations related to the overall regimen for discharge, verification of completion of patient education components, caregiver education, and plans for discharge.
7. The discharge day should be a time to review and communicate with identified providers rather than to initiate new therapies.
8. The elements of the hospitalization events and plans that are most crucial for continuity of care after discharge should be documented in a format that is available to all members of the outpatient team and easily accessible when a patient calls or returns with worsening symptoms.
9. Principles of palliative care applied by the in-hospital care team or by palliative care specialists may be

particularly relevant when an unfavorable trajectory warrants communication about prognosis, options, and decision-making with patients and families.

10. The first follow-up visit should address specific aspects, including volume status, hemodynamic stability, kidney function and electrolytes, the regimen of recommended therapies, patient understanding, adherence challenges (including insurance/coverage issues), and goals of care.

6. NODE: ADMISSION

6.1. Evaluation in the ED

ED data show that 80% of all HF hospitalizations are admitted from the ED (27,28). Although many advances have improved chronic HF management, there is sparse evidence regarding strategies for triage and management in the ED (13,15-19,25,26,29,30). Most patients with acute decompensated heart failure (ADHF) are admitted for symptomatic treatment of congestion with intravenous diuretics and to a much lesser degree for respiratory failure, cardiogenic shock, incessant ventricular tachycardia, or the need for urgent diagnostic or therapeutic procedures (6,20,21,31-40). Although fewer than 10% of ED visits with ADHF have acute life-threatening illness, and the majority of patients presenting are clinically stable (11,38,39,41), the post-discharge event rate is high even though over 80% to 90% of patients are admitted (28,42,43).

A framework for risk stratification in the ED is shown in **Figure 2**, intended as a guide to thought processes during initial evaluations rather than a formal description of admission criteria and administrative processes surrounding admission. Patients who are critically ill at presentation or those with new-onset HF are admitted. Patients with known HF and a marked degree of congestion and those not at low risk (**Table 1**) are also usually admitted. Some patients with a clear correctable trigger, such as a brief lapse in diuretic dose, can be treated and discharged. ED decisions are typically constrained by the need for rapid assessment without knowledge of baseline clinical status and previous disease course, with pressure for rapid disposition under crowded conditions. Thus, the decision to admit an ED patient with HF is guided less often by severity of disease than by lack of information about physiological and social triggers, the complexity of comorbidities, and the uncertainty around disease trajectory in a setting in which later return to the ED may be seen as an error in disposition (28). If information and good baseline regimen are confirmed, but limitations to adherence are identified, a review of triggers and focused re-education may allow patients to be triaged to an observation unit or to be discharged with close follow-up. Without adequate information and arrangements for appropriate follow-up, patients may have to be admitted. Even with astute triage, successful discharge directly from the ED depends heavily on the immediacy, thoroughness, and ongoing access to HF management post-discharge, for which personnel resources vary widely. Although the presence of high-risk conditions may impact level of hospital care in admission decisions, national guidelines and prior studies have not outlined definitive criteria for safe ED discharge (17,30,32,44).

Multiple models predicting risk of hospital mortality and early death or readmission after discharge incorporate factors assessed in the ED (22-24,47,49,56-75), including clinical factors at emergency triage such as obvious hemodynamic instability, hypoxia, and oliguria; concomitant events such as acute coronary syndromes, stroke, and sepsis; and self-care (43,47,53). Physical examination findings notable in the ED include blood pressure and a third heart sound (31,49,60). The natriuretic peptides have been most extensively studied in the ED, often using point-of-care assays (24,55,63,68), and many other routine laboratory results and biomarkers at presentation have been correlated with risk (51,55,56,61-64,68,69,71-73). Several models address early mortality based on admission characteristics (22,49,52,57-59,67).

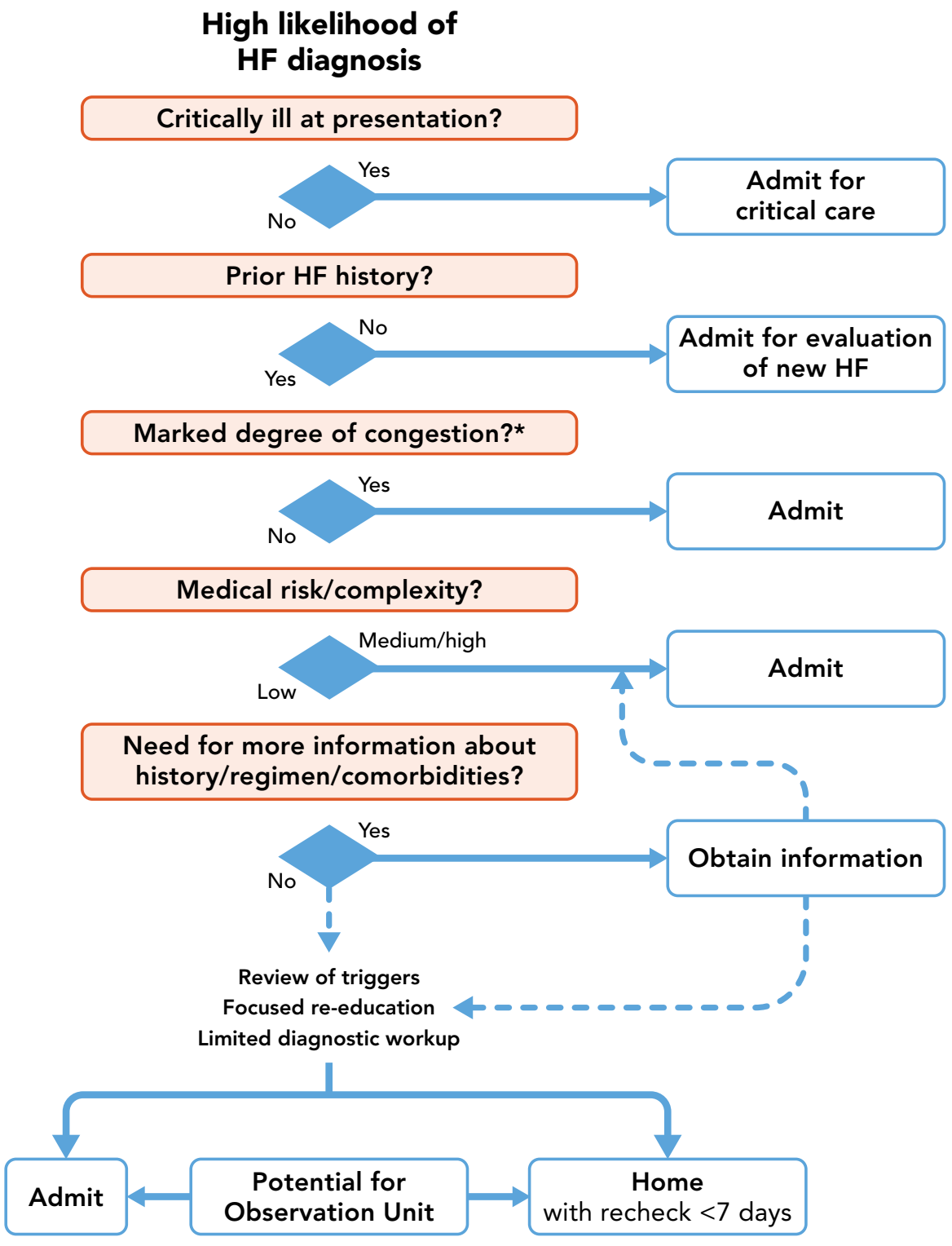
Patients recently discharged from the hospital often pass again through the ED. Careful documentation of optimized status, clear plans for rescue dose diuretics and the triggers to use them, and overall goals of therapy may enhance not only the efficacy of outpatient management, but also the efficiency of triage if patients nonetheless return to the ED. This is further discussed in Section 9 on discharge and the Focused Discharge Handoff (Section 10), a 1-page document that could provide this information in an actionable form at the time of emergency care.

Early therapy for acute HF is crucial even if patients are ultimately admitted. Medical therapy is discussed in Sections 7.2, 7.4, 7.5, and 7.6. Diuretic dosing for decongestion is considered in detail in Section 7.2.

6.2. Comprehensive Initial Assessment—Setting the Inpatient Goals

The integrated plan for the hospitalization should be developed as soon as the multidisciplinary care team can assemble to review the relevant data. The plan should incorporate the big picture of long-term disease trajectory and factors at admission that could be favorably modified by interventions in the hospital. In addition to the attending physician and the inpatient nurse, the team ideally would include a pharmacist and discharge coordinator and/or advanced practice nurse guiding patient education and addressing obstacles to successful transition to outpatient care. Current trends of acceleration in patient and staff flow increase the need for formal coordination of the initial plan, often on the morning after

FIGURE 2 Risk Stratification Algorithm for Emergency Department Patients With Acute Heart Failure



*Marked leg edema, ascites, or scrotal or perineal edema may be clinical signs of marked congestion. The degree of radiographic and biochemical abnormalities may also indicate the degree of congestion. ED = emergency department; HF = heart failure.

TABLE 1 Predictors of Risk in Emergency Care Studies Evaluating Patients With Acute Heart Failure

■ **Immediate risk** (measures of acute severity) (45)
Hypoxia, shock/hypoperfusion, respiratory distress, anuria, and acute and worsening condition (sepsis, stroke, acute coronary syndrome, hemodynamically significant arrhythmia)
■ **Intermediate risk** (predictors of events through 30 days) (45–56)
■ New-onset HF
■ Low BP without shock or hypoperfusion
■ Tachycardia
■ Kidney dysfunction
■ Hyponatremia
■ Elevated cardiac troponin without ACS
■ Degree of BNP elevation
■ Liver dysfunction
■ **Lower risk** (45–56)
■ Normal BP and HR
■ Brisk response to initial intravenous diuretic with diuresis and symptom relief
■ Rapid resolution of symptoms in the ED
■ Normal kidney and liver function without recent decline
■ Normal BNP and cardiac troponin

ACS = acute coronary syndrome; BP = blood pressure; BNP = B-type natriuretic peptide; ED = emergency department; HR = heart rate.

admission, by which time further details of the history and medication reconciliation have often emerged.

The two central themes of care for patients hospitalized for decompensated HF are decongestion and optimization of the therapies recommended for HF, but multiple other goals also need to be met. The coordinated care plan includes evaluation as necessary of the primary etiology of the heart disease and potential aggravating factors that would require specific intervention, both cardiac and noncardiac (34) (see **Table 2**). Careful evaluation should continue even after one trigger has already been recognized. Decompensation should not be too quickly ascribed to nonadherence, as most patients describe occasional lapses in salt restriction and medication schedules; this likely also occurs in patients without HF decompensation. Over-attribution of nonadherence, in addition to missing alternative explanation of symptoms, may unfairly stigmatize patients, and this error may occur more commonly for patients with limited education, health literacy, and socioeconomic status.

TABLE 2 Common Factors That Can Contribute to Worsening Heart Failure

Acute myocardial ischemia
Uncontrolled hypertension
Atrial fibrillation and other arrhythmias
Nonadherence with medication regimen, sodium, or fluid restriction
Medications with negative inotropic effect
Medications that increase sodium retention (NSAIDs, thiazolidinediones, steroids)
Excessive alcohol intake or illicit drug use
Anemia
Hyper or hypothyroidism
Acute infections (upper respiratory infection, pneumonia, urinary tract infections)
Additional acute cardiovascular diagnoses (aortic valve disease, endocarditis, myopericarditis)

Adapted from Yancy, et al. 2013 ACCF/AHA Guideline for the Management of Heart Failure (15).

ACCF = American College of Cardiology Foundation; AHA = American Heart Association; NSAIDs = nonsteroidal anti-inflammatory drugs.

Gaps identified in patient understanding should focus on the teaching and reinforcement needed during hospitalization. Concerns for limitations of support in the home environment or recognized barriers to self-care should trigger social work or other appropriate consultation (37). Discovery at admission of nonadherence with previously prescribed medications identifies the need for motivational education regarding adherence (76). Consultation for physical therapy or nutrition, as needed, should be included in the formal plan and initiated early during hospitalization.

All members of the team should contribute to an initial assessment of the likely outcome both in hospital and after discharge. A profile conferring high risk from factors that do not appear modifiable should trigger early discussion with the patient and family regarding anticipated outcomes and their priorities for remaining quality and quantity of life. Regardless of prognosis, all patients admitted to the hospital should have a designated surrogate decision maker, ideally identified in the outpatient setting and documented during admission. If not already done, however, this designation should be supervised by the inpatient care team.

Patients hospitalized with an HF diagnosis often carry a greater burden of complex medical problems than in the past, some relating to chronic comorbidities (see Section 6.2.2, Consideration of Comorbidities). However, other active problems may drive the admitting diagnosis and care team. The goals outlined for in-hospital treatment of decompensated HF should be applicable whether HF is the primary admitting or a secondary diagnosis, although the appropriate staffing models for comanagement of HF on other services have not been established.

6.2.1. Assessing Hemodynamic Profiles

Most patients present with at least 1 symptom and 1 sign of congestion that can be tracked as targets during decongestion and may serve as sentinel symptoms for recurrent congestion after discharge (70,74,77,78) (**Table 3**). The jugular venous pressure (JVP) reflects elevated right-sided filling pressures and is also a sensitive indicator of elevated left-sided filling pressures in patients with HF (75,79). Rales, when present, usually indicate higher filling pressures than baseline, but are often absent in chronic HF due to pulmonary lymphatic compensation. Extensive pitting edema, ascites, or large pleural effusions reflect large extravascular reservoirs that may take many days to mobilize.

TABLE 3 Clinical Evidence of Congestion

Symptoms
Orthopnea
Dyspnea on minimal exertion
Paroxysmal nocturnal dyspnea
Nocturnal cough*
Bendopnea
Abdominal swelling
Early satiety
Anorexia, nausea
Right upper quadrant pain
Peripheral swelling
Rapid weight gain
Signs†
Elevated jugular venous pressure
Rales‡
Pleural effusion‡
Increased intensity of pulmonary component of second sound
Third heart sound
Murmurs of mitral and/or tricuspid regurgitation
Pulsatile hepatomegaly
Ascites§
Pre-sacral, scrotal, or peroneal edema
Peripheral edema

*Often when supine.
†JVP is the most sensitive sign. Rales may not always be present.
‡Not common in chronic HF.
§May be difficult to distinguish from central adiposity.
HF = heart failure; JVP = jugular venous pressure.

Clinical profiles of patients with HF are shown in Figure 3. Patients identified with congestion should be further considered for whether filling pressures are elevated in proportion for both the right heart and the left heart (right atrial pressure >10 mm Hg and pulmonary capillary wedge pressure >22 mm Hg; 75% to 80% of patients with chronic HFrEF, less defined for HFpEF) (42,80). The wet and warm clinical profile without evidence of hypoperfusion characterizes over 80% of patients admitted with reduced EF and almost all with preserved EF except those with small left ventricular cavities of restrictive or hypertrophic cardiomyopathies (39,42). The cold and wet profile describes congestion accompanied by clinical evidence of hypoperfusion, as suspected from narrow pulse pressure, cool extremities, oliguria, reduced alertness, and often recent intolerance to neurohormonal inhibition. Sleepiness, impaired concentration, and very low urine output may also be present. These patients may require adjunctive therapy with vasodilator or inotropic agents or decrease of medications with negative inotropic effects to improve cardiac output and facilitate diuresis. Patients who appear to have low cardiac output without clinical congestion (cold and dry profile) often have unrecognized elevation of filling pressures, which may be revealed by invasive hemodynamic measurement. Uncertainty regarding hemodynamic status is associated with worse outcomes and is an indication for invasive hemodynamic assessment (15,81). True hypoperfusion without elevated filling pressures accounts for fewer than 5% of admitted patients (39) and usually reflects aggressive prior therapy with tight adherence. A patient hospitalized with apparent decompensation in whom **both** filling pressures and perfusion appear to be normal

FIGURE 3 Classification of Patients Presenting With Acutely Decompensated Heart Failure

