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EXPERT CONSENSUS DECISION PATHWAY

2024 ACC Expert Consensus Decision Pathway on Clinical Assessment, Management, and Trajectory of Patients Hospitalized With Heart Failure Focused Update

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

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1. EXECUTIVE SUMMARY

Heart failure (HF) affects nearly 6.7 million Americans, and its prevalence continues to increase.[1-3] Inpatient admissions for HF are associated with high mortality, and readmissions and subsequent health events are common.[4,5] Although symptoms often improve rapidly during HF hospitalization, episodes of worsening HF nevertheless may mark a fundamental change in the HF trajectory; patients admitted with HF have a 20% to 30% risk of death within 1 year.[1,6] Goals of hospitalization thus include not only good clinical response, but also the assessment and optimization of therapy to address the long-term trajectory after discharge.

The purpose of this focused update is to revise the "2019 ACC Expert Consensus Decision Pathway on Risk Clinical Assessment, Management, and Clinical Trajectory of Patients Hospitalized with Heart Failure"[7] in areas where new evidence has emerged since its publication. This focused update has undergone a rigorous, multilevel review and approval process, similar to that of the full 2019 Expert Consensus Decision Pathway (ECDP).

A principal goal of this update is to harmonize with the most recent 2022 American College of Cardiology (ACC)/American Heart Association (AHA)/Heart Failure Society of American (HFSA) HF Guideline[8] and also with the ECDP documents concerning optimization of therapy in chronic heart failure with reduced ejection fraction (HFrEF)[9] and chronic heart failure with preserved ejection fraction (HFpEF).[10] **Figure 1** retains the general approach of tailoring therapy to the clinical trajectory but now places increased emphasis on establishing all 4 pillars of guideline-directed medical therapy (GDMT) for HFrEF in the hospital, when possible, along with ensuring appropriate follow-up to monitor tolerance and continue titration. This figure also now includes sodium-glucose cotransporter (SGLT) inhibition; given the robust evidence for safety and efficacy of SGLT inhibitors,[11-14] their use can be considered earlier than or concomitant with neurohormonal antagonists such as beta-blockers, angiotensin receptor/neprilysin inhibitors (ARNIs)/angiotensin-converting enzyme (ACE) inhibitors/angiotensin receptor blockers (ARBs), and mineralocorticoid antagonists (MRAs).

This update focuses on modifications of the original figures dealing with initiation and titration of therapy. Characteristics of patients hospitalized for HF often differ from populations enrolled in outpatient trials, and thus strategies for initiation of GDMT may need to be adapted in these patients. A new figure describes titration strategies in patients with different presentations. Patients may have a new HFrEF diagnosis, in which case initiation of all 4 pillars of GDMT should be attempted. Patients with chronic HF on partial GDMT should receive personalized therapy to fill in gaps, considering a switch from an ACE inhibitor/ARB to ARNI, if appropriate. Caution is required for patients with chronic Class IV HFrEF with decompensated HF, some of whom may not tolerate neurohormonal antagonists, even in low doses. The description of management strategies in patients with different short-term clinical trajectories in the hospital has also been updated.

Decongestion remains an important therapeutic goal in the hospital. The sections on diuresis have been updated to include alternative agents and dosing strategies based on new data. Recommendations about communication and follow-up have been adapted to changing trends such as the use of telehealth. Finally, there is additional emphasis on long-term disease trajectory, and guidance on goals of care discussions and identification of patients for whom specialist palliative care referral may be indicated.

Here are the 10 key changes to the 2019 ECDP in this focused update.

1. This update emphasizes SGLT inhibitor therapy throughout hospitalization regardless of LVEF, and places a greater emphasis on initiation of the other pillars of therapy for HFrEF after stabilization.
2. Hospital admission from the ED is generally indicated for a new diagnosis of HF with rapidly progressive symptoms, severe congestion, or higher complexity of disease; some low-risk patients may potentially receive care in an observation unit or Hospital at Home (HaH) setting.
3. The typical routes to HF admission include newly diagnosed HF, chronic HF with previous therapy, or advanced HF with chronic Class IV symptoms despite previous recommended therapies.
4. Daily review of the hospital trajectory often shows continuing progress toward effective decongestion and stabilization for initiation of guideline-directed neurohormonal therapies.
5. Daily trajectory review may also show stalling after initial response, failure to respond, or worsening HF, which may warrant adjunctive diuretic agent therapies, reconsideration of etiology, physiology and comorbidities, possible escalation to other therapies, and re-evaluation of goals of care.
6. SGLT inhibitors and mineralocorticoid antagonists have little effect to reduce blood pressure and in the absence of contraindications, can be initiated at any time during hospitalization and continued at discharge if feasible.
7. Strategies for optimization of guideline-directed neurohormonal therapies of beta-adrenergic blocking agents and ARNI/ACE inhibitor/ARB should consider previous tolerance of these therapies, current hemodynamics, and kidney function.
8. Selection of ARNI/ACE inhibitor/ARB or switch from ACE inhibitor/ARB to ARNI are indicated for HFrEF, and in combination with beta-blockers can generally be initiated after clinical stabilization to optimal volume status, with careful titration to avoid hypotension or kidney dysfunction during hospitalization and early after discharge.
9. Detailed information regarding diagnoses, discharge regimen and plans should be provided to patients and referring providers and used as a reference for the follow-up phone calls and first postdischarge visits, including those conducted via telehealth.
10. Palliative care plays an increasingly important role in helping patients recognize progressive disease and re-evaluate goals of care, with benefit shown for palliative care referral tools and palliative care consultation to increase completion of advance directives and reduce hospital readmission rates.

Changes to the 2019 ECDP included in this focused update are summarized in Table 1 of the Supplemental Online Appendix.

In accordance with the ACC Relationships With Industry and Other Entities Policy, relevant disclosures for the writing committee and comprehensive disclosures for external peer reviewers are available in Appendixes 1 and 2. A list of abbreviations relevant to this ECDP can be found in Appendix 3.

For additional details concerning ECDPs, please refer to the Preface and Methods sections. To ensure complete transparency, a comprehensive table of the writing committee's relationships with industry and other entities, including relationships not pertinent to this document, has been created. All these items are available in the Supplemental Online Appendix.

2. UPDATED CONTENT FROM THE 2019 ECDP ON PATIENTS HOSPITALIZED WITH HEART FAILURE

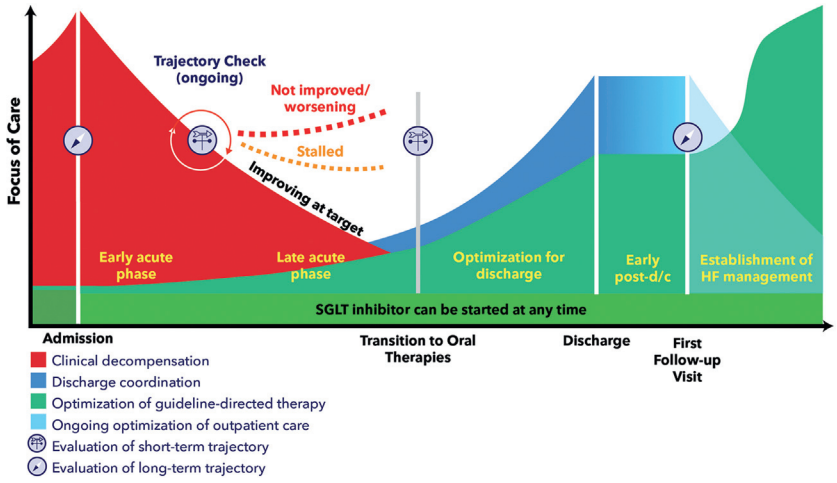
This section contains updated content from the 2019 ECDP on Patients Hospitalized with Heart Failure. Modified figure and table numbers remain unchanged, while the new figures and tables are numbered accordingly.
The following tables and figures have been modified or are new for this focused update:

- Modified Figures: 1, 2, 5, 6, 10-15
- New Figures:
  - 7A (combined from Figures 7, 8, and 9 from the 2019 ECDP)
  - 7B
- Modified Table: 7
- New Table: 7A

The following tables and figures were not modified and are not included in this focused update. Please consult the 2019 ECDP on Patients Hospitalized With Heart Failure to access them:

- Figures: 3 and 4
- Tables: 1-6 and 8

FIGURE 1 Pathway Summary Graphic—Clinical Course of Heart Failure



Graphic depiction of the course of HF admission, showing the degree of focus on clinical decompensation (red), discharge coordination (blue), ongoing optimization of outpatient care (light blue), and optimization of guideline-directed therapy (green). Ongoing assessment of the inpatient clinical course is depicted as a circle of arrows, with key timepoints for evaluation of short-term trajectory indicated by weathervane signs. Key timepoints for evaluation of the long-term clinical trajectory for the HF journey are depicted by compass signs. HF = heart failure; post-d/c = postdischarge; SGLT = sodium-glucose cotransporter.