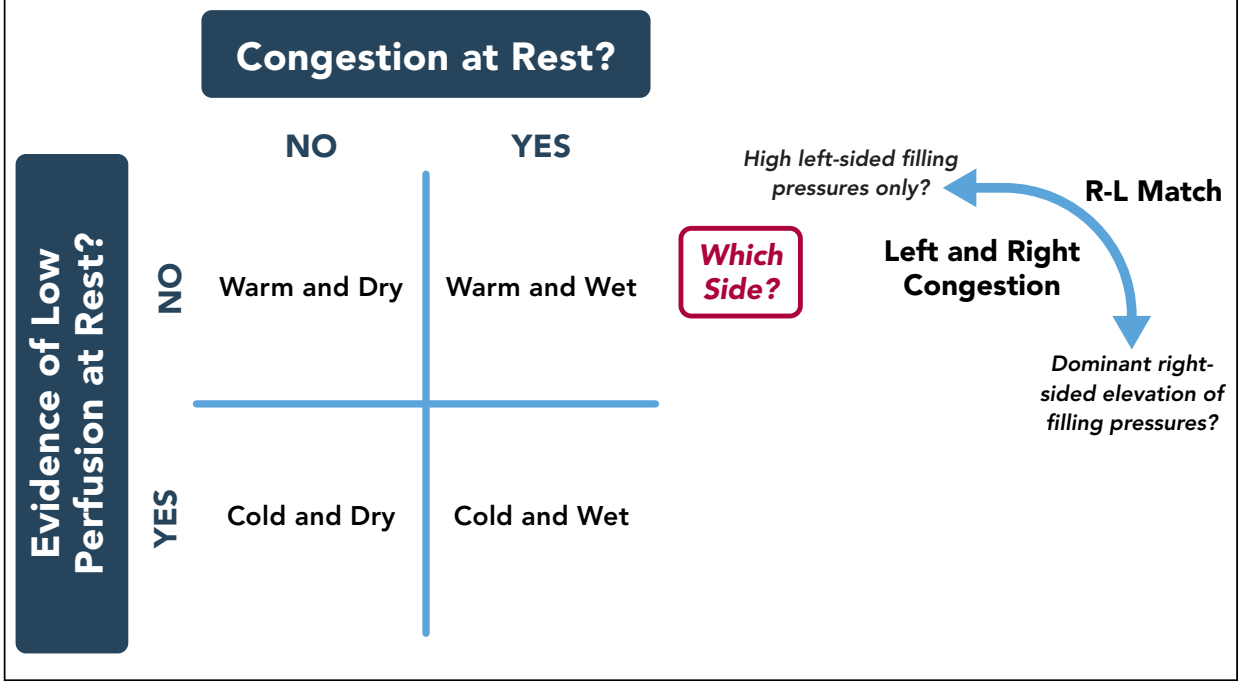


TABLE 4 Key Comorbid Conditions to Consider

Comorbidity	Management	Relevant Guidelines/Pathways
Cardiovascular		
Coronary artery disease/acute coronary syndrome	Assess and treat ischemia, and consider revascularization.	2014 AHA/ACC Guideline for the Management of Patients With Non–ST-Elevation Acute Coronary Syndromes 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction
Atrial fibrillation/ flutter	Achieve optimal rate control. Consider restoration of normal sinus rhythm. Anticoagulation as warranted.	2014 ACC/AHA/HRS Guideline for the Management of Patients with Atrial Fibrillation 2019 AHA/ACC/HRS Focused Update of the 2014 AHA/ACC/HRS Guideline for the Management of Patients With Atrial Fibrillation
Cerebrovascular disease, TIA/stroke	Treat according to current guidelines.	Guidelines for the Prevention of Stroke in Patients With Stroke and Transient Ischemic Attack
Peripheral vascular disease	Treat according to current guidelines.	2016 AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease
Aortic stenosis	Treat according to current guidelines.	2017 AHA/ACC Focused Update of the 2014 AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease
Mitral regurgitation	Refer to structural heart disease expert and treat according to current guidelines.	2017 AHA/ACC Focused Update of the 2014 AHA/ACC Guideline for the Management of Patients with Valvular Heart Disease
Hypertension	Optimize GDMT for HF for control of BP. consider IV vasodilators in addition to IV diuretics if hypertensive urgency or emergency. ACEI/ARB/ARNI/beta-blocker/aldosterone antagonists are first line in HFrEF. Avoid nondihydropyridine calcium channel blockers (CCB) and alpha blockers in HFrEF; dihydropyridine CCB are acceptable for BP control if on maximum evidence-based therapy.	2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/ PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults 2017 ACC/AHA/HFSA Focused Update of the 2013 ACCF/AHA Guideline for the Management of Heart Failure
Systemic Disease		
Diabetes mellitus	Monitor hyperglycemia throughout hospitalization, optimize therapy, avoid thiazolidinediones, consider metformin, SGLT2 inhibitors, follow current standards of care.	American Diabetes Association Standards of Medical Care in Diabetes– 2019 2018 ACC Expert Consensus Decision Pathway on Novel Therapies for Cardiovascular Risk Reduction in Patients With Type 2 Diabetes and Atherosclerotic Cardiovascular Disease
Chronic kidney disease	Evaluate etiology, avoid nephrotoxic agents. Can consider potassium binders to maximize neurohormonal blockade. Comanagement with nephrologist. Patients on dialysis are especially problematic.	KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease
Acute worsening of kidney function	Evaluate etiology, recognize that transient rise in creatinine with appropriate decongestion strategies or RAAS initiation is not usually associated with worse outcomes.	KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease
Liver disease	Evaluate for etiology and appropriate treatment strategies if primary liver disease. Note increasing prevalence of nonalcoholic fatty liver disease that may progress to nonalcoholic steatohepatitis.	The Diagnosis and Management of Nonalcoholic Fatty Liver Disease: Practice Guidance From the American Association for the Study of Liver Diseases
Acute exacerbation of chronic lung disease	Monitor oxygenation, optimize therapy, treat hypoxia, consider noninvasive ventilation.	Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease: 2019 Report
Infection	Diagnose and treat as needed.	
Sleep apnea	Facilitate diagnosis by sleep study to distinguish central from obstructive sleep apnea, initiate appropriate treatment.	Management of obstructive sleep apnea in adults: A clinical practice guideline from the American College of Physicians.
Anemia/iron deficiency	Evaluate and treat according to underlying etiology. Consider intravenous ferric carboxymaltose or nondextran IV iron intravenous iron replacement for improvement in symptoms and functional capacity, even if anemia is mild. Consider transfusion for severe and symptomatic anemia.	Treatment of anemia in patients with heart disease: a clinical practice guideline from the American College of Physicians.
Rheumatologic diseases	Treat according to current guidelines, recognize that some biological agents may have cardiotoxicity or adverse effects in HF patients.	2015 American College of Rheumatology Guideline for the Treatment of Rheumatoid Arthritis
Amyloidosis	Screen for cardiac or systemic amyloidosis with or without polyneuropathy, with genetic testing as appropriate; consider treatment for ATTR and AL.	Guidelines on the management of AL amyloidosis
Cancer	Assess for cardiac involvement or cardiotoxicity of chemotherapy or radiotherapy.	2016 ESC Position Paper on cancer treatments and cardiovascular toxicity developed under the auspices of the ESC Committee for Practice Guidelines: The Task Force for cancer treatments and cardiovascular toxicity of the European Society of Cardiology (ESC)
Thyroid	Gradually try to achieve euthyroid state.	Guidelines for the Treatment of Hypothyroidism 2016 American Thyroid Association Guidelines for Diagnosis and Management of Hyperthyroidism and Other Causes of Thyrotoxicosis

Continued on the next page

TABLE 4 Continued

Comorbidity	Management	Relevant Guidelines/Pathways
General Condition		
Obesity	Screen for diabetes and sleep apnea, educate on lifestyle modification. Referral to nutritionist. Consider gastric bypass surgery. Exercise program/cardiac rehabilitation.	Behavioral Weight Loss Interventions to Prevent Obesity-Related Morbidity and Mortality in Adults: US Preventive Services Task Force Recommendation Statement
Malnutrition	Assess for protein calorie malnutrition. Referral to dietician.	Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient
Frailty, deconditioning	Assess for frailty, consider physical therapy and/or referral for rehabilitation.	
Psychosocial		
Dementia /cognitive decline	Assess precipitating factors, possible delirium, evaluate cognitive and mental executive function.	Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology
Depression	Screen for depression and other mood disorders. Consider referral (87) for counseling and potential pharmacotherapy.	Nonpharmacologic Versus Pharmacologic Treatment of Adult Patients With Major Depressive Disorder: A Clinical Practice Guideline From the American College of Physicians
Substance abuse	Monitor and treat for cardiotoxicity and withdrawal, educate on cardiotoxicity, refer for substance abuse rehabilitation.	Health and Public Policy to Facilitate Effective Prevention and Treatment of Substance Use Disorders Involving Illicit and Prescription Drugs: An American College of Physicians Position Paper
Tobacco abuse	Smoking cessation counseling.	Behavioral and pharmacotherapy interventions for tobacco smoking cessation in adults, including pregnant women: U.S. Preventive Services Task Force recommendation statement 2018 ACC Expert Consensus Decision Pathway on Tobacco Cessation Treatment
Alcohol abuse	Monitor and treat for withdrawal, educate on cardiotoxicity, refer for rehabilitation.	Guidelines for biological treatment of substance use and related disorders, part 1: Alcoholism, first revision
Inadequate social support	Assess for self-neglect, barriers to care, ability and necessary support systems for self-care. Referral to social work.	
Nonadherence	Assess for reasons for nonadherence, including health illiteracy; address goals of care; provide education and support to overcome barriers.	**2017 ACC Expert Consensus Decision Pathway for Optimization of Heart Failure Treatment: Answers to 10 Pivotal Issues About Heart Failure With Reduced Ejection Fraction (Table 9, Table 10, Table 11)**

ACC = American College of Cardiology; ACEI = angiotensin-converting enzyme inhibitors; AHA = American Heart Association; AL = amyloid light chain; ARB = angiotensin receptor blockers; ATTR = amyloid transthyretin; GDMT = guideline-directed medical therapy; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; HTN = hypertension; RAAS = renin-angiotensin-aldosterone system; SGLT2 = sodium-glucose cotransporter-2; STEMI = ST-segment elevation myocardial infarction.

should be carefully evaluated for other causes of symptoms, such as transient ischemia or arrhythmias, or noncardiac diagnoses such as pulmonary disease.

6.2.2. Consideration of Comorbidities

A key component of the comprehensive initial assessment is evaluation of patient comorbidities (Table 4). These comorbidities and their therapies should be carefully considered for their role in HF decompensation and as independent targets for intervention. For example, diabetes mellitus and pulmonary disease are each present in 30% to 40% of patients hospitalized with HF and play a role in disease severity and risk for decompensation (82). Kidney dysfunction can precipitate congestion and can also limit initiation of GDMT. Frailty is another common comorbidity in HF, particularly for the elderly (83,84), and its association with health, functional status, and late-life disability is an increasingly important focus for patients with HF and their caregivers. Approximately 50% to 70% of older patients admitted with ADHF present with some degree of frailty, although this may be reversed or attenuated with interventions (85,86). Consideration should be given at the time of hospitalization to the need for physical therapy consultation.

6.2.3. Initial Risk Assessment

This document centers on evaluation of the clinical trajectory of HF, as an assessment of both daily clinical progress and the long-term disease course, incorporating the prior history with specific risk factors at admission, the day-by-day progress toward the goals of hospitalization, and the re-assessment before discharge. The risk factors that enter into this assessment may be fixed, as for age or number of previous hospitalizations, or potentially modifiable, as for natriuretic peptide levels. Much of the available data concerning risks in HF can be viewed through their impact on trajectory. Improvement in modifiable risk factors, such as clinical congestion, elevated natriuretic peptide levels, and inadequate recommended medical therapy, is associated with improved prognosis.

Multiple factors have been demonstrated to increase risk of HF mortality and rehospitalization (22-24,39,47,49,51,54-67,69,70,72,88-92). Many of these factors from registries and

TABLE 5 Assessing Risk During Hospitalization

Chronic History Prior to Admission

- Older Age (robust in all models)
- Number of Previous HF hospitalizations (41,92)
- Comorbidities, especially diabetes, COPD, liver disease, cancer, dementia (41,82)
- Frailty (54,85)
- Known low LVEF in HFrEF (39,41,91)
- RV dysfunction

Assessment at Admission	Reassessment at Discharge
Class IV symptoms (39,91,99)	***Effective decongestion improves prognosis.***
Nonadherence to medications or salt/fluid restriction (37,76)	***Focused education during hospitalization with increased home and community support may improve adherence*** (107,108).
Progressively higher risk with higher admission natriuretic peptide (NP) levels (24,51,55,63,68)	***Larger % reduction (>30%–60%) in NP levels associated with better outcomes*** (68,109–112). Progressively higher risk with higher discharge NP levels (51,113).
Renal dysfunction markers: ■ Elevated serum creatinine or low clearance (41,49)	Risk increased, but small increases in creatinine accompanying successful decongestion are associated with better prognosis (118–120).
■ Additional risk of high BUN (49,62)	High BUN at discharge increases risk (66).
■ Low spot urine sodium after first IV diuretic dose (114)	Low total urinary sodium excretion may be a more important marker than total urine output during hospitalization (121).
■ Diuretic resistance with high outpatient doses (115–117)	Diuretic resistance in-hospital associated with longer LOS and worse outcomes (66,77). High risk if discharged on high loop diuretic doses (66,115–117).
Degree of congestion at admission not predictive of outcome except longer length of stay with greater excess volume (39,67,70,104)	Residual congestion after treatment confers high risk (67,70,78,104,122). ■ High measured filling pressures (78) ■ Orthopnea (67,70,104) ■ Edema (67,70,104) ■ Composite congestion scores (67,104) ■ Lack of hemoconcentration (118,120,123)
Hemodynamic profile of "cold and wet" at admission (39,65,124)	Discharge with either cold or wet profile associated with higher risk (65,124).
Low systolic blood pressure (39,49,60,91,124)	Low systolic blood pressure at discharge also identifies high risk (65–67,124).
Troponin elevation (57,64)	Risk if elevated at any time during hospitalization.
Hyponatremia (39,41,61,91)	Lower sodium at discharge predicts higher risk (66).
Increased risk at admission if: ■ No RAS therapy (39,88) ■ No beta blocker therapy (66,88)	Discontinuation of ACEI/ARB in hospital for hypotension or kidney dysfunction is associated with poor outcomes (90). Unknown impact of reinitiation after discontinuation for circulatory and/or renal reasons. Discharge without RAS inhibition or discharge without beta-blocker associated with high risk (88,103,106).
Unexpected in-hospital events conferring additional risks	
■ Resuscitation or Intubation (66) ■ Intravenous inotropic therapy even if brief (125)	
Integrated Risk at Transition to Discharge = Admission Risk + In-Hospital Trajectory + Unexpected Events	

Modification of bolded/italicized items decreases risk. Note that the references for risk factors are provided as examples and are not meant to list all sources of validation.

ACEI = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; BUN = blood urea nitrogen; COPD = chronic obstructive pulmonary disease; eGFR = estimated glomerular filtration rate; HF = heart failure; IV = intravenous; LOS = length of stay; LV = left ventricular; NP = natriuretic peptide; RAS = renin-angiotensin system; RV = right ventricular.

trial databases are continuous variables, while those from administrative databases are often binary (i.e., history of kidney disease). Demographic variables and psychosocial determinants of health (93–97), physiological risk factors reflecting severity of cardiac disease or effectiveness of therapy, serum biomarkers that reflect cardiac and systemic stress and response, and the adequacy of the medication regimen are commonly implicated.

Specific risk factors that predict events across many models are advanced age, HF hospitalization history, decreased kidney function, high natriuretic peptide concentrations, and low blood pressure (58,91). Age is of central importance when integrating prognosis, as older age predicts higher mortality, particularly combined with higher number of previous HF hospitalizations (92). The combination of advanced age, multiple recent HF hospitalizations, and chronic kidney disease identifies a population whose long-term prognosis is particularly unfavorable.

Multiple risk scores validated in ambulatory populations with chronic HF provide significant discrimination between patients who are more and less likely to experience the combined endpoint of death or hospitalization (59,98,99). These models predict recurring hospitalization better than they predict death, for which calibration is weak (100). For hospitalized patients predicted by recent models to die during the current HF admission, more than 9 of 10 are discharged alive (101).

TABLE 6 Interventions for Patients at High Risk of Unfavorable Outcomes

Discussion of prognosis
Evaluation for *advanced therapies** if appropriate
Review/revision of goals of care and advanced directives
Consideration before *interventions*† that may be difficult to discontinue
Education regarding palliative care and hospice options

*Transplantation, mechanical circulatory support. †Intravenous inotropic therapy, temporary circulatory support, mechanical ventilation, dialysis.

To be useful, the identification of a high-risk status needs to be actionable (102). Integrated risk scores may help allocate limited hospital resources and may trigger and inform discussions regarding prognosis and appropriate goals of care. Often, however, the components of risk need to be parsed into their components to target more specific intervention. The assessments of risk described during hospitalization in this pathway document focus on those that can guide in-hospital management to improve outcomes after hospital discharge (9,33,103–106). For example, strategies to decrease readmission linked to nonadherence may not help patients approaching the end of life.

Because a key message of this document is the importance of serial assessment from admission through discharge, the risk factors listed in **Table 5** are categorized according to the time when they may be known during the hospitalization. In setting goals to decrease risk and improve outcomes after hospitalization and later, it may be helpful to focus on those risk factors most likely to be modifiable.