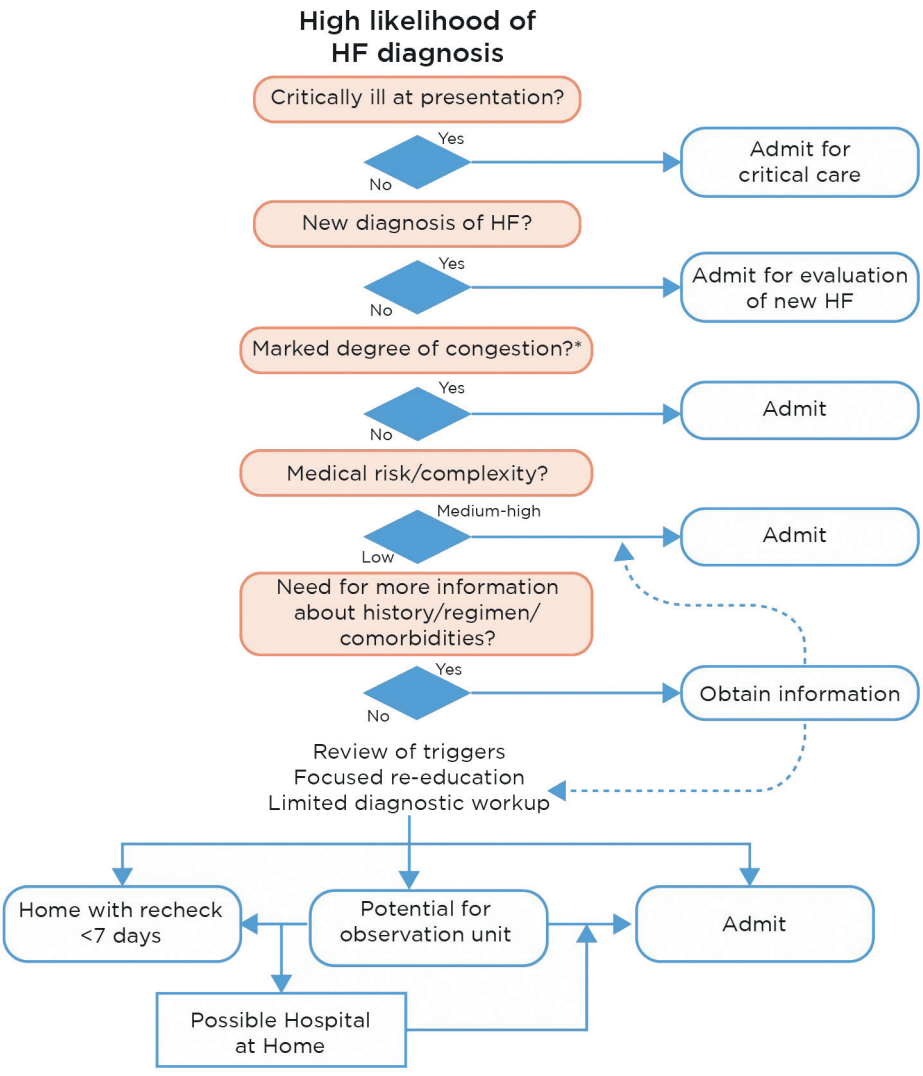
Figure 1 has been revised from the original document to provide more emphasis on optimization of guideline-directed therapy and extension and intensification of that optimization after discharge and at the first follow-up visit. The most notable change is the addition of SGLT inhibition to guideline-directed management for both HFrEF and HFpEF. SGLT inhibition is distinct from neurohormonal inhibition in that the minimal effects on blood pressure and kidney function allow for its initiation at any time in the treatment course, as long as contraindications such as type 1 diabetes and end-stage kidney failure are not present.

2.1. Rationale for and Data Concerning SGLT Inhibition in Hospital

2.1.1. Sodium-Glucose Cotransporter Inhibitors

Robust evidence supports the use of SGLT inhibitors to further reduce morbidity and mortality in patients with chronic HF across a wide spectrum of left ventricular ejection fractions and with or without type 2 diabetes mellitus (T2DM). Evidence now supports initiating or continuing their use in the hospital setting for patients admitted with acute decompensated HF.[11-15] The SOLOIST-WHF (Effect of Sotagliflozin on Cardiovascular Events in Participants with Type 2 Diabetes Post Worsening Heart Failure) trial with sotagliflozin, an inhibitor of SGLT 1 and 2; EMPULSE (Empagliflozin in Patients Hospitalized with Acute Heart Failure who have been Stabilized), EMPA-RESPONSE AHF (Effects of Empagliflozin on Clinical Outcomes in Patients with Acute Decompensated Heart Failure), and EMPAG-HF (Effects of Early Empagliflozin Initiation on Diuresis and Kidney Function in Patients with Acute Decompensated Heart Failure) with empagliflozin; and DICTATE-AHF (Efficacy and Safety of Dapagliflozin in Acute Heart Failure) and DAPA-Resist (Dapagliflozin Versus Thiazide Diuretic in Patients with Heart Failure and Diuretic Resistance) with dapagliflozin have all demonstrated that these medications can be safely started in inpatients.[11-15] Similar criteria for clinical stability were used by these trials before initiating SGLT inhibitors, consisting of a systolic blood pressure (SBP) of at least 100 mm Hg, no inotropic support for at least 24 hours, no symptoms of hypotension, no increase in intravenous (IV) diuretic agent dose in the previous 6 hours, and no IV vasodilators, similar to the stability criteria from the sacubitril/valsartan trials.[16] In addition to reductions in major adverse cardiovascular events, data from the DAPA-Resist trial of dapagliflozin and the RECEDE-CHF (Renal and Cardiovascular Effects of SGLT2 Inhibition in Combination with Loop Diuretics in Patients with Type 2 Diabetes and Chronic Heart Failure) and EMPAG-HF trials of empagliflozin showed that SGLT inhibition augmented urine output and diuretic response.[13,15,17] Results from the DICTATE-AHF trial of dapagliflozin indicated that initiation soon after admission decreased congestion and shortened length of stay; the primary endpoint of diuretic efficiency (cumulative change in weight per cumulative diuretic agent dose) was not met.[18]

FIGURE 2 Risk Stratification of Acute HF in the Emergency Department



*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. HF = heart failure.

Early initiation of an SGLT inhibitor can be considered any time after admission in patients who are hemodynamically stable with estimated glomerular filtration rate ≥20 mL/min/m^2. SGLT inhibitors should not be initiated in patients with evidence of hypovolemia and should trigger reassessment of diuretic requirements, which can decrease acutely and chronically. SGLT inhibitors are associated with euglycemic diabetic ketoacidosis in settings in which insulin levels are decreased and are thus not approved in type 1 diabetes. In patients with T2DM, euglycemic diabetic ketoacidosis is rare, but signs and symptoms of hypoglycemia should be monitored, and blood glucose levels should be reassessed after the initiation of SGLT2 inhibitors in the context of the diabetic regimen. Glucosuria increases the risk of urinary tract and genital mycotic infections, especially in patients with T2DM.

Out-of-pocket cost is currently a concern with novel agents, including SGLT inhibitors, sacubitril/valsartan, direct-acting anticoagulants, and other agents. Thus, affordability should be routinely determined before discharge with these and other medications for which coverage may be limited. Although patients affected by the social determinants of health are at the highest risk for HF readmission, they are also most likely to be

affected by issues related to the affordability of novel therapeutic agents. Wherever possible, clinical teams should assist in directing patients to financial assistance and other programs that might help to improve access to these novel agents.

2.2. Initial Triage of HF for Admission

Figure 2 has been modified to include consideration of HaH. During the public health emergency in November 2020,[19,20] the Centers for Medicare & Medicaid Services launched the Acute Hospital Care At Home (AHCaH or HaH) program, offering hospitals expanded flexibility to care for patients in their homes.[21,22] HaH provides health care to acutely ill patients in their homes using methods like telehealth, remote monitoring, cardiac rehabilitation,[5] and regular in-person visits by nurses or physicians. As clinicians[23,24] and patients[25] reconsider how and where care is delivered, some are using the HaH model as a possible approach to improve value[26] and address hospital capacity constraints.[27] This care delivery model has been shown to reduce costs,[28] improve outcomes,[28-32] reduce readmissions,[28,31] improve activities of daily living,[33] reduce levels of family member stress,[34] and provide equitable care.[35] Although questions remain[36] on outcome measurement,[37] criteria for participation,[38] training, stakeholder perceptions,[39,40] regulatory issues, payment models, scaling, ethics,[41] and length of treatment,[42] it is an alternative for patients with acute HF,[40] particularly those who present to the ED or after an observation stay.[42] HaH care models applied to small cohorts of patients with HF, with strict inclusion criteria, have suggested increased time to readmission,[43] lower costs,[44,45] and improved health-related quality of life,[43] with no significant differences in adverse outcomes. A recent meta-analysis provided additional confirmation of these results.[46] Identifying patients with HF expected to benefit from HaH is challenging, and eligible patients should be selected based on multiple factors, including etiology of reduced heart function, functional capacity, comorbidities, and system limitations. As this care model expands, attention should focus on ensuring that HaH is inclusive of those whose home situations may be more challenging due to the social determinants of health.