Risk factors known at the time of hospitalization include age, duration of HF, and frequency of hospitalization. Chronic risks that may be previously or newly recognized at the time of admission include right ventricular dysfunction, persistent Class IV symptoms, and nonadherence with medications and/or salt/fluid restrictions. Multiple correlates of chronic renal dysfunction and right ventricular dysfunction predict higher risk, but it is not clear how and whether these risks are modified by the interventions during hospitalization. Of the biomarkers measured clinically, natriuretic peptide levels are the most robust predictors of readmissions and death (24,55,63,68,111,112), and also highly modifiable with successful decongestion, after which levels continue to decrease for days after discharge. The magnitude of decrease in natriuretic peptide levels during therapy is closely associated with decreased risk, and increase or failure to decrease levels is associated with higher risk (109,110). Absolute levels at discharge are also highly predictive of rehospitalization, need for advanced therapies such as transplant or mechanical circulatory support, and mortality. There is increasing interest in urinary sodium concentration during intravenous diuretic therapy as a biomarker of better outcomes that relates closely to renal responsiveness, whether in a 24-hour collection or as the first spot urine after intravenous diuretic (114,126).

Troponin elevations at admission or during HF hospitalization, even in the absence of acute coronary syndromes, are associated with worse outcome but have not been integrated comprehensively into overall risk assessment (64). It is not clear whether risk is modified by changes that occur in troponin after admission. Elevated concentrations of other biomarkers, including ST2 (68,127), galectin 3 (68,73), copeptin, and forms of adrenomedullin (88,128) implicate pathways associated with more advanced disease and worse outcomes, but have not been validated as endpoints to guide specific interventions. Multimarker profiles may combine several aspects of risk (69,71).

Of central importance are the symptoms and signs of congestion, typically part of the presentation for admission. Once present, higher degrees of congestion are associated with more net volume loss and longer hospital stay before decongestion, but if decongestion can be achieved, the degree of initial congestion is not associated with higher risk (67,70,120). Either admission or discharge with the cold and wet profile is associated with worse outcomes (124). Frailty is unlikely to improve during hospitalization but may be favorably affected after discharge by improved clinical status, nutrition, and rehabilitation (83–86). Nonadherence identified at the time of admission predicts nonadherence and readmission after discharge (76,107), but some interventions have shown to decrease the risk of nonadherence (108).

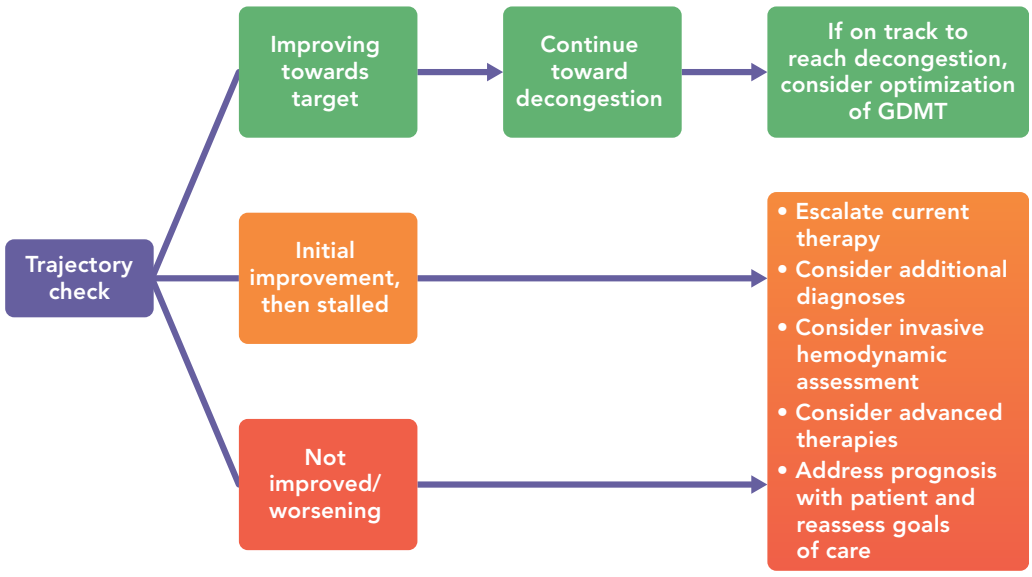
In this document, we describe risk assessment of patients at admission, daily review throughout the active phase of therapy, and review at the transition from intravenous to oral diuretics prior to the day of discharge. For calibration of risk during the hospitalization, the writing team felt it was important to standardize the nodes and goals of hospitalization as much as possible. In the final risk assessment prior to discharge, the progress and events from the hospital course, including unexpected need for resuscitation or intravenous inotropic therapy, are incorporated into the overall assessment to revise the long view of disease trajectory after discharge. At any time between admission and discharge, recognition of high risk for unfavorable outcomes should trigger specific considerations (**Table 6**), including caution regarding the initiation of therapies that may be difficult to discontinue.

6.2.4. Documentation

From admission through discharge, information should be systematically documented in a format easily accessible to clinicians both in and out of the hospital to optimize care and outcomes. Availability of that information is crucial for a patient who presents soon after discharge and is considered for readmission (**Figure 2**).

Several principles concerning collection and recording of information will be stressed throughout this document.

FIGURE 4 Clinical Trajectories and Their Implications for Therapy



GDMT = guideline-directed medical therapy.

- Collection and recording of information starts early (129) and continues throughout hospitalization. This should not be left exclusively to the day of discharge.
- Different members of the team should be able to record and also have access to information.
- Sending appropriate information from the inpatient team to other clinicians who interact with the patient outside of the hospital is crucial.
- Curating the information to include the most relevant data while at the same time streamlining the process of data entry to the degree possible is important.
- Standardized methods of recording and transmitting information (using apps, electronic health records, or paper forms, depending on setting and resources) may be helpful.

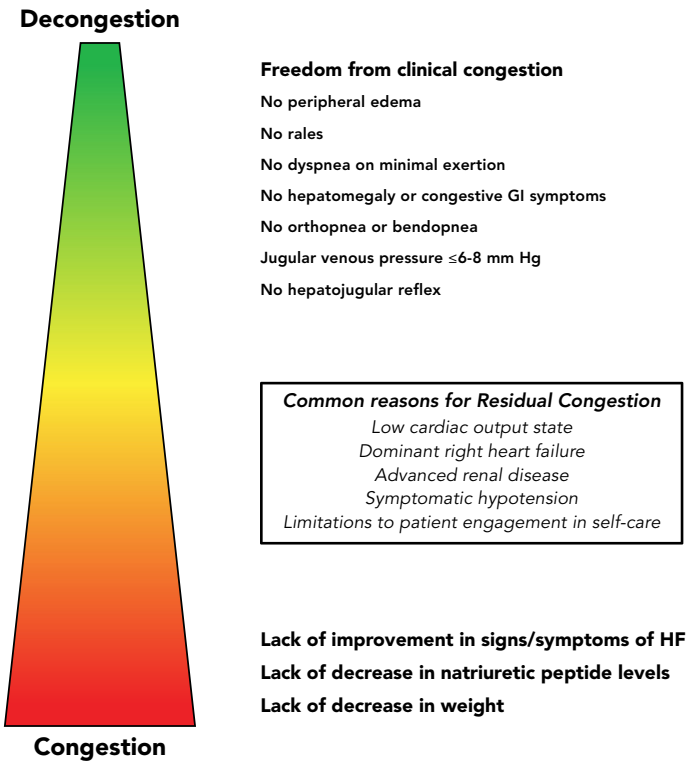
7. NODE: DAILY TRAJECTORY CHECK

The near-term ***clinical trajectory*** during hospitalization represents responsiveness to therapy in terms of clinical HF symptoms and signs and laboratory and diagnostic tests. This trajectory helps define the next steps for management, care coordination, health outcomes risk and prognosis, and disposition.

We have also highlighted a ***long-term trajectory assessment*** as a specific evaluation of progress toward resolution of symptoms and signs of congestion, adequacy of perfusion, stability of vital signs, and trends in kidney and other organ function (compass symbols in Figure 1). The concept of long-term trajectory stresses the importance of stepping back to gain perspective of not only where the patient stands, but in which direction the patient is headed, as a crucial component of determining whether to continue along the current course of therapy or to change direction. Usually, the clinical trajectory of a patient is assessed at least daily, but a clear understanding of the long-term trajectory is particularly important at points such as the day after admission or at the transition to the discharge regimen.

Three main in-hospital trajectories have been defined according to changes in patient symptoms, clinical signs, laboratory markers and imaging if done, presence or absence of complications, assessment and treatment of comorbidities, and treatment alignment with goals of care: 1) *improving towards target;* 2) *stalled after initial response;* or 3) *not improved/worsening*. These trajectories translate into different management strategies throughout the hospitalization and post-discharge (Figure 4). Patients improving toward target should be considered for initiation and/or further optimization of GDMT. In those who are not improved/worsening, therapy should be intensified, and additional diagnoses, including conditions other than HF, should be considered. If deterioration continues, hemodynamic assessment and advanced therapies merit consideration. Further deterioration should prompt discussion about prognosis and goals of care. Patients who are *stalled* represent those whose symptoms may have improved

FIGURE 5 Evaluation of the Degree of Clinical Congestion, With Common Reasons for Residual Congestion Listed in the Text Box



GI = gastrointestinal; HF = heart failure.

initially but in whom residual congestion remains and diuretic resistance and/or kidney function, or other problems, may be limiting progress. The key issue in such patients is whether escalation of therapy will suffice to bring about complete decongestion, or whether that target needs to be modified, allowing a "compromise with congestion."

7.1. Targets for Decongestion

Inpatient trajectories are primarily defined by the pace and extent of decongestion. Evaluation of the degree of clinical congestion is depicted in **Figure 5**. The usual goal is for complete decongestion, with absence of signs and clinical symptoms of elevated resting filling pressures (70,78,117,130). Rates of rehospitalization and death are consistently lower in patients rendered free of clinical congestion by the time of discharge (67,78). National Heart, Lung, and Blood Institute-sponsored trials of ADHF have specified goals of resolution of edema, orthopnea, and jugular venous distention (74,77,78,131,132). JVP should generally be reduced to <8 cm, dyspnea at rest should be relieved, and there should be no residual orthopnea, bendopnea, or edema (77,78). Peripheral reservoirs of anasarca, large pleural effusions, and ascites as detectable should gradually be depleted, after which intravascular filling pressures as indicated by JVP will more rapidly decrease. The amount of net diuresis that will be needed for complete decongestion cannot be ascertained at the time of admission, and the difference between admission weight and a previous target weight often underestimates the excess fluid. Postural hypotension is often interpreted as indication of overdiuresis, but frequently reflects overvasodilation.

Most patients report early improvement in symptoms, particularly dyspnea. In admissions with HFrEF, shortness of breath was reported as the worst symptom by about one-half of patients, fatigue by about one-third, and abdominal discomfort, swelling, or edema by the remaining patients. The magnitude of patient-reported improvement was least for patients with a worst symptom of fatigue. Symptoms of congestion (**Table 3**) usually improve before the signs of congestion have fully resolved. If guided only by symptom relief, diuresis will often be stopped too soon. Before discharge, the clinical signs of congestion (**Table 3**) have usually resolved in 50% to 70% of patients (6,77,78,121,133-139). Evidence of improvement in filling pressures is closely associated with the improvement in breathing early during

hospitalization (140) and is consistently associated with better outcomes (67,109,113,115,116,118-120). Reports differ about the association between weight loss and symptom improvement (122,137,139). Early relief of symptoms correlated with both fluid loss and weight loss in the Heart Failure Network trials, but there was poor correlation between amount of weight loss and symptom improvement (122,137,139,141,142), and weight loss alone is not associated with better outcomes (141,142) likely because of the disparity between urine sodium output and fluid output (126) and the variable amount of fluid accumulation prior to admission. Average weight loss in recent inpatient HF trials ranges from 4 to 8 kg (74,77).

Substantial reduction in B-type natriuretic peptide levels is anticipated during effective diuresis, frequently decreasing by 50% or more from admission (111), and a decrease in natriuretic peptide concentrations of at least 30% before discharge is strongly associated with better outcomes. Targeting reduction in natriuretic peptide concentrations, however, did not result in better outcomes than treating congestion and optimizing other guideline-directed medical therapy empirically (111,112). Kidney function is not a reliable biomarker for volume status or change in volume status; modest increases are not linked to worse outcomes as long as the rise in creatinine is transient (143,144) and accompanied with successful decongestion (118-120,123), or occurs after initiation of renin-angiotensin system (RAS) or aldosterone antagonists (145-147).

The targets for decongestion may need modification for mismatch of right and left and right-sided filling pressures (**Figure 3**, right side). Approximately 70% to 75% of patients with decompensated chronic HFrEF have concordance of relative right and left filling pressures around thresholds of right atrial pressure of 10 mm Hg and pulmonary capillary wedge pressure of 22 mm Hg (75,80). Clinical assessment can be helpful to confirm or challenge concordance (42), but clinical evidence for elevated left-sided pressures may be subtle in the presence of prominent right-sided findings. Information from recent invasive studies or echocardiographic hemodynamic evaluation should be brought forward to inform the hemodynamic targets. Patients in whom elevated right atrial pressures approach or exceed left-sided filling pressures often cannot undergo diuresis to a normal JVP and may be more likely to receive inotropic support (79). Conversely, patients with elevated left-heart pressures in the presence of normal right-sided pressures may continue to have orthopnea and dyspnea on minimal exertion despite diuresis to JVP in the normal range; their optimal right-sided pressures may be in the lower range of normal.

The goal of edema resolution may need to be relaxed for patients with other known contributors to peripheral edema such as chronic venous insufficiency. However, persistent net diuresis in combination with light compression, such as with elastic sport bandages, can lead to marked improvement in peripheral edema even after years of chronic swelling was presumed refractory or attributed to lymphedema. The goal to eliminate edema must also be revised in the setting of low plasma oncotic pressure, often seen in the elderly with poor nutrition. Measurement of serum albumin should be routine during HF admission to gauge mobility of edema and also to target malnutrition.

7.2. Diuretic and Adjunctive Therapy

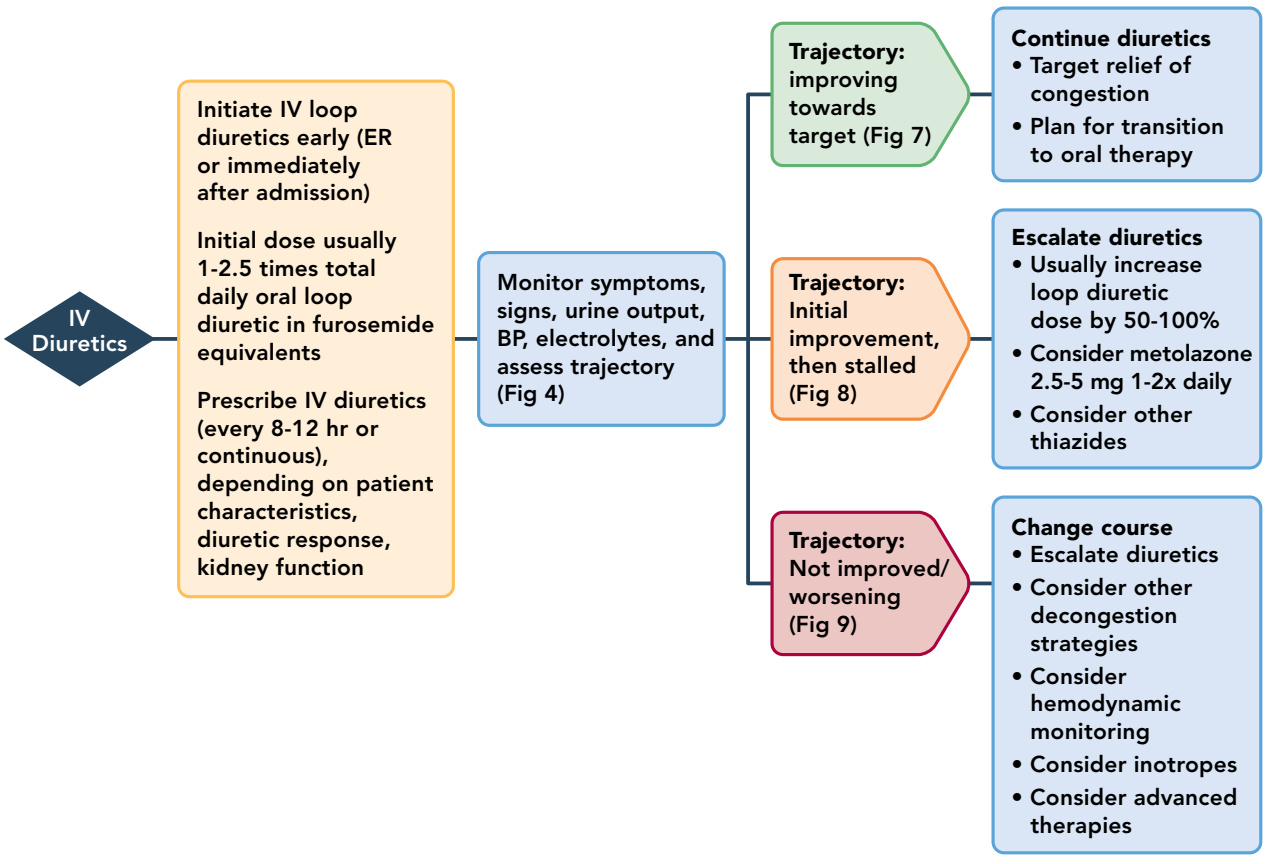
Establishing an effective diuretic regimen is crucial for achieving decongestion. Usually patients require the first dose of intravenous (IV) diuretics at presentation or in the ED, and IV diuretics are continued throughout the hospitalization until effective decongestion warrants transition to oral diuretics before discharge. On admission, for patients who have been on loop diuretic therapy as an outpatient, the total daily dose should be changed to an

TABLE 7 Diuretic Dosing

Class	Drug	Usual Inpatient Dosing* (Maximum†)	Usual Outpatient Dosing (Maximum†)
Loop diuretics	Bumetanide	0.5–4 mg/hour IV once to 3 times daily (5 mg/dose) *Or* 0.5–2 mg/hour IV infusion (4 mg/hour)	0.5–2 mg orally once to twice daily (10 mg/day)
	Furosemide	40–160 mg IV once to 3 times daily (200 mg/dose) *Or* 5–20 mg/hour IV infusion (40 mg/hour)	20–80 mg orally once to twice daily (600 mg/day)
	Torsemide	N/A‡	10–40 mg orally once daily (200 mg/day)
Thiazide-type diuretics	Chlorothiazide	0.5–1 g IV once to twice daily (2 g/day)	N/A
	Hydrochlorothiazide	25–50 mg orally once to twice daily (100 mg/day)	25–50 mg orally once daily (100 mg/day)
	Chlorthalidone	12.5–25 mg orally once to twice daily (100 mg/day)	25–50 mg orally once daily (100 mg/day)
	Metolazone	2.5–5 mg orally once to twice daily (20 mg/day)	2.5–5 mg orally once daily (20 mg/day)

*For patients receiving loop diuretics prior to admission, the oral dose should be changed to an intravenous dose of 1–2.5 times the home dose. For patients naïve to therapy, the lower end of the dosing interval should be used.
†"Usual" dose ranges reflect approved product labeling and safety and efficacy results from large, randomized controlled trials. Higher ranges may be considered on the basis of observational data and clinical experience.
‡Torsemide is not available as an intravenous formulation in the United States; oral therapy may be initiated prior to discharge to assess patient response.
IV = intravenous; N/A = not applicable.