2.3. Decongestion With Diuretic and Adjunctive Therapy

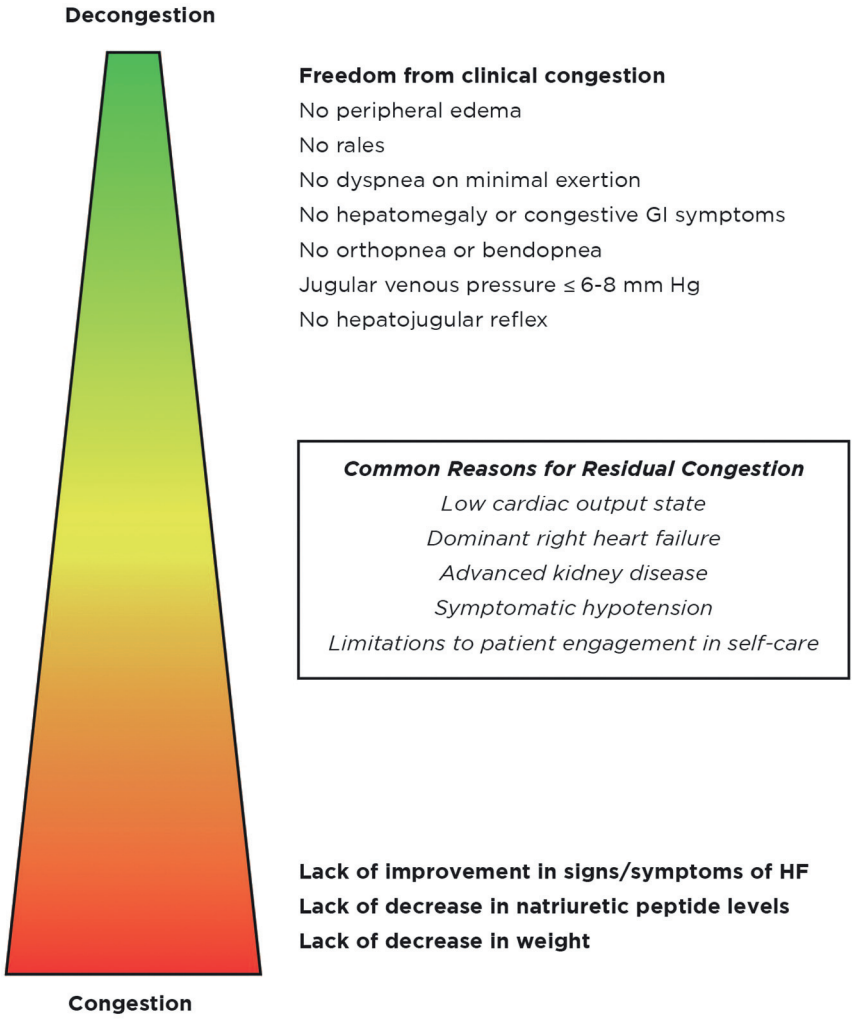
Establishing an effective diuretic regimen is crucial for achieving decongestion. The first doses, often given in the ED, are generally based on previous home loop diuretic doses, as in the DOSE (Diuretic Optimization Strategies Evaluation) trial.[47] For patients who have been on loop diuretic therapy as outpatients, the total daily dose should be changed to an oral furosemide equivalent and administered IV at 1 to 2.5 times the total daily dose. For those patients who have not been on diuretic agents as outpatients, the initial furosemide dose can vary according to patients' fluid overload, kidney function, and age, and usually is around 40 to 80 mg IV daily. In the DOSE trial, initiation of the 2.5 times higher dose led to 37% more fluid loss, 43% more weight loss, and greater dyspnea relief by 72 hours.[47] IV diuretic agents are usually continued throughout the early hospital stay, either by IV bolus every 8 to 12 hours or by continuous IV infusion. Table 7 describes diuretic agent dosing in hospital.

TABLE 7 Diuretic Dosing

Class	Drug	Usual Inpatient Dosing* (Maximum)†	Usual Outpatient Dosing (Maximum)†
Loop diuretic agents	Bumetanide	0.5-4 mg IV 1-3 times daily (12 mg/d) OR 0.5-2 mg/h IV infusion (4 mg/h)	0.5-2 mg orally once to twice daily (10 mg/d)
	Furosemide	40-160 mg IV 1-3 times daily (200 mg/dose) OR 5-20 mg/h IV infusion (40 mg/h)	20-80 mg orally once to twice daily (600 mg/d)
	Torsemide	Not available commercially in IV form‡	10-40 mg orally once daily (200 mg/d)
Carbonic anhydrase inhibitors	Acetazolamide	500 mg orally/IV once daily or in divided doses	N/A
Thiazide-type diuretic agents	Chlorothiazide	0.5-1 g IV once to twice daily (2 g/d)	N/A
	Hydrochlorothiazide	25-50 mg orally once to twice daily (100 mg/d)	25-50 mg orally once daily (100 mg/d)
	Chlorthalidone	12.5-25 mg orally once to twice daily (100 mg/d)	25-50 mg orally once daily (100 mg/d)
	Metolazone	2.5-5 mg orally once to twice daily (20 mg/d)	2.5-5 mg orally once daily (20 mg/d)

*For patients receiving loop diuretic agents before 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 based on observational data and clinical experience.
‡Oral therapy may be initiated before discharge to assess patient response.
IV = intravenous; N/A = not applicable.

FIGURE 5 Evaluation of Initial and Residual Congestion



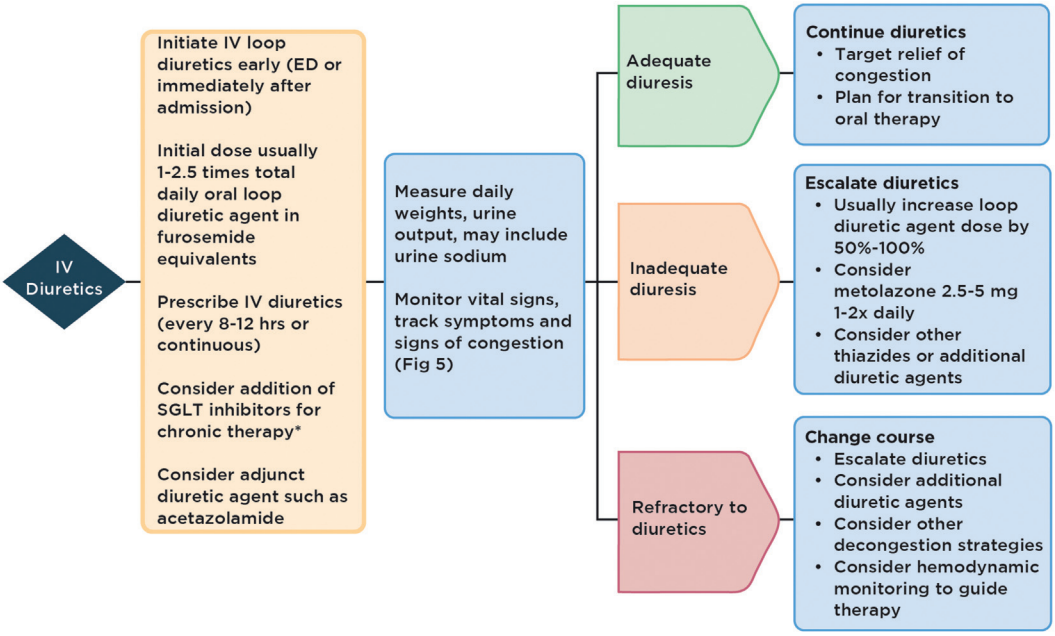
GI = gastrointestinal; HF = heart failure.

Table 7A provides guidance on timing and dosing for initiation of sacubitril/valsartan based on patient profile as represented in the randomized ARNI trials.

Subsequent IV diuretic doses are modified to achieve the desired daily response, which depends on the estimated total volume excess, the likely refill rate into the central venous compartment, kidney function, hemodynamic profile, comorbidities, and serum electrolytes. Doses are often doubled until reaching 400 to 500 mg of furosemide equivalent per day. When the response is brisk but transient, the frequency should be increased to 3 to 4 times daily. The DOSE trial did not demonstrate improved outcomes with continuous infusion of IV furosemide, but patients with chronic furosemide equivalent doses of over 240 mg daily were excluded.[47] A Cochrane review suggested that loop diuretic agent infusions may produce greater net diuresis and better tolerability at equivalent doses compared with bolus dosing.[48]

Measurement of urine sodium shortly after the start of therapy can be useful to evaluate diuretic responsiveness. A spot urine sodium content <50 mEq/L at 2 hours after the diuretic dose is correlated with a poor diuretic response and suggests consideration of dose

FIGURE 6 Diuretic Therapy



This figure has been updated to include information from recent clinical trials. When high loop diuretic doses are not effective, dual nephron blockade with the addition of either a thiazide-like diuretic or a carbonic anhydrase inhibitor (acetazolamide) can be considered. In the CLOROTIC (Safety and Efficacy of the Combination of Loop with Thiazide-type Diuretics in Patients with Decompensated Heart Failure) trial, addition of hydrochlorothiazide to an IV loop diuretic resulted in an increased diuretic response and more weight loss without a change in patient-reported dyspnea.[52] Metolazone can be added to the IV loop diuretic at 2.5- to 5-mg doses once or twice daily. In the ADVOR (Acetazolamide in Decompensated Heart Failure with Volume Overload) trial, acetazolamide added to a loop diuretic for 3 days exhibited a greater rate of successful decongestion and a shorter duration of hospital stay compared with placebo, with no statistically significant differences in renal safety, hypokalemia, or hypotension between groups. *If plan to continue SGLT inhibitors after discharge, determine financial feasibility. BP = blood pressure; ED = emergency department; IV = intravenous; Na = sodium; SGLT = sodium-glucose cotransporter.

escalation; higher levels predict a good response.[49,50] The recent randomized PUSH-AHF (Pragmatic Urinary Sodium-based algoritHm in Acute Heart Failure) trial tested this strategy and showed greater natriuresis when diuretic agents were guided by spot urine sodium concentrations, without a difference in mortality or HF rehospitalization.[51]