FIGURE 6 **Diuretic Therapy in Different Clinical Trajectories**

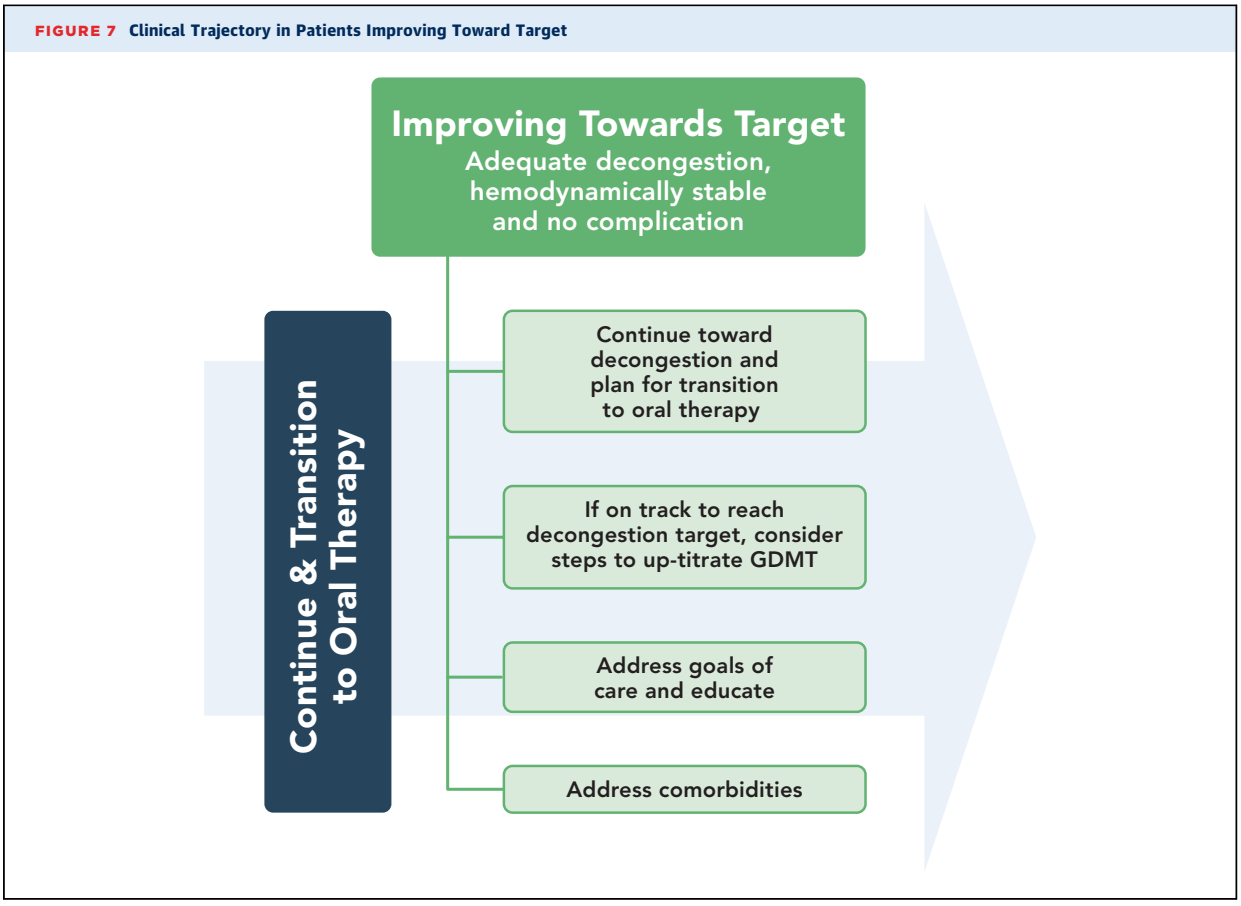


BP = blood pressure; ER = emergency room; IV = intravenous.

oral furosemide equivalent and administered IV at 1 to 2.5 times the total daily dose (e.g., an outpatient bumetanide dose of 1 mg twice daily would convert to 80 mg daily furosemide equivalent, and the IV dose would be furosemide 80 to 200 mg IV daily). For those patients who have not been on diuretics as an outpatient, initial furosemide dose can vary according to patients' fluid overload, kidney function, and age, and usually is around 40 to 80 mg IV daily dose. IV diuretics are usually continued throughout the early hospital stay either by IV bolus every 8 to 12 hours or by continuous IV infusion. Doses of various diuretics are shown in **Table 7**. Acetazolamide 250 to 500 mg/day may also be considered in refractory patients (148). The escalation or modification of the diuretic dose depends on the diuretic response, decongestion target, kidney function, and other patient-related factors such as hemodynamic factors, comorbidities, and serum electrolytes.

If the patient is improving at the expected pace, IV diuretics should generally be continued until optimal decongestion is achieved, and then transitioned to the oral dose estimated for maintenance. If initial improvement is stalled, if there is inadequate improvement, or if the patient is worsening and the patient is still congested, IV diuretics should be increased. Usually, the IV loop diuretic dose can be increased by 50% to 100% until the total diuretic dose exceeds 400 to 500 mg of furosemide equivalent total daily dose. The doses should be increased until a response is apparent. When the response is brisk but transient, the frequency should be increased to 3 or 4 times daily. The DOSE (Diuretic Optimization Strategies Evaluation) trial did not demonstrate any improvement with continuous infusion of IV furosemide, but these patients were also less likely to require dose increases or the addition of a thiazide-type diuretic; additionally, those patients with chronic furosemide equivalents of over 240 mg daily were excluded (77). Consequently, some patients have been observed to respond better to continuous infusion. When high furosemide doses are not effective, metolazone can be added at 2.5 to 5 mg doses once or twice daily. Other thiazide diuretics can be added

FIGURE 7 Clinical Trajectory in Patients Improving Toward Target



to loop diuretics and given intravenously if needed (**Figure 6**).

During hospitalization, electrolytes should be measured at least daily and corrected. Similarly, daily weights, patient intake and urine output, kidney function by measurement of serum creatinine, and BUN should be monitored. Serum creatinine commonly rises slightly during effective decongestion (143,144), but generally returns to baseline early after discharge, and is not associated with worse outcomes (118–120,123).

For patients with volume overload refractory to diuretics, extracorporeal ultrafiltration or hemodialysis can be considered. Although these strategies remove fluid effectively and can improve serum sodium, trials did not show improved clinical outcomes or kidney function (74,139). Ultrafiltration remains an option for patients not responding to intensified medical management.

Intravenous vasodilators (e.g., nitroglycerin, nitroprusside) represent another strategy in patients with refractory congestive symptoms. When added to diuretic therapy, IV vasodilators improve symptoms and hemodynamic evidence of congestion, but have not been associated with reductions in length of stay or mortality. Vasodilators may be particularly helpful in patients with symptomatic crashing acute pulmonary edema (SCAPE), a subset of patients with elevated systolic blood pressure requiring urgent treatment (149). Some evidence suggests that patients with HFpEF may respond differently to IV vasodilators than those with HFrEF (150,151); indeed, recent studies containing a broader population of patients with acute HF have not been associated with improved clinical outcomes (134,152). In the absence of elevated blood pressure, IV vasodilators should generally be initiated at low doses and titrated every 5 to 10 minutes as tolerated.

Similar hemodynamic effects can be achieved with the combination of long-acting oral nitrates and hydralazine (153), a strategy that may be used to limit the duration of IV vasodilators and permit the initiation of GDMT (154). Although combined nitrate and hydralazine therapy is often substituted for RAS inhibition in patients with persistent severe kidney dysfunction, there is no evidence for this indication in acute HF, nor are there data concerning whether and how patients should be transitioned back to RAS inhibition after discharge. If kidney function improves on this strategy, reinitiation could be considered in the hospital where it can be closely monitored, but not within 24 hours of discharge due to insufficient time to assess stability of kidney function and potassium handling.

7.3. TRAJECTORY: Improving Towards Target

In this trajectory (**Figure 7**), the patient has stable vital signs and is making steady progress toward resolution of signs and symptoms of congestion (**Table 3**), without major complications, and with appropriate alignment of management strategies with goals of care. Some patients, especially those with a clear trigger that has been reversed, such as rapid atrial fibrillation, may have prompt improvement. Such patients may differ from those with chronic myocardial dysfunction.

Net fluid loss and weight loss are expected with intravenous diuretics, usually at least 1 kilogram of weight loss per day (77,78,137,139). However, this response can vary depending on the doses of diuretics, baseline kidney function, diuretic resistance, and comorbidities (77,139,155–157). Diuretic doses should be titrated as necessary (**Figure 6, Table 7**). Patients with obvious large volume reservoirs such as anasarca sometimes lose several kilograms of weight daily, depending on the speed of volume redistribution into the intravascular space. In these patients, fluctuating fluid shifts from the periphery into the intravascular compartment may cause delay or fluctuation in reduction of symptoms and JVP.

When sufficient progress has occurred to render it likely that targets of decongestion will be reached, it is usually appropriate to initiate or up-titrate components of the GDMT regimen (see Sections 7.4 and 8.3). Throughout the hospitalization, it is important to tailor education to the patient's needs and to continue to address and manage comorbidities.

7.4. Optimization of GDMT

Neurohormonal antagonists have dramatically improved outcomes for HFrEF. When possible, continuation of GDMT through hospitalization or initiation before discharge is associated with substantially better outcomes, both due to the benefit of the therapies and to the better prognostic profile of patients who can tolerate them (106). Expert advice concerning initiation of GDMT for chronic HF can be found in the 2017 ACC Expert Consensus Decision Pathway for Optimization of Heart Failure Treatment (13).

Most studies demonstrating safety and efficacy of GDMT, however, have enrolled stable patients, and specifically excluded those with a recent decompensation (with the exception of the early use of angiotensin-converting enzyme inhibitors [ACEIs] in CONSENSUS and beta blockers in COPERNICUS) (158,159). Hospitalization provides a pivotal opportunity to decrease risk and improve clinical trajectory in patients who respond well to diuresis and who have not previously received adequate trials of GDMT. This therapy modifies and frequently reverses disease progression (9). The introduction of GDMT during hospitalization for HF with reduced ejection fraction is thus a key target to reduce risk (9,160). This has been shown for ACEI, beta blockers, and most recently supported for angiotensin receptor-neprilysin inhibitor (ARNI). Patients with good early response to diuresis should be considered for addition of recommended therapies or up-titration toward trial targets for neurohormonal antagonist therapy as decongestion is approached, recognizing that the diuretic response may diminish acutely with increasing neurohormonal antagonism, particularly if blood pressure is lowered.

Most studies of GDMT have investigated the addition or titration of a single agent to stable background therapy. There are no bases of evidence for patients in whom beta blockers or ACEIs/angiotensin receptor blockers (ARBs) were decreased or discontinued during hospitalization, most commonly for hypotension, progressive kidney dysfunction, or use of intravenous inotropic therapy (161). The 2017 ACCF/AHA guidelines emphasize that "caution should be used when initiating beta blockers in patients who have required inotropes during their hospital course or when initiating ACEIs, ARBs or aldosterone antagonists in those patients who have experienced marked azotemia or are at risk for hyperkalemia" (26). For these reasons, expectations regarding the prescription and dosing of GDMT in patients with ADHF are more conservative and individualized than for stable patients in outpatient HF management. For patients with HFpEF, beyond diuretics, clinical trial evidence that medical therapy improves outcomes is limited, but it seems reasonable to titrate RAS inhibitors to desired blood pressures in hospital.

One important common principle is to start at a low dose and titrate slowly upward as tolerated (Table 1 in Yancy et al. [13]). High starting doses and/or overly aggressive titration can result in hypotension and worsening kidney function, setbacks that limit both decongestion and initiation of different components of GDMT.

7.4.1. RAS Therapy

RAS inhibition is part of GDMT for patients with HFrEF, and should be continued or initiated in the absence of hypotension or unstable kidney function. If prior therapy was held during hospitalization, lower doses may be required when therapy is resumed. Transition through a short-acting agent such as captopril is rarely necessary, although it may be better tolerated in some patients with advanced HF (105,154,162,163). RAS inhibition can decrease blood pressure in patients with pre-existing intense neurohormonal activation, so particular care should be taken in patients recently weaned from intravenous inotropic therapy or in those diuresed extensively prior to its initiation. Caution should be exerted also in patients with acute kidney injury or hyperkalemia (143). Discharge information to the outpatient clinician should

TABLE 8 Eligibility and Initial Dosing for the PIONEER-HF Trial

Eligible Patients	Trial Exclusions	Dosing
HFrEF (EF ≤40%)	ACS, stroke, or revascularization within 1 month	*Initial dose*
NT-proBNP ≥1,600 pg/mL or BNP ≥400 pg/mL	Planned revascularization within 6 months	SBP 100–120 mm Hg: sacubitril/valsartan 24/26 mg twice daily
>24 hours and <10 days after initial HF hospitalization and still in hospital	Cardiac resynchronization within past 3 months or planned	SBP ≥120 mm Hg: sacubitril/valsartan 49/51 mg twice daily
Hemodynamically stable: SBP ≥100 mm Hg for at least 6 hours	eGFR <30 mL/min/1.73 m^2	*Dose adjusted after discharge every 1–2 weeks according to SBP*
No increase in diuretic or vasodilator dose for at least 6 hours	Potassium >5.2 mEq/L	
No intravenous inotropes for 24 hours	Hepatic failure with bilirubin >3 mg/dL	

ACS = acute coronary syndrome; eGFR = estimated glomerular filtration rate; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; NT-proBNP = N-terminal pro-B-type natriuretic peptide; SBP = systolic blood pressure.

include a reminder to consider reinitiation of neurohormonal therapies stopped in the hospital.

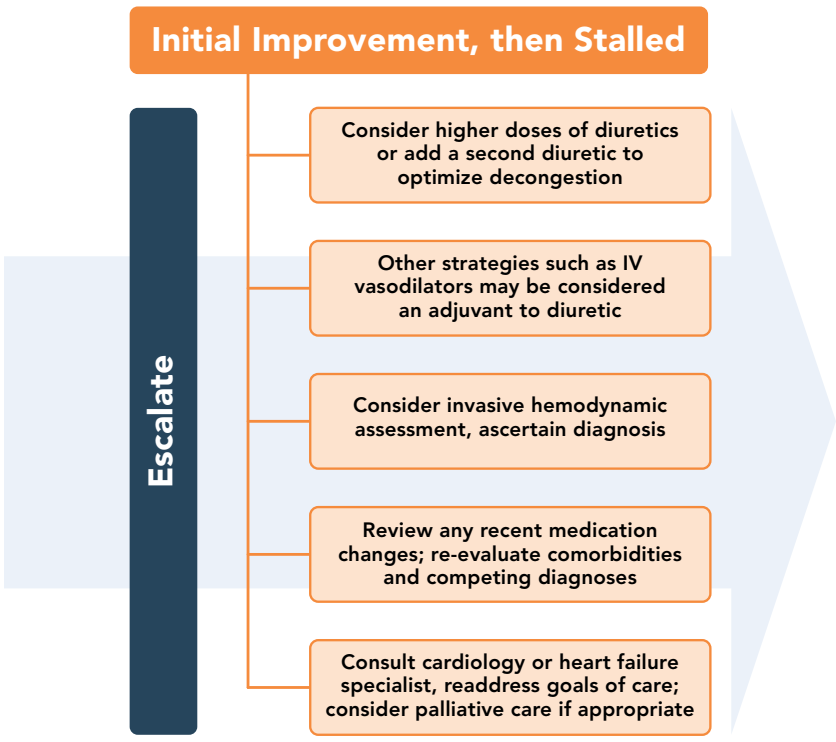
7.4.2. Angiotensin Receptor–Neprilysin Inhibitors

Current recommendations include ACEI, ARB, and ARNI as approved inhibitors of the RAS in chronic HF (164). Although there is extensive experience with initiation of ACEI and ARB as recommended therapies for hospitalized HF, the pivotal PARADIGM-HF (Prospective Comparison of ARNI with ACEI to Determine Impact on Global Mortality and Morbidity in Heart Failure) trial for ARNI focused exclusively on stable chronic HF, and excluded patients recovering from acute decompensated HF; this trial also included a run-in period with enalapril (165). The PIONEER-HF (Comparison of Sacubitril-Valsartan versus Enalapril on Effect on NT-proBNP in Patients Stabilized from an Acute Heart Failure Episode) trial now provides evidence to support safety of careful initiation of sacubitril-valsartan for hospitalized patients with and without prior exposure to ACEI or ARB, selected for hemodynamic stability, with systolic blood pressure ≥100 mm Hg and without escalation of intravenous diuretics or vasodilators for 6 hours, and without intravenous inotropic therapy within the previous 24 hours. (166). Compared with patients started on enalapril, patients randomized to sacubitril/valsartan 24/26 mg twice daily (or 49/51 mg for SBP ≥120 mm Hg) had more reduction in N-terminal pro-B-type natriuretic peptide (BNP) levels and in the exploratory clinical outcome of HF rehospitalization, similar to previous results in the outpatient setting (167). At week 1, more subjects treated with ARNI than ACEI had systolic blood pressure <100 mm Hg (22% vs. 13%, respectively), without differences in reported symptoms of hypotension (15% vs. 12.7%). These data suggest that consideration of initiation of ARNI during the hospitalization is warranted, either in the Trajectory phase in patients who have stabilized after initial diuresis, or in the Transition period. **Table 8** outlines eligibility and initial dosing for the PIONEER-HF trial, which includes patients with more advanced disease than in PARADIGM-HF but still excludes patients with recent hypotension or marked kidney dysfunction. Given this, the similar rate of adverse events in the 2 trials may reflect the lower initial dosing in PIONEER-HF. As in the outpatient setting, patients need to be off ACEI therapy for 36 hours before starting ARNI therapy to decrease the risk of angioedema. Diuretic dosing may need to be adjusted after ARNI, as diuretic requirements sometimes decrease, but anticipatory reductions are not recommended (166). Additionally, before initiating ARNI therapy in the hospital, it is important to determine that the patient will have uninterrupted access to ARNI therapy in the outpatient setting after discharge (i.e., factoring in cost and insurance coverage) (13). Changing ACEI to ARB early in the hospitalization to facilitate ARNI initiation without unduly increasing length of stay can be considered for some patients.

7.4.3. Beta Blockers

In patients with HF with the wet and warm profile who are taking beta blockers on admission, they should generally be continued unless blood pressure is low. If HF remains refractory to diuretics, the dose should be halved. Discontinuation should be considered if congestion remains unresponsive and certainly if the addition of intravenous inotropic therapy is contemplated. If decreased or held, beta blockers may be initiated or resumed in the absence of symptomatic hypotension or bradycardia, but a margin of stability is required in view of the known acute effects to lower cardiac output and increase filling pressures. Low doses and slow up-titration are recommended for a patient after recent decompensation. In the case of metoprolol, it is reasonable to give test doses of 6.25 mg of the short-acting metoprolol tartrate, but escalation of short-acting doses may be paradoxically less tolerable due to higher and more rapid peak effects (168). Alternatively, test doses of carvedilol 3.125 mg may be administered. Planned testing of increased doses of beta blockers should be part of the treatment plan either during the index hospitalization or following discharge. In hospitalized patients in whom GDMT medications have been held or not initiated, the optimal sequence of reinitiation of

FIGURE 8 Clinical Trajectory in Patients Who May Have Had Initial Improvement in Symptoms and Congestion, But Who Are Now Stalled



IV = intravenous.

ACEI and beta blockers has not been established, although outpatient studies suggest that that either an ACEI or beta blocker may be initiated first (169,170).

Consideration should be given to initiating or resuming beta blockers after decongestion, particularly in patients with more advanced disease or those in whom other GDMT has been titrated. Studies in selected stable patients have shown that low-dose beta blocker therapy can be safely initiated as late as a half-day prior to discharge (121,171), but this requires very early and frequent post-discharge surveillance. Patients who have required temporary IV inotropic therapy during hospitalization represent a higher-risk cohort and require longer periods of observation prior to and after beta blocker initiation. When it has been difficult to wean inotropic therapy, use of beta blockers is often deferred until stability has been confirmed after discharge.

7.4.4. Aldosterone Antagonists

The initiation or resumption of aldosterone antagonist therapy requires particular attention given evidence that less judicious use and failure to adhere to monitoring recommendations increase the risks of hyperkalemia and other adverse events (172). Patients in whom an aldosterone antagonist was initiated or continued while receiving IV loop diuretics should be monitored closely for rebound hyperkalemia as the diuretic dose is decreased or transitioned to oral therapy. Discontinuation of potassium supplementation may also be required. Lower than standard doses (i.e., less than eplerenone 50 mg or spironolactone 25 mg daily) may also be considered in those with at least moderate kidney impairment or other risks for hyperkalemia. For patients in whom 2 inhibitors of the renin-angiotensin system are being reinitiated or uptitrated, a sufficient period of time should be allowed to observe the combined effects of the 2 therapies on kidney function and serum potassium concentrations. It should be emphasized that the peak effect on potassium retention is generally not observed for several days; kidney function and potassium should be checked within 72 hours of discharge. Nonetheless, initiation in the hospital is safe with careful monitoring, and inpatient initiation will most likely lead to greater long-term use.

7.5. TRAJECTORY: Initial Improvement, Then Stalled

This trajectory would represent a patient who has had some improvement in symptoms and signs of congestion but does not reach the targeted goals of decongestion (**Figure 8**). Such patients tend to have more advanced disease, a history of frequent hospitalizations, and worse baseline kidney function. They commonly have high outpatient diuretic doses, and kidney function may worsen progressively with diuresis, a pattern associated with residual congestion and worse outcomes (119). In some cases, a high calculated net fluid loss is not reflected in weight changes due to high unrecorded intake. Addition of loading doses of amiodarone for therapy of atrial or ventricular arrhythmias can stall the progress of diuresis. Approximately 30% to 40% of ADHF patients discharged from the hospital still have moderate to severe congestion at the time of release (77,135,137,138,173).

No large randomized trial evidence exists to guide appropriate management for these patients, but it is considered reasonable to intensify the diuretic regimen using higher doses of intravenous loop diuretics or addition of a second (e.g., thiazide) diuretic, or to consider other therapies such as intravenous nitroglycerin as an adjuvant to diuretic therapy if symptomatic hypotension is absent (15). Temporary down-titration of neurohormonal antagonists may be necessary. In recent studies of decompensation with kidney dysfunction during HF hospitalization, neither low-dose dopamine nor low-dose nesiritide enhanced decongestion or improved kidney function when added to diuretic therapy, but there was a significant interaction, with a trend for enhanced diuresis with both therapies in patients with lower EF (<0.50) and lower blood pressures (SBP <114 mm Hg) (132).

If the patient has improved but has continued symptoms and/or signs, it is important to ascertain whether the signs and symptoms are predominantly due to HF. Persistent symptoms may reflect comorbidities as detailed in **Table 4**, particularly chronic pulmonary, kidney, or liver disease. In some cases, there may be mismatch between right- and left-sided filling pressures, which may require clarification of hemodynamics. Optimal JVP may be higher than normal in the setting of disproportionately high right-sided pressures. Conversely, orthopnea that does not resolve despite normal JVP may be due to left ventricular filling pressures disproportionately elevated compared with right-sided filling pressures, such that further diuresis or therapy with vasodilators may be needed. Uncertainty about the hemodynamic contribution and targets is a reasonable indication for invasive hemodynamic measurement in patients who are not clinically stable (15).

A significant proportion of patients do not receive optimized GDMT during hospitalization. Escalation of treatment in patients who are stalled may also entail adjustment of GDMT, with measures such as higher doses of diuretics and optimization of doses of neurohormonal antagonism and other therapies (103,174). In some cases, this may entail reduction of neurohormonal antagonist therapy, particularly in the setting of hypotension and progressive kidney dysfunction. In addition to optimization of GDMT and consideration of escalation of HF therapies, ongoing education of the patient and family members, addressing prognosis and goals of care, and treatment of comorbidities are of vital importance. Multidisciplinary care coordination and consultations as appropriate are important as well.

For some patients, the optimal hemodynamic state is not what was anticipated from initial presentation. The patient may have sufficient symptomatic improvement to be discharged but still show evidence of persistent congestion on examination. Further attempts at decongestion may have resulted in hypotension or progressive worsening of creatinine and/or blood urea nitrogen. At this point, it may be necessary to revisit goals of care and "compromise with congestion." However, persistent congestion defines a worse trajectory after discharge. Before accepting this compromise, the situation and options should be carefully reviewed as outlined in **Figure 8**.

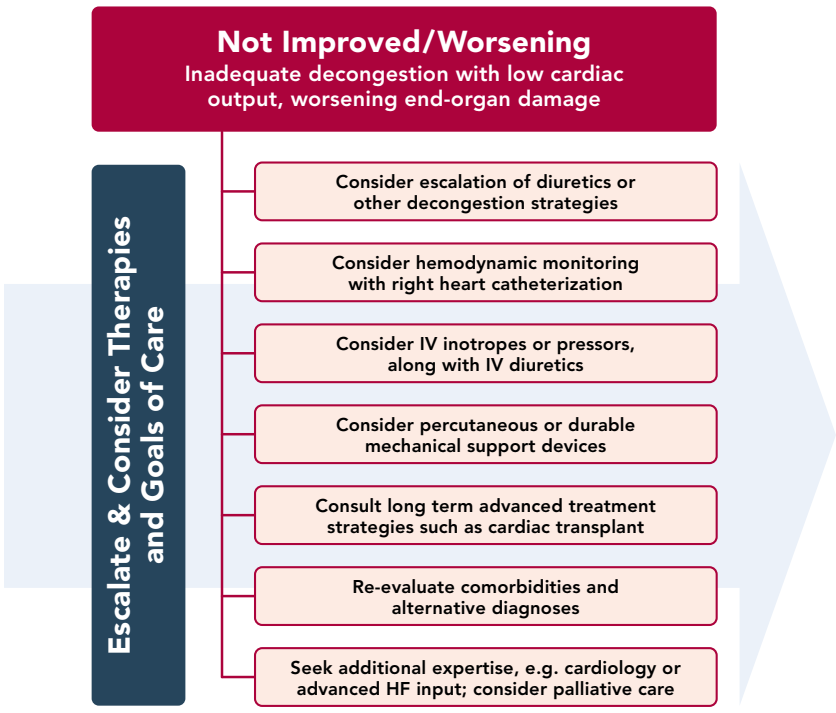
7.6. TRAJECTORY: Not Improved or Worsening

This trajectory represents a patient who is not responding to therapy, who has either failed to improve at all or has worsened during hospitalization (**Figure 9**). Some patients who appear to have stalled, as described in the previous text, may progress into this category. Approximately 20% to 30% of patients have no improvement in their symptoms or signs during hospitalization (175,176), and similarly, 15% to 20% of clinical trial patients have worsening HF that needs rescue therapy with additional diuretics, IV vasoactive agents, or mechanical circulatory or respiratory support (77,81,89,135,137,138,177).

These patients generally have refractory symptoms and signs of congestion. They often have a history of frequent hospitalizations after which they have continued Class III or IV symptoms. Those with HFpEF often have diuretic resistance and evidence of pulmonary hypertension and right HF. Patients with HFrEF may have borderline hypotension or hypoperfusion, evidence of end-organ damage such as progressive deterioration in kidney function and diuretic resistance, intolerance to GDMT, persistently high natriuretic peptide, and positive cardiac troponin levels. These factors are associated with worse in-hospital mortality rates and very poor overall prognosis.

The hospital trajectory of worsening may reflect acceleration of a previous gradual decline or a more acute process, possibly triggered by ischemia, arrhythmias or

FIGURE 9 Clinical Trajectory in Patients Who Are Not Improved or Are Worsening



HF = heart failure; IV = intravenous.

RV pacing, infection, medication side effects, or worsening of comorbidities (**Table 4**). It is not uncommon for these patients to demonstrate clustering of hospitalizations with increasing frequency, which may be a reflection of refractory HF (102,178).

If a patient is not improving or worsening, additional diagnostic strategies or specialist consultation may be considered. Intensification of diuretic therapy is appropriate, even if kidney function has worsened, because congestion is usually the main problem (**Figure 5**). It is crucial to re-evaluate the level of care, which may warrant escalation, including admission to an intensive care unit. It is reasonable to consider invasive hemodynamic monitoring to clarify right and left heart filling pressures and vascular resistances. In some cases, there may be more contribution of intrinsic pulmonary parenchymal or vascular disease than appreciated clinically. Knowledge of hemodynamics can guide more effective diuresis, in some cases facilitated by intravenous vasodilators or inotropic therapy. Intravenous inotropic therapy is frequently considered, with the anticipation of brief support until clinical improvement. However, it is common for patients already on a downward trajectory to become difficult to wean from intravenous inotropic therapy. Such therapy should not be initiated without considering the long-term plans for an exit strategy, including recognition that continuous home inotropic therapy can present a substantial financial and logistic burden for families. Exit strategies should also be carefully considered before embarking on dialysis or temporary mechanical circulatory support, even if anticipated to be temporary (15).

Especially in the setting of refractory or worsening congestion, which is commonly accompanied by worsening kidney function, unstable hemodynamic status, hypotension, and/or low perfusion state, it may not be feasible or safe to escalate GDMT, and doses may need to be decreased or held. After improvement to clinical stability and optimal volume status, however, it is important to re-address optimization of GDMT.

Review of the long-term trajectory of the patient who continues to worsen may warrant accelerated discussion regarding prognosis and goals of care (see Section 12). Aligning patients with local and distant family members with whom to share prognostic information and care options may require days of planning. This includes selection and arrangement of post-discharge care options, such as home health care, skilled nursing facilities, long-term care facilities, palliative care at home, or hospice.

7.6.1. Unexpected Sudden Event

Patients may deteriorate suddenly due to an unexpected event such as cardiac or respiratory arrest, shock, or arrhythmia. Potential etiologies could include ventricular tachycardia or fibrillation, pulmonary embolus, acute coronary syndrome, severe pump failure, shock, or other competing diagnoses such as acute kidney failure, acute infection or sepsis, respiratory failure, gastrointestinal bleed, and other sudden events. The acute precipitating factors should be sought and addressed if possible, including consideration that the decompensation may be due to something other than HF.

These patients may have varying levels of severity of HF ranging from new-onset to chronic advanced disease, and management should target the underlying etiology with an intent to restore hemodynamic stability and improve organ perfusion. In the setting of shock, hemodynamic evaluation with right heart catheterization and monitoring should be strongly considered. Lactate levels may indicate more critical hypoperfusion than recognized. Blood pressure-lowering medications may need to be held or given at reduced doses, and intravenous inotropic or vasoactive support may be required. For hemodynamic instability due to arrhythmia, medications should be re-evaluated, with discontinuation of proarrhythmic medications and consideration of new drugs, along with correction of electrolyte abnormalities, adjustment of implantable cardioverter-defibrillator parameters, and treatment of ischemia. Sepsis may be difficult to distinguish from a systemic inflammatory state related to circulatory collapse.