When high furosemide doses are not effective, dual nephron blockade with the addition of either a thiazide-like diuretic or a carbonic anhydrase inhibitor (acetazolamide) can be considered. In the CLOROTIC (Safety and Efficacy of the Combination of Loop with Thiazide-type Diuretics in Patients with Decompensated Heart Failure) trial of patients with acute HF, the addition of hydrochlorothiazide to an IV loop diuretic resulted in an increased diuretic response and more weight loss without a change in patient-reported dyspnea.[52] Those receiving hydrochlorothiazide exhibited increases in serum creatinine, hypokalemia, and impaired kidney function compared with placebo, but the study was stopped prematurely due to slow enrollment, making clinical safety conclusions difficult to assess.[52] IV chlorothiazide has a short elimination half-life but it is substantially more expensive, and observational data suggest that it does not increase net 24-hour urine output when compared with oral metolazone.[53,54] Metolazone can be added to the IV loop diuretic at 2.5- to 5-mg doses once or twice daily. In the ADVOR (Acetazolamide in Decompensated Heart Failure with Volume Overload) trial, acetazolamide added to a loop diuretic for 3 days exhibited a greater rate of successful decongestion (42.2% vs 30.5%; $P < 0.001$) and a shorter

duration of hospital stay (8.8 days vs 9.9 days; $P = 0.016$) compared with placebo, with no statistically significant differences in renal safety, hypokalemia, or hypotension between groups.

In a prespecified analysis of ADVOR, acetazolamide was associated with modestly higher creatinine levels during decongestion in the overall cohort and specifically in the HFrEF cohort.[55] Carbonic anhydrase inhibition affects multiple aspects of CO_2 exchange and should not be used chronically for HF. In a meta-analysis of 9 studies with a total of 229 patients with HF, acetazolamide was associated with significant decreases in serum pH, pCO_2, and serum bicarbonate.[56] Kidney function and serum bicarbonate should be monitored if acetazolamide is used for dual nephron blockade for longer durations, because plasma bicarbonate can fall dramatically and lead to severe acidosis, particularly in elderly patients with renal insufficiency.

Ongoing assessment of the extravascular and intravascular volume compartment and the "refill rate" between them is key to avoiding "overshooting" during diuresis. Excessive volume depletion can decrease tolerance to renin-angiotensin system (RAS) antagonism, particularly with ARNI administration, during which vasodilation may further decrease cardiac preload. These considerations necessitate continued close tracking of congestion and decongestion throughout the HF hospitalization. Postural vital signs can help assess both volume status and tolerance of vasodilation and are particularly useful when finalizing the discharge regimen.

Background therapy with an SGLT inhibitor can enhance diuretic efficiency. Although this may increase urine output[13] and improve decongestion,[14,57,58] initiation of SGLT inhibitor during hospitalization should prompt reassessment of the diuretic regimen planned for discharge.

Although **Figure 5** remained unchanged since the previous version, larger patient series have since confirmed the importance of achieving decongestion before hospital discharge, as demonstrated in the ESC-EORP HFA registry following HF hospitalization.[59] In this study from 21 countries with intentional selection of different center types, 7,865 patients were classified in terms of admission and discharge hemodynamic profiles. "Wet and warm" profiles were present in 70% of patients at admission and 31% of patients at discharge, with 5.6% of patients discharged with "cold-wet" profiles. Moderate to severe tricuspid regurgitation, diabetes, and worse chronic New York Heart Association (NYHA) class predicted higher risk of congestion at discharge, which was associated with 1-year mortality of 28% compared with 18.5% without congestion ($P < 0.001$). The likelihood of being discharged without congestion was higher in patients with de novo HF.[59]

To better interpret clinical congestion, serum albumin levels should be checked routinely at HF admission. Peripheral edema is a major component of congestion but should be interpreted with caution in the presence of hypoalbuminemia, which has been associated with up to 6-fold higher in-hospital mortality.[60] Reduced plasma oncotic pressure from hypoalbuminemia enhances extravascular fluid accumulation, for which overzealous diuresis may deplete intravascular volume and decrease renal function and blood pressure, particularly with nephrotic syndrome or chronic malnutrition (**Figure 6**).