Percutaneous mechanical circulatory support may be instituted as a bridge to a decision about further advanced therapies or cardiac transplantation in patients for whom there is a reversible factor and/or advanced options for definitive therapy. As in patients with more gradual worsening of trajectory, exit strategies should be carefully considered when embarking on support anticipated to be temporary. It is critical to determine whether this event is truly unexpected or is a reflection of end-stage HF for which palliative care and end of life options may be more appropriate than aggressive invasive interventions. However, many sudden events can be treated effectively to return the patient to a favorable trajectory.

8. NODE: TRANSITION POINT

8.1. Need for a Distinct Transition Phase

The transition point heralds a distinct phase of care that begins after the decompensation leading to admission has resolved or has been addressed within the limitations of the chronic clinical profile. The focus then shifts toward how best to maintain stability of compensation. This most commonly occurs when clinical assessment reveals complete resolution of congestion and diuretic therapy is switched from intravenous to oral dosing.

Evidence suggests that many patients hospitalized with HF are discharged too early, before meeting criteria described in Section 7.1 and shown in **Figure 5**. The average length of stay in the U.S. has decreased to 4 days, compared with an average of at least 7 days in the rest of the world (179). The risk of readmission for HF has been linked to shorter lengths of stay, which may lead to incomplete decongestion, lack of appropriate titration of GDMT, and incomplete translation of plans to post-discharge care (21,104,180,181). As such, early discharge can lead to Excess Days In Acute Care (EDAC), a measure of acute care days within 30 days of discharge that is being incorporated into quality standards.

Verifying the effectiveness of oral diuretic therapy prior to discharge, as recommended in the guidelines (14,15,25), generally requires at least 24 hours of observation after discontinuation of intravenous diuretics. In a recent retrospective study, observing patients on their intended discharge diuretic regimen for ≥24 hours was associated with a significant reduction in 30- and 90-day HF readmissions (182). Discharge before 24 hours of stability on oral diuretics may occasionally be appropriate, particularly for well-known patients frequently hospitalized for whom the trigger for decompensation is obvious, net diuresis has been easily achieved, and early follow-up is available with a familiar clinician. Recent survey data from physicians at 1 center regarding attitudes toward discharge readiness questioned the utility of targeting complete decongestion and observing patients for a day on oral diuretics (183). However, their post-discharge practices were not surveyed and the readmission rates did not capture those admitted to other hospitals (87).

Separation of the transition point from the discharge day acknowledges the sequential steps and personnel time usually required for the intricate steps of discharge coordination, which is particularly important in high-risk patients. While assessment of educational gaps and other challenges for discharge begins early after admission, a directed multidisciplinary alert triggered by identification of the transition point can focus and finalize plans to support stability and further optimization of recommended therapies in the outpatient setting (13).

8.2. Planning Diuretic Therapy for Discharge

The dominant role of recongestion in HF readmissions suggests that current strategies for implementing a discharge diuretic plan are not reliable. This likely relates both to inadequate dosing at the time of discharge and lack of an adequate response plan that includes both an increased diuretic dose and the right clinical trigger for its use. The regimen taken prior to admission and the intravenous IV dosing required to achieve negative balance in

the hospital should both influence planning of a new discharge regimen.

Maintenance diuretic dosing should be planned recognizing that lower doses are required for fluid balance than for net diuresis, but also that fluid balance is usually harder to maintain at home than in the hospital, where patients have controlled intake and spend more time supine, a position that enhances renal blood flow. Torsemide and bumetanide are more reliably absorbed than furosemide and may be considered when daily furosemide doses are high. Kidney dysfunction may lead clinicians to underdose diuretics, despite evidence that transient worsening of creatinine during effective decongestion does not confer long-term decrements in kidney function (143,184) and that kidney dysfunction itself decreases diuretic responsiveness.

A **rescue dosing plan** should be included in the intended discharge regimen, to specify not only the increased diuretic therapy but also the personalized trigger that should prompt the rescue; patients should be encouraged to call their clinician if unsure, and to avoid delay in starting therapy. In the recent PIONEER-HF trial comparing initiation of sacubitril/valsartan to enalapril before hospital discharge, one-half of patients required an increase in diuretic dosing during the next 6 weeks (166). Reliance upon changes in weight or symptoms of congestion are most often used for this purpose, despite their low sensitivity and delayed kinetics for predicting decompensated HF (185). When possible, a patient's sentinel symptom of congestion should be recalled and emphasized, with care taken not to instruct reliance upon the appearance of edema or orthopnea for patients who have never experienced them. Adjustments to therapy may include increases in the dose and/or frequency of loop diuretic therapy or one-time doses of thiazide-type diuretics for sequential nephron blockade. Increases in mineralocorticoid dosing are rarely effective for rescue in outpatients and should not be made when kidney function might be declining. Dosing recommendations are in **Table 7**. Patients should be encouraged to call their clinician for clarification if unsure of their rescue plan.

A decision should also be made regarding **fluid restriction** after discharge, which is frequently done in hospital to accelerate net fluid loss. Stringent fluid restriction may not be necessary in patients who respond to low diuretic doses and do not habitually have high fluid intake. Two liters (64 ounces) is the usual practical limit by consensus, particularly for patients taking many medications.

Transition to oral diuretics should also trigger consideration of if and how **potassium supplements** should be prescribed for home. Need for supplementation is lower on oral diuretic dosing intended to maintain rather than decrease net fluid balance. Testing the oral potassium replacement schedule when switching to oral diuretics is preferable to relying upon potassium scale dosing until the time of discharge. Key considerations include changes in therapies that alter potassium elimination (ACEI, ARB, ARNI, and especially aldosterone antagonists) as well as conditions associated with increased risk of hyperkalemia (chronic kidney disease and diabetes mellitus). Patients who have undergone large volumes of diuresis without needing more potassium replacement in hospital require particular vigilance regarding risk of hyperkalemia after discharge.

8.3. Evaluating Tolerance of GDMT and Opportunities for Optimization

Patients who have received all of the recommended therapies for their HFrEF have consistently better long-term outcome than patients who have not, for whom optimization of GDMT is a high priority during and after hospitalization (15). For those in whom prior GDMT therapy was held during hospitalization, reinitiation should be attempted in the absence of contraindications. Lower doses may be required when therapy is resumed. It is equally important to evaluate the degree to which the patient has progressed in achieving target doses (Table 1 in Yancy et al. [13]). When neurohormonal antagonist therapies have been reduced or interrupted during hospitalization, the up-titration of GDMT may need to continue through the outpatient follow-up visits. Extensive guidance for GDMT optimization after discharge is provided by the 2017 ACC Expert Consensus Decision Pathway for Optimization of Heart Failure Treatment (13).

The transition point provides clinicians with an additional opportunity to consider enhancement of GDMT for the outpatient setting (88,103,106,121,160). For patients lacking a recommended medication, initiation is indicated if careful history reveals no contraindications (89). Some patients who have a long or complex history may not recall the reason for previous discontinuation; of particular concern would be a history of angioedema. A step of up-titration toward target dose should again be considered if not already attempted, recognizing the need to limit the number of changes in the setting of recent decompensation.

The period between the transition point and discharge should include confirmation that the patient tolerates the planned GDMT regimen for discharge. Absorption and vasodilation often increase after decongestion and can necessitate a reduction in dosing. Confirmation of tolerability includes documentation of the absence of postural hypotension and the administration of all doses as scheduled, without any being held for hypotension or dizziness (179,182,186). Postural symptoms early after discharge can sometimes lead to indiscriminate reduction

or discontinuation of recommended therapies. In addition, some medications may have been changed in the hospital due to formulary restrictions, so medicine reconciliation is crucial.

Whether further titration as an outpatient is expected, and also what factors may limit such titration, are crucial pieces of information to disseminate to the clinicians assuming care after the hospitalization. There is a place for such information on the **Focused Discharge Handoff** in this document (Section 10), and clinicians are encouraged to start thinking about these issues and documenting plans early in the hospitalization, well before the discharge day itself. This is a critical part of the "to-do" list for early post-discharge follow-up.

8.4. Additional Drug Therapy Considerations

Although most drug therapy decisions in ADHF will involve the optimization of core GDMT, some patients may be eligible for further optimization, such as the addition or titration of fixed dose long-acting nitrates and hydralazine in African Americans already receiving an ACEI, ARB, or ARNI and a beta blocker (13). One important caution regarding the optimization of GDMT is the premature addition of ivabradine in patients not receiving a maximally-tolerated dose of beta blocker therapy. Because beta blockers are sometimes decreased or stopped during an ADHF episode, an evaluation of tolerability may need to be deferred to long-term follow-up, as ivabradine was evaluated in outpatients and trial eligibility required persistently elevated heart rate on target or maximally tolerated doses of beta blockers (187). Digoxin may be considered in patients with advanced disease specifically for the purposes of symptomatic relief and reducing the risk of hospitalization as well as facilitation of resting rate control in atrial fibrillation, although caution should be exercised in those with renal impairment or other high-risk features for drug toxicity (e.g., advanced age, low body weight, female gender), as target serum concentrations are <1.0 ng/ml (188).

Medication regimens for comorbidities also merit consideration, particularly with respect to potential interactions with HF. The importance of considering the impact of medications for diabetes on cardiovascular risk is increasingly recognized (189). Although data for new therapies, including sodium-glucose cotransporter-2 inhibitors (-gliflozins) and glucagon-like peptide-1 receptor antagonists (-glutides) are confined to outpatients, inpatient initiation in some settings may permit an evaluation of tolerability and impact of concomitant HF therapy by a multidisciplinary team, and increase the potential for improved compliance (189). As diabetes therapy in the hospital is often limited to a sliding scale insulin regimen to avoid hypoglycemia, caution should be taken and information shared with the outpatient clinicians for patients discharged on different diabetic regimens. If sodium-glucose cotransporter-2 inhibitors are started in the hospital, careful consideration should be given to the dose of diuretic therapy, because these agents have potent osmotic diuretic effects. Regardless, close follow-up with a primary care provider, endocrinologist, or diabetes educator is recommended within 2 to 4 weeks for patients with hyperglycemia in the hospital, especially when medications have been changed (190).

8.5. Assessment of Risk at Discharge

The view of the trajectory and risk profile at admission may have included factors subsequently modified favorably during the HF hospitalization. Although the hospital trajectory has ideally been monitored throughout hospitalization, the transition node provides the last opportunity before discharge to re-evaluate the long-term prognosis. This review is important for the patient and family and for the clinicians who will provide care after discharge.

Favorable modification of risks from admission relates most often to the effectiveness of decongestion, to the enhancement of guideline-recommended therapies for patients with reduced LVEF (**Table 4**), and to improvement in patient education for adherence. It is vital to recognize that the degree of clinical congestion at admission does ***not*** confer increased risk after discharge, as long as decongestion has been achieved (67,104). Regardless of how it is measured, multiple factors relating to the severity of congestion at discharge predict worse quality of life, rehospitalization, and mortality. These factors include not only the symptoms and signs individually or combined into congestion scores (67,70,104), but also the natriuretic peptide levels, with progressively higher risk conferred by high absolute levels or with failure to reduce levels by at least 30% (24,55,63,68,109–113).

Discharge with residual congestion may reflect different limitations. Regardless of ejection fraction, severe underlying renal disease can lead to diuretic refractoriness and persistent fluid retention. Repeated discharge with residual congestion may be unavoidable when education and follow-up support has not improved adherence to the outpatient regimen, which may need to include different motivational interventions (76). Prolonged hospitalization can be futile when brisk daily sodium and urine output are exceeded by even higher daily intake despite restrictions. In the contemporary era of increasing GDMT, patients hospitalized despite adherence may be in later stages of HF, particularly with right HF, cardiorenal limitations, or chronic hypoperfusion (161). Increasing information is urgently needed to recognize when decongestion goals should be modified and how care should be redesigned to decrease the risk of

further decompensation after discharge with residual congestion (161).

For hospitalized patients in whom ACEI/ARBs were previously tolerated but then discontinued due to hypotension or kidney dysfunction (90), 1-year mortality may be as high as 50%, particularly if intravenous inotropic therapy is added. Discharge without beta blocker therapy is also associated with poor outcomes. If advanced hemodynamic instability precludes tolerability of neurohormonal antagonist therapies, the patient is on a downward trajectory and should be considered for advanced therapies or revision of goals of care. Another component of the discharge regimen that carries prognostic significance is the dose of loop diuretic (66). High doses required to maintain fluid balance indicate diuretic resistance, for which a major factor is chronic kidney disease (107,108).

Elements of risk that carry over from admission to discharge include advanced age, history of prior hospitalizations, and socioeconomic status (93–97). It remains unclear how often chronic patterns of nonadherence can be modified (108). Baseline kidney function remains a strong predictor of outcome, as transient changes are less relevant than the absolute levels of kidney function before and after discharge (120). New risk factors that can arise during the hospitalization include use of intravenous inotropic therapy, even if transient (20,125). Need for cardiopulmonary resuscitation or intubation are associated with a much higher risk of death within the next 6 months (66,81).

The risk assessment in the transition day should guide the priority for early follow-up. It is difficult to mandate the timing of follow-up as clinic personnel resources vary between institutions. However, residual congestion, discharge without ACEI/ARB/ARNI, discharge without beta blockers, or consideration for advanced therapies warrant the earliest clinic slots available for follow-up. Patients started on new medications in the hospital should be contacted every few days until their first follow-up visit, and should generally have electrolytes and renal function checked within a week. Instability of renal function and electrolytes prior to discharge also warrants repeat testing early after discharge, with the results sent to the receiving clinician identified in the hand-off form.

9. NODE: DISCHARGE DAY

Planning for discharge is ideally initiated at admission, with consideration regarding long-term goals of care, gaps in patient understanding and adherence, and optimization of the chronic regimen, while the patient is undergoing evaluation and treatment of the decompensation that led to admission. Successful transition from the hospital back into the residential setting is critical. General checklists have been provided for discharge of Medicare patients (https://www.medicare.gov/pubs/pdf/11376-discharge-planning-checklist.pdf) and for people with cardiac disease in the ACC hospital-to-home initiatives. The Target: HF program (https://www.heart.org/en/professional/quality-improvement/target-heart-failure) provides a checklist for completion at the time of HF discharge, and the Optimize Heart Failure Care Program has adapted best practice protocols to meet preferences and needs in numerous countries (191). Patients should also be considered for participation in exercise rehabilitation in a center near their home.

The discharge node has been organized into 3 major areas for communication: 1) summary of the ***medical course***, trajectory, and plans; 2) ***education to the patient and family*** that is culturally appropriate delivered verbally and in writing (**Figure 10**); and 3) ***identification of the continuing care clinicians*** to receive the handoff. The plans are multidisciplinary and should facilitate care around discharge and link it to the discharge phone calls and the outpatient clinic. Checklists can provide organizers to optimize communication, but multiple formats and structures are possible. Multiple team members will be involved in completing documents and checklists between the transition node and the day of the discharge, but it is recommended that the institution assign clear roles and responsibilities among care team members to ensure completion of key data elements.

10. FOCUSED DISCHARGE HANDOFF

We have proposed a focused distillation of crucial information, which could be at the beginning or end of the discharge summary or as a stand-alone communication (**Figure 11**). Many clinicians have found it difficult and time-consuming to locate this crucial information in the usual discharge summaries. Despite the utility of a detailed chronological summary of the hospital course, lack of a standard format limits it as a reference tool to be used in transition to continuing care clinicians. For example, one study examined nearly 700 hospital discharge summaries and found that only about one-half mentioned the primary care provider who would assume care of the patient (192). The following framework is designed as a predictable outline for those providing discharge phone calls and post-follow up clinic visits. It should also be included in the information provided to visiting nurses or other home health workers. Furthermore, this should be readily available for review of a patient who might return to the ED soon after discharge. The focused handoff is designed with selection menus for ease of use within a clinical decision support tool (electronic health record or mobile device) but could also be printed as a completed paper document. A version

FIGURE 10 Education for Patients, Families, and Caregivers

EDUCATION TO PATIENT/FAMILY/CAREGIVER THAT IS CULTURALLY APPROPRIATE DELIVERED VERBALLY AND IN WRITTEN FORM

☐ Current meds
- Dose/frequency
- Indication
- Potential side effects
- Potential adherence issues

☐ Activity level

☐ Dietary sodium restriction _____mg/day

☐ Fluid restriction ☐ Yes _____L/day or ☐ No_____

☐ Daily weight monitoring
- Has scale ☐ Yes ☐ No
- Logbook ☐ Yes ☐ No

☐ Assessment for peripheral edema

☐ Smoking cessation counseling for current or recent smokers

☐ Substance use counseling, if applicable

☐ List of warning signs of decompensation

☐ What to bring to each outpatient appointment
- List of meds
- Recordings of daily weights

☐ Who to call for increased weight / worsening symptoms / ICD discharge

_____

☐ Plans for continuation of care
- Cardiologist follow-up appointment _____/_____/_____
- Primary care follow-up appointment _____/_____/_____
- HF disease management program _____/_____/_____
- Cardiac rehabilitation _____/_____/_____
- Anticoagulation services follow-up, if applicable _____/_____/_____

with shading indicating selection menus in a clinical decision support tool is included in Appendix 5.

Multiple versions of the handoff could be created to match the needs of different institutions and different settings. However, it would ideally include most of these components in a common order, as there are advantages to uniformity of communication tools. A considerable amount of these data could potentially be retrieved automatically from the electronic health record (EHR). As such, there is a pressing need for information technology solutions to be developed on a broad basis to facilitate widespread adoption of communication tools for all patients being discharged from the hospital for decompensated HF to improve continuity of care during this transition phase.

Although a complete medication list is a necessary component of a full discharge summary and formal discharge document, the list can be very long. This focused hand-off highlights the most important medications central to optimizing the long-term outcome with HF. Lessons learned from the hospitalization put the inpatient team in the best position to estimate maintenance daily diuretic doses as well as potential rescue doses and the triggers for their use. Plans for optimization of GDMT, a process that often requires more up-titration in the stable outpatient setting, should be formulated and transmitted as well. Patients should be strongly encouraged to become active participants in their own care programs, and this form provides information about

FIGURE 11 Model Focused Discharge Handoff

FOCUSED DISCHARGE HANDOFF

Name_____ Age_____ MRN_____ Date of Discharge____/____/____Days in hospital_____

HF TYPE: ☐ HFrEF ☐ HFpEF ☐ Mid-range ☐ HFrEF with improved EF **HF ETIOLOGY:** ☐ Ischemic ☐ Non-ischemic ☐ Infiltrative ☐ Other

Last LVEF_____ Hospital Triggers_____

Arrhythmia history ☐ AF ☐ VT ☐ OTHER_____ Device Type _____

CONDITION AT DISCHARGE:

D/C BP: Sitting__/__Standing__/__ ***HR__Rhythm*** ☐ Sinus ☐ Afib ☐ paced sinus ☐ paced AFib ☐ freq PVCs ☐ freq PACs ***Congestion*** at D/C? ☐ Yes ☐ No

Edema (0-4+) ____**JVP**____ **Orthopnea** ☐ Yes ☐ No **Rales** ☐ none ☐ ¼ ☐ ½ ☐ wheezes ☐ pl eff **Ascites** ☐ Yes ☐ No **Liver**_____cm

Weight at D/C ____lbs Admission weight ____lbs ***Est target weight***____lbs____ ***If still wet, limited by***
☐ Dominant right heart failure ☐ Renal failure ☐ Hypotension ☐ Excessive fluid in hospital ☐ Frequent readmission pattern ☐ Other _____

Biomarkers: Admit BNP___or NT proBNP_____Troponin_____Discharge BNP (if known)_____or NT proBNP_____

Kidney Function: Discharge BUN/Cr_____ Worst in hospital_____ Baseline Cr (if known)_____

Comorbidities: _____

Psychosocial Factors: _____

Other hospital events: ☐ Code ☐ Sepsis ☐ Dialysis ☐ intubation IV inotropes used? ☐ Yes ☐ No Type: _____

Code Status: ☐ Full code ☐ Full code but recent discussions ☐ DNR/DNI ☐ DNI only ☐ ***Needs discussion***

DISCHARGE HF MEDICATIONS:

DIURETIC: Loop type_____, Dose_____mg/day. Metolazone_____mgs,_____(frequency or prn).

Triggers for rescue dose: If_____lbs up, or_____(sentinel symptoms)

Rescue dose _____orally, and / or metolazone_____mg for_____days before recheck

In hospital effective loop dose_____mgs IV ☐ daily ☐ BID ☐ TID ☐ drip at _____mg/hr Metolazone used? ☐ Yes ☐ No

K+ replacement_____mEq / day _____ Plan for K+ with rescue dose? ☐ Yes ☐ No

GUIDELINE DIRECTED MEDICAL THERAPY (For history EF < 40 only):

RAS meds: ACEI____mg/day ARB____mg/day ARNI____mg/day____ Dose decrease in hospital? ☐ Yes ☐ No

If none or dose decrease, why? ☐ Hypotension ☐ orthostasis/dizzy ☐ worsening renal fx ☐ hyperkalemia ☐ angioedema ☐ cough ☐ other

→ Is there a PLAN for outpatient increase or initation? ☐ Yes ☐ No

Beta blocker:_____mg/day Dose decrease in hospital? ☐ Yes ☐ No

If not, or dose decrease, reason? ☐ Hypotension ☐ bradycardia ☐ worsening renal function ☐ hyperkalemia ☐ fatigue ☐ other_____

→ Is there a PLAN for outpatient increase or initation? ☐ Yes ☐ No

Spironolactone or eplerenone ☐ Yes ☐ No if not, why ☐ Hypotension ☐ worsening renal function ☐ hyperkalemia

Other HF meds: Digoxin ☐ started ☐ continued ☐ stopped Ivabradine ☐ started ☐ continued ☐ stopped
Hydral/Iso ☐ started ☐ continued ☐ stopped

Anticoagulation *for* ☐ AF ☐ DVT/PE ☐ Mech valve ☐ hx embolism ☐ LV thrombus *with* ☐ Warfarin ☐ Apixaban ☐ Rivaroxaban ☐ Other DOAC

Antiplatelet *for* ☐ ACS ☐ PCI ☐ CAD ☐ stroke/TIA *with* ☐ ASA ☐ clopidogrel ☐ ticagrelor ☐ prasugrel Any hx bleeding? ☐ Yes ☐ No

Antiarrhythmic medications ☐ Amiodarone ☐ Dofetilide ☐ Sotalol ☐ Mexilitene ☐ Other_____

See patient discharge document and full discharge summary for complete med list

FOLLOW-UP: Discharge follow-up team_____, Appointment date and time_____

Home Health referrals (visiting nurses, PT, home infusion)_____

Post-discharge labs: Will be drawn at: _____Results sent to:_____

HF medication refills to_____

For worsening heart failure, contact_____ Phone Number_____

For non-cardiac issues, contact_____ Phone Number_____

Rhythm device follow-up_____

Other care providers_____

Is additional support needed for optimal care?_____

FIGURE 12 Checklist for Communication to Continuing Care Providers

COMMUNICATION TO CONTINUING CARE PROVIDERS

HOSPITAL COURSE

- ☐ Reason for admission
- ☐ Sentinel symptoms
- ☐ Congestion status
  - Admission, discharge, and target weight
  - Admission and discharge kidney function
  - Diuretic dosing
  - Rescue dosing
- ☐ Unexpected events

PLANNED THERAPIES AND MONITORING

- ☐ Plan for initiation, titration, and optimization of GDMT
  - ACE/ARB
  - Beta blockers
  - Aldosterone antagonists
  - ARNI
  - Ivabradine
  - Hydralazine/isosorbide
- ☐ Plan to monitor electrolytes and kidney function
- ☐ Follow-up for pending or planned diagnostic tests
- ☐ Plan for EP consult if sudden death risk or potential candidate for device therapy
- ☐ Recommendations for when to assess response to therapy
- ☐ Pneumovax and Influenza vaccination

FOLLOW-UP RELATED TO COMORBIDITIES

- ☐ Kidney function
- ☐ Diabetes
- ☐ Sleep-disordered breathing
- ☐ Depression
- ☐ Anemia
- ☐ Other

PSYCHOSOCIAL ISSUES RELEVANT TO ONGOING ADHERENCE

CONTINGENCY PLAN

- Diagnostic uncertainty
- What could go wrong and expected action plan

ADVANCE CARE PLANNING OR GOALS OF CARE DISCUSSIONS

self-care monitoring (e.g. serial weights, BP, HR, and symptoms), and who to contact for problems.

It is vital to identify the outpatient clinicians assuming responsibility after discharge. Difficulty filling in these names or appointment times should give clinicians pause during the discharge process. The last line concerning the need for additional support to optimize care provides an opportunity for thought about whether the way in which the patient is receiving care needs to be addressed, something that includes not only medical follow-up, but medication availability and support, access to care and medical advice, and psychosocial factors.

The Focused Discharged Handoff is specifically designed to travel with the patient and to provide the most important information for continuing care clinicians in multiple disciplines. This document is not, however, intended to replace direct communication with continuing care clinicians. A checklist of potential issues that might be discussed in that communication is listed in **Figure 12**.

11. NODE: EARLY POST-DISCHARGE FOLLOW-UP

The recently hospitalized patient is particularly vulnerable to decompensation after discharge. In the first 30 days following an admission for HF, up to 25% of patients will be readmitted (193). Risk factors for decompensation include not only incomplete recovery from acute illness, but also nutritional issues, sleep deprivation, and deconditioning (194). Issues not addressed during hospitalization, or those addressed but incompletely followed up, can also contribute. Management of the transition from inpatient to outpatient care is crucial, and the first post-discharge follow-up visit can serve as an essential fulcrum for these efforts (195). The post-discharge follow-up comprises 2 distinct events: 1) a follow-up phone call within 2 to 3 days of discharge; and 2) the clinic visit, within 7 to 14 days of hospital discharge.

11.1. Follow-Up Phone Call Within 48 to 72 Hours

The follow-up phone call should assess clinical signs of congestion, check on availability (and affordability, when pertinent) of medications, confirm understanding of and adherence with the medical regimen, and ensure that follow-up appointments have been made and that transportation to those appointments is not an issue. An important question to be asked is whether the patient feels that there were issues that were not addressed during the hospitalization. A systematic approach with a checklist can help organize and streamline the phone call to ensure that it is comprehensive yet focused. The follow-up phone call checklist (**Figure 13**) can be used on its own or integrated into the EHR. An important component of this assessment is to ensure that the patient (or caregiver) can verbalize understanding of the discussion (teach-reteach method) (196).

11.2. First Post-Discharge Visit

The first post-discharge appointment provides the opportunity to reassess clinical status, to provide additional patient education, to review medications and adjust their doses, and to address issues that might lead to readmission or worsening HF. The post-discharge risk assessment should be tailored to the patient's unique situation and needs. Social determinants of risk are often underappreciated; some of the most important risks that have been linked to HF readmission include income (94), socioeconomic status (95,197), employment (97), insurance status (198,199), lack of social support (96), and location factors (93,129,198).

Whereas the structure of the post-discharge visit can vary between medical centers, depending on the resources available, we believe that the key components listed in **Figure 14** should be considered, ideally linked to specific recommendations and potentially integrated into the EHR for ease of documentation. Of particular importance are evaluation as indicated by clinicians with expertise in HF, and other consultation such as from nutrition and social work, measurement of laboratories, management of comorbid conditions, and education. Outpatient health care services that have been assigned before discharge should be linked in, both to inform and be informed about the ongoing progress after discharge.

Management of Comorbidities. HF patients are often readmitted for diagnoses other than HF, and so active comorbid conditions (**Table 4**) should be aggressively addressed at the time of the post-discharge visit. Given the interplay between these disorders and the associated complexity, HF clinicians may need to serve as overseers of both cardiac and extracardiac disorders and to coordinate subspecialty care (200). Care of HF patients therefore requires a special understanding of the latest information on the comorbidities common to HF, and development of strategies to interface seamlessly with other disciplines, including primary care providers and other specialists.

Medication Reconciliation. HF patients are often prescribed multiple chronic medications, and errors in prescription are particularly common during transitions of care. The early post-discharge period offers an opportunity for comprehensive patient-centered medication reconciliation and continued progress toward optimization of recommended medical and device therapies (https://www.cardiosmart.org/SDM/Decision-Aids/Find-Decision-Aids/Heart-Failure).

As HF medications frequently remain at suboptimal doses, perhaps due to "therapeutic inertia" (201,202), a standardized approach to medication titration may be ideal, as has been addressed in prior decision pathway documents (TreatHF). Optimization of dosages is addressed in the Trajectory and Transition nodes, and information about future plans and potential impediments to increased dosing forms part of the Focused Discharge Handoff (**Figure 11**).

Laboratory Testing. Laboratory studies usually performed during the post-discharge visit include an assessment of kidney function when patients are in the transition period with medications. Electrolytes and renal function should be monitored closely, especially after major changes in diuretics, ACEI, ARB, ARNI, or aldosterone antagonists. Natriuretic peptide levels can be useful for following disease severity and prognosis in outpatients with chronic HF (26). (Care should be taken to measure N-terminal proBNP instead of BNP in patients taking ARNI, as ARNI increase BNP levels due to their inhibition of BNP degradation.) New biomarkers may add additional information regarding risk, but further studies are needed to establish the utility and cost-effectiveness of multimarker panels (127).

FIGURE 13 Checklist for Follow-Up Phone Call

CHECKLIST FOR FOLLOW-UP PHONE CALL WITHIN 48-72 HOURS

INTRODUCTION: My name is _____. I am calling from (either provider's office or hospital, depending on care coordination structure) to see how you are feeling and after your recent discharge from the hospital.

TOPIC	VITAL QUESTION	CAUSE FOR IMMEDIATE CONCERN	TEACHING POINTS TO BE COVERED IN CALL / CLINIC USING TEACH BACK
Symptoms - Sentinel symptom from hospitalization - Shortness of breath - Orthopnea - Edema	How is _____? ☐ Same ☐ Better ☐ worse than at discharge	Alert If **WORSE**	Do you know what symptoms you should be paying attention to?
Dizziness	Are you having trouble with dizziness? ☐ Yes ☐ No Is it just when you first stand up or does it last longer? _____	**FREQUENT DIZZINESS**	Review dizziness as potential symptom of concern
Daily Weights	Are you weighing yourself daily? ☐ Yes ☐ No If not, do you have a scale? ☐ Yes ☐ No What was your first weight at home after discharge? _____ What is your weight now? _____	**ALERT** If no weights or if weight increase > trigger	Importance of weights as short-term indication of fluid balance. Review diuretic plan from discharge Do you have a plan for what to do if your weight increases?
Medications (Refer to discharge list)	Do you have these medications prescribed at discharge? ☐ Yes ☐ No Do you know how to take them? ☐ Yes ☐ No Do you think you are having side effects from any of them? _____	**ALERT** If Not obtained, Or not taking correctly	Types and purposes of HF medications
Salt restriction	Are you watching your salt intake? ☐ Yes ☐ No What is your daily limit? _____ What are you doing to make sure you don't eat too much salt? _____		Review contribution of salt to fluid retention Common high-salt items How to read labels
Fluid restriction (for patients who have one)	Are you keeping track of your fluid intake? ☐ Yes ☐ No What is your daily limit? _____ What are you doing to stay within your limit? _____ _____		Review contribution of fluid to symptoms, Importance of fluid restriction for fluid balance and how to account for fluids in food as well as beverages. Reassure: this is often not a sign of dehydration in heart failure Present tricks such as frozen fruit, etc
Follow-up	When is your follow-up appointment? _____ Do you have a way to get there? _____	**NO F/U APPT** or no way to get there	
Physical Activity			

Vital questions are listed by topic, with highlights of responses that should raise immediate concerns. Inclusion of teaching points is desirable if time permits.

FIGURE 14 First Post-Discharge Visit Checklist

FIRST POST-DISCHARGE VISIT

☐ **History**
- Discharge summary reviewed.
- Etiology of cardiomyopathy identified.
- Precipitant of exacerbation identified.
- Heart failure compensated?
  - NYHA class.
  - Weight log reviewed?
  - Symptoms reviewed?
- Important concomitant disease states
  - CKD
  - Diabetes
  - Hypertension
  - COPD
  - OSA
  - Others

☐ **Physical Exam**
- Vital signs
- BMI
- Orthostatic blood pressure
- Jugular venous distention
- Rales +/-
- "cold/warm", "wet/dry" profile
- S3 present/absent

☐ **Diagnostic Testing**
- Basic metabolic panel
- Complete blood count
- BNP or NT pro-BNP
- Liver function panel (per discretion of clinician)
- Iron studies (per discretion of clinician)
- High sensitivity troponin, sST2, Gal-3 (per discretion of clinician)
- 12 lead ECG
- Chest X-Ray (per discretion of clinician)
- Review LVEF (__%). If not available, attain TTE
- Follow-up EF:
  - 40-days post MI
  - 3-months post NICM
- Ischemia evaluation needed?

☐ **Medications**
- Comprehensive medication reconciliation
- Beta-blocker?
  - Dose optimized?
- ACEI/ARB/ARNI
  - Dose optimized?
  - Contra-indication to ARNI?
- Aldosterone antagonist
  - Dose optimized?
- Diuretics?
  - Dose adjustment?
- Ivabradine? (Consider initiation if heart rate remains elevated despite beta blocker optimization)

☐ **Interventional therapies (if applicable)**
- Revascularization
- CRT
- ICD
- Valvular intervention

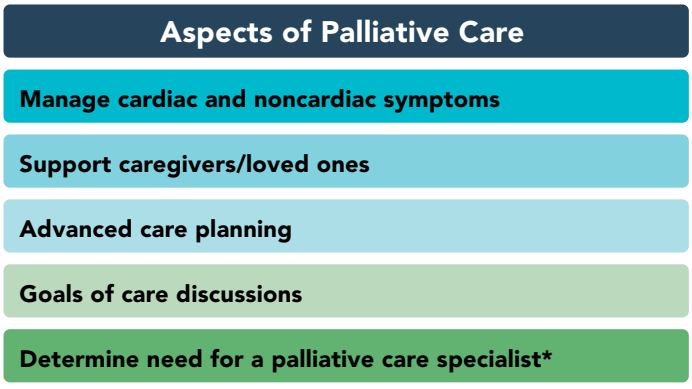
☐ **Patient education**
- Importance of adherence
- Medication education
- Dietary education
- Activity education
- Smoking cessation
- Cessation in alcohol consumption
- Follow-up appointment scheduled

☐ **Consultations**
- Home health services
- Cardiac rehab referral
- Advanced heart failure clinic referral
- Palliative/hospice referral

ACE-I = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor–neprilysin inhibitor; CKD = chronic kidney disease; BNP = B-type natriuretic peptide; COPD = chronic obstructive pulmonary disorder; CRT = cardiac resynchronization therapy; ECG = electrocardiogram; ICD = implantable cardioverter-defibrillator; LVEF = left ventricular ejection fraction; MI = myocardial infarction; NICM = nonischemic cardiomyopathy; NT-proBNP = N-terminal pro-B-type natriuretic peptide; OSA = obstructive sleep apnea.

Trajectory of Clinical Decline. A subset of patients with HF will continue to have symptoms and rapid disease progression despite being on maximally tolerated GDMT and may even need downtitration of neurohormonal blockade (196). When advanced treatment strategies such as transplantation or mechanical circulatory support may be options, referral to advanced HF specialists may be indicated or a shift in focus to palliative care may be appropriate for many stage D patients (203). The I-NEED-HELP algorithm can be useful to guide patient selection for referral follow-up to an advanced HF specialist (13). Clinicians caring for these patients need to consider carefully the variation among patients regarding their expectations and priorities (204). Goals of care that

FIGURE 15 Aspects of Palliative Care



*For management of complex noncardiac comorbid conditions (including psychosocial-spiritual distress), end of life symptom control, complicated advance care planning, disagreement between clinicians and patient/family, marked caregiver or family distress, and hospice referral.

include the potential for a focus on comfort should be a key component of discussions between clinicians and patients during the post-discharge period, particularly in high-risk patients.

12. PALLIATIVE CARE

Palliative care addresses goals of care, advance care planning, and symptom management for patients with life-threatening conditions or debilitating illness. Palliative care seeks to assess and mitigate the burden of disease experienced by patients, their caregivers, and their loved ones, including physical and psychosocial-spiritual distress (**Figure 15**). There is a growing recognition of the importance of palliative care in the management of patients with HF (109,110) and an emerging evidence base to support its routine incorporation (205). Important principles concerning integration of palliative care were outlined in the 2017 expert consensus document for optimization of treatment (13). Palliative care can coexist with active and even invasive treatments up to the point of transition to hospice care. Data show that referral to palliative care for these patients remains underutilized (54).

Practically, advance care planning involves prospective identification of a surrogate decision-maker and consideration of the type and degree of care that patients would choose in the event they lose decision making capacity. Ideally, all patients with HF would have advance care planning discussions about these issues as stable outpatients, but sometimes it is necessary to consider them in the inpatient or post-discharge setting. Even if a patient is not ready to discuss goals of care on admission, he or she should still be asked about confirming or establishing a surrogate decision-maker. Goals of care discussions should play an important part in the care of many patients admitted with HF (196), particularly at the point of trajectory checks (**Figure 4**). These discussions may consist of simply reviewing and confirming advance care plans, or may be more extensive and complicated (**Table 9** and Appendix 4).

The patient's values and preferences should have been explored prior to making advance care plans, but often require re-evaluation or clarification. A crucial step in the process is assessing a patient's readiness to engage in goals of care discussions. Understanding the patient's

TABLE 9 Goals of Care/Advanced Care Planning

Assess Readiness to Discuss Goals of Care
Assess Understanding of Prognosis
Confirm/Discuss Goals of Care
• Confirm/elicit patient values and preferences pertaining to quality of life and life prolongation (cultural, religious).
• Discuss aspects of what the patient would consider an unacceptable quality of life.
• Discuss benefits/burdens of reasonable therapeutic options.
Confirm/Establish Surrogate Decision Maker
• Person best able and willing to represent patient's values and preferences and patient's best interests.
Establish/Reassess Code Status
• Based on goals of care discussion.
• Do not attempt resuscitation (DNAR).
• Full code.
• Attempt shock without other measures.
Discuss Management of Defibrillator When Appropriate
• Pacing function is often left intact even if defibrillation is deactivated.
Determine Need for Specialist Palliative Care Consultation

perspective will allow the clinicians to address the issue in a sensitive manner. The clinician should then assess the patient's understanding of prognosis, a key foundation for goals of care discussions. Resources useful for both patients and clinicians in these discussions can be found at acc.org (https://www.cardiosmart.org/Palliative-Care/Planning-Your-Care) and in an HFSA Advanced Care Training Module (http://www.hfsa.org/wp-content/uploads/2018/03/HFSA-Module-9-03.14.2018-LR.pdf), and useful language is in Appendix 4.

Shared decisions among patients, families, and clinicians should harmonize goals of care with consideration of any new interventions related to the current hospitalization, particularly therapies like intravenous inotropic infusions, mechanical circulatory support, dialysis, and the defibrillation function of implantable cardioverter-defibrillators. These interventions require thoughtful consideration, with benefits to quality of life and longevity weighed in relation to burden and subsequent consequences. For example, many of these therapies, once instituted, may complicate options and timing for decisions regarding enrollment into hospice. Part of advance care planning includes consideration under what circumstances these therapies should be terminated/discontinued.

Ideally, decisions about termination of therapies, deactivation of devices, and code status should be congruent with each other, and also with prognosis, reasonable therapeutic options, and patients' overall goals. However, these decisions can be emotionally charged for patients and family members (who may play a significant role in decision-making, even when patients retain decision-making capacity), and sometimes they may be incongruent for a time. For many patients, there will be a stepwise progression away from life prolongation by means of all available interventions to simplification of care toward comfort. Attention to quality of life for patients and offers of bereavement counseling for families can help to alleviate distress and are often best accomplished under the aegis of hospice care. However, many patients understanding their prognosis continue to value life extension, even in the setting of suffering (196,206), and may not choose hospice care until within a few hours or days of death. Clinicians participating in advance care planning and goals of care discussions should be attuned to these issues and help patients and caregivers/loved ones to explore their values, fears and hopes.

Specialists in palliative care can be useful at several points during the hospitalization. They are particularly skilled at helping patients and families navigate the difficult process of complicated advance care planning and goals of care discussions, particularly in the setting of unrealistic expectations, which may play out as demands for medically inappropriate care. Working to establish realistic expectations early on, as part of trajectory checks, can help to avoid this situation. When it does occur, ethics consultants can plan an important role, in addition to palliative care experts. Palliative care specialists also provide expertise in managing noncardiac symptoms and holistically improving quality of life near the end. While treatment to relieve symptoms of congestion usually continues until death and is the purview of clinicians caring for patients with HF, palliative care specialists can provide help regarding use of opiates for refractory dyspnea and pain and treatment of other end-stage symptoms such as agitation and sleeplessness. Palliative care specialists may also help to facilitate the transition to hospice.

Involvement of continuing care providers is of obvious importance; the focused discharge handoff should specify code status and also note when discussion of goals has been deferred to the outpatient setting (**Table 9**).

13. DISCUSSION AND IMPLICATION OF PATHWAY

This Expert Consensus Decision Pathway document complements current guidelines by addressing unresolved issues in patients hospitalized with HF. We have construed our task broadly to comprise assessment extending from the original ED visit through the first post-discharge visit, with more focus on optimizing patient care and improving outcomes than on length of stay and prevention of readmission. We have also focused on assessments and goals of therapy more than on specific therapies, which are discussed extensively in other consensus documents. Although this document follows the path for patients admitted with a primary diagnosis of HF, increasing evidence suggests that patients admitted with secondary diagnosis of HF also have a high rate of HF events including hospital readmission. We would envision that many of these principles would be adapted into a more integrated approach to HF and its comorbidities in the hospital, regardless of the primary diagnosis listed at the time of admission.

Risk assessment involves collection of information but is most useful when that information is translated into strategies to address risk factors and so minimize risk going forward. The comprehensive initial assessment provides an opportunity to review data and formulate a multifaceted, multidisciplinary care plan that includes not only relevant cardiac issues but also patient-specific comorbidities and barriers to care.

We have outlined management of patients based on clinical trajectory. While near-term trajectory guides day-to-day management of the hospitalized HF patient, a broader view of the long-term trajectory is crucial to anticipate problems and plan therapy going forward.

Hospitalization provides an excellent opportunity to improve clinical trajectory by reducing congestion and optimizing GDMT both in the hospital and after discharge. In patients with clinical decompensation and/or incomplete resolution of congestion, it may be appropriate to re-address goals of therapy.

As important as formulating a therapeutic plan going forward is communicating that plan to patients and continuing care providers. The focused discharge handoff in the document provides one framework to integrate the data most crucial for continuity of care after discharge in a format easily accessible to team members.

This pathway aims to help clinicians make good decisions, but does not replace good clinical judgment; strategies must be adapted to individual patient situations. Those strategies will continue to evolve as new evidence becomes available.

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PRESIDENT AND STAFF

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KEY WORDS ACC Expert Consensus Decision Pathway, angiotensin receptor-neprilysin inhibitors, beta blockers, CHF, comorbidities, diuretics, guideline-directed medical therapy, heart failure

APPENDIX 1. AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (RELEVANT)—2019 ACC EXPERT CONSENSUS DECISION PATHWAY ON RISK ASSESSMENT, MANAGEMENT, AND CLINICAL TRAJECTORY OF PATIENTS HOSPITALIZED WITH HEART FAILURE

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Continued on the next page

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CEC = clinical events committee; DSMB = Data and Safety Monitoring Board; KC = Kansas City.

APPENDIX 2. PEER REVIEWER RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES —2019 ACC EXPERT CONSENSUS DECISION PATHWAY ON RISK ASSESSMENT, MANAGEMENT, AND CLINICAL TRAJECTORY OF PATIENTS HOSPITALIZED WITH HEART FAILURE

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APPENDIX 3. ABBREVIATIONS

ACC = American College of Cardiology

ACEI = angiotensin-converting enzyme inhibitor

AHA = American Heart Association

ARB = angiotensin receptor blocker

ARNI = angiotensin receptor-neprilysin inhibitor

BNP = B-type natriuretic peptide

ED = emergency department

EF = ejection fraction

EHR = electronic health record

GDMT = guideline-directed medical therapy

HF = heart failure

HFpEF = heart failure with preserved ejection fraction

HFrEF = heart failure with reduced ejection fraction

NT-proBNP = N-terminal pro B-type natriuretic peptide

RAS = renin-angiotensin system

APPENDIX 4. ADVANCE CARE PLANNING

Clarifying and articulating patients' values

- "What are the things that give your life meaning?"
- "Given your current situation, what do you hope for? What are you most worried about?"
- "Some patients say that if they became so sick that they could not do certain things (like recognize or talk to their loved ones), they would want all possible treatments to prolong their life. Other patients say they would rather have care focused on comfort, rather than life-prolongation. How would you say this applies to you?"
- "What health situation would you find so unacceptable that you would consider it worse than death?"

Choosing a surrogate

- "If you were to become so sick that you could no longer make decisions for yourself, who would you trust to make medical decisions for you? Who would make the same healthcare choices for you that you would make for yourself? Who knows your wishes the best?"
- "Does this person know that you have chosen him/her for this role?"
- "Have you had a discussion with this person about the values that guide your healthcare decisions and/or situations in which you would not want certain treatments?"

APPENDIX 5. ALTERNATIVE FORMAT FOR THE FOCUSED DISCHARGE HANDOFF

Shaded portions indicate responses that should populate selection menus, either written as shown on the second page, or as part of a clinical decision support tool.

FOCUSED DISCHARGE HANDOFF

Name_____ Age_____ MRN_____ Date of Discharge____/____/____Days in hospital_____

HF TYPE: **HF ETIOLOGY:**
Last LVEF_____ Hospital Triggers_____
Arrhythmia history Device Type _____

CONDITION AT DISCHARGE:
D/C BP: Sitting _/_ Standing _/_ ***HR_Rhythm*** ***Congestion*** at D/C?
Edema (0-4+) ____**JVP**____ **Orthopnea** **Rales** **Ascites** **Liver**_____cm
Weight at D/C ____lbs Admission weight ____lbs ***Est target weight***____lbs____ ***If still wet, limited by***
Biomarkers: Admit BNP_____or NT proBNP_____Troponin_____Discharge BNP (if known)_____or NT proBNP_____
Kidney Function: Discharge BUN/Cr_____ Worst in hospital_____ Baseline Cr (if known)_____
Comorbidities: _____
Psychosocial Factors: _____
Other hospital events: IV inotropes used? Type: _____
Code Status:

DISCHARGE HF MEDICATIONS:
DIURETIC: Loop type_____, Dose_____mg/day. Metolazone_____mgs,_____(frequency or prn).
Triggers for rescue dose: If_____lbs up, or___(sentinel symptoms)
Rescue dose _____orally, and / or metolazone_____mg for_____days before recheck
In hospital effective loop dose_____mgs IV drip at _____mg/hr Metolazone used?
K+ replacement_____mEq / day _____ Plan for K+ with rescue dose?

GUIDELINE DIRECTED MEDICAL THERAPY (For history EF < 40 only):
RAS meds: ACEI____mg/day ARB____mg/day ARNI____mg/day____ Dose decrease in hospital?
If none or dose decrease, why?
→ Is there a PLAN for outpatient increase or initation?
Beta blocker:_____mg/day Dose decrease in hospital?
If not, or dose decrease, reason?
→ Is there a PLAN for outpatient increase or initation?
Spironolactone or eplerenone if not, why
Other HF meds: Digoxin Ivabradine
Hydral/Iso
Anticoagulation *for* *with*
Antiplatelet *for* *with* Any hx bleeding?
Antiarrhythmic medications
See patient discharge document and full discharge summary for complete med list

FOLLOW-UP: Discharge follow-up team_____, Appointment date and time_____
Home Health referrals (visiting nurses, PT, home infusion)_____
Post-discharge labs: Will be drawn at: _____Results sent to:_____
HF medication refills to_____
For worsening heart failure, contact_____ Phone Number_____
For non-cardiac issues, contact_____ Phone Number_____
Rhythm device follow-up_____
Other care providers_____
Is additional support needed for optimal care?_____

Continued on the next page

SELECTION MENUS

HF TYPE:
☐HFrEF
☐HFpEF
☐mid-range
☐HFrEF with improved EF

HF ETIOLOGY:
☐ischemic
☐nonischemic
☐infiltrative
☐other

CONDITION AT DISCHARGE
Rhythm
☐sinus
☐Afib
☐paced
☐freq PVC
☐freq PAC

Rales
☐ none
☐ ¼
☐ ½
☐ wheezes
☐ pl eff

If still wet, limited by
☐dominant right heart failure
☐renal failure
☐hypotension
☐excessive fluid in hospital
☐frequent readmission pattern
☐other_____

Other hospital events:
☐code
☐sepsis
☐dialysis
☐Intubation

Code Status:
☐full code
☐full code but recent discussions
☐DNR/DNI
☐DNI only
☐*Needs discussion*

DISCHARGE HF MEDICATIONS
In-hospital effective loop dose ___ mgs IV ☐daily ☐BID ☐TID ☐

GUIDELINE DIRECTED MEDICAL THERAPY
RAS meds
If none or dose decrease, why?
☐hypotension
☐orthostatic/dizzy
☐worsening renal fx
☐hyperkalemia
☐angioedema
☐cough
☐other

Beta blocker
If none or dose decrease, why?
☐hypotension
☐bradycardia
☐worsening renal function
☐fatigue
☐other_____

Spironolactone or eplerenone ☐yes ☐no, If not, why
☐hypotension
☐worsening renal function
☐hyperkalemia

Other HF meds:
Digoxin ☐started ☐continued ☐stopped
Ivabradine ☐started ☐continued ☐stopped
Hydral/Iso ☐started ☐continued ☐stopped

Anticoagulation *for*
☐AF
☐DVT/PE
☐mech valve
☐hx embolism
☐LV thrombus *with*
- ☐warfarin
- ☐apixaban
- ☐rivaroxaban
- ☐other DOAC

Antiplatelet *for*
☐ACS
☐PCI
☐CAD
☐stroke/TIA *with*
- ☐ASA
- ☐clopidogrel
- ☐ticagrelor
- ☐prasugrel

Antiarrhythmic medications
☐amiodarone
☐dofetilide
☐sotalol
☐ mexilitene
☐other_____