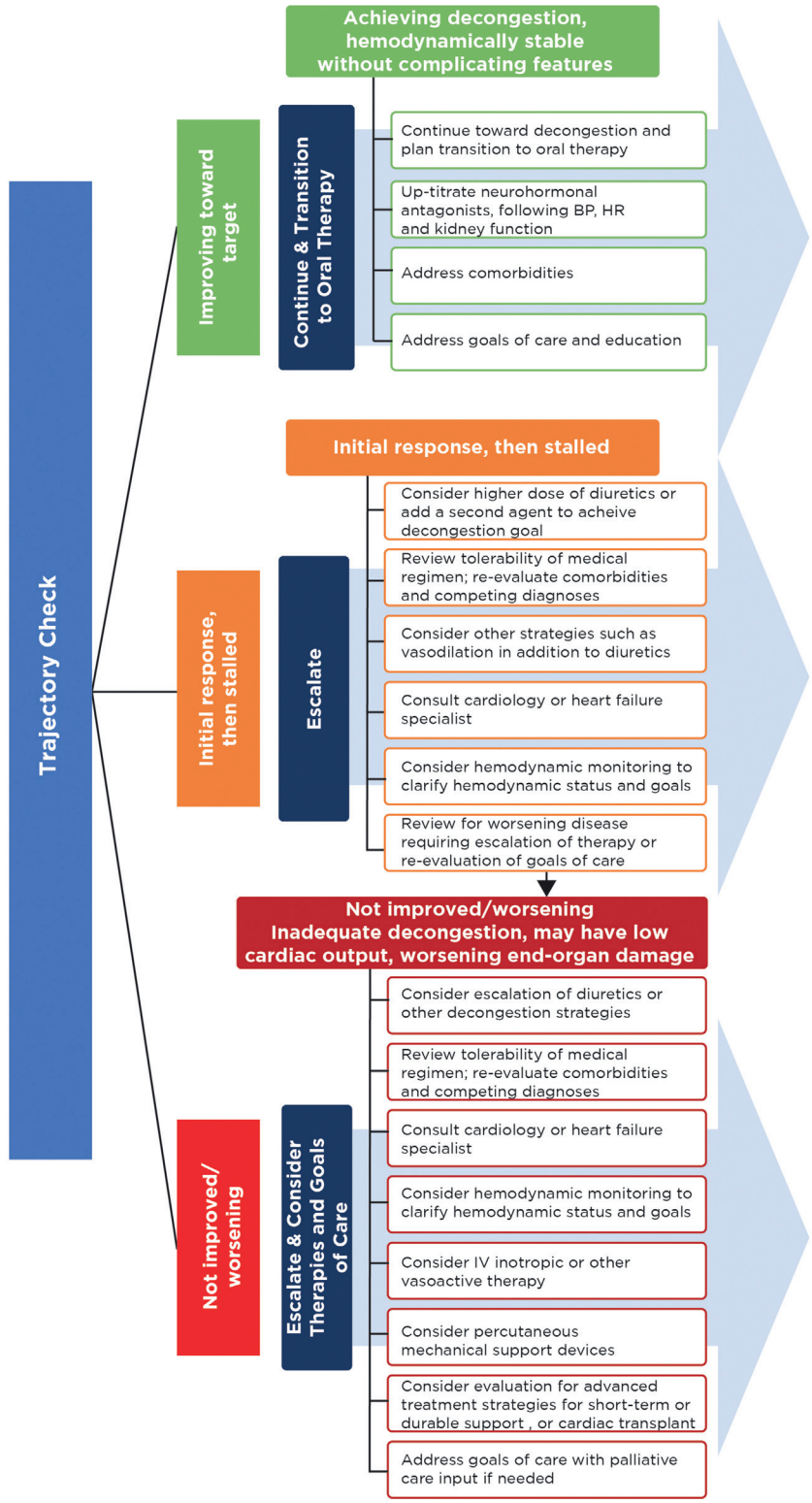
2.4. Clinical Trajectories

Clinical trajectories are primarily defined by the pace and extent of decongestion along with hemodynamics. In patients who are responding well to diuresis and are hemodynamically stable, GDMT should be up-titrated as tolerated, aiming to implement all 4 components of GDMT for HFrEF (**Figure 7A**). Comorbidities should be evaluated and addressed, and discharge planning should be accelerated. IV diuretic agents should generally be continued until optimal decongestion is achieved, and patients should then transition to the oral dose estimated for maintenance.

Careful assessment of residual volume reservoirs and jugular venous pressure elevation is needed to avoid intravascular volume depletion, which can cause hypotension and intolerance to RAS antagonists, particularly an ARNI. In the large Optum database, there was a higher rate of early mortality and readmission in the 19% of patients who had IV diuretic agents stopped (for an average of 3 days) and then restarted before discharge.[5] This underlines the importance of mapping the clinical trajectory of diuretic therapy and decongestion during and after HF hospitalization.

In patients who respond initially but then stall, who fail to respond, or who worsen, intensification of the diuretic regimen may be needed, along with reevaluation of medications and comorbidities, with the consideration that a diagnosis other than HF may be dominant. Invasive monitoring and/or the addition of vasodilators or inotropic therapy may be considered, usually best guided by subspecialty consultation. A trajectory of worsening HF warrants discussion of goals of care, which, for some patients, may be facilitated by a palliative care team, where available.

FIGURE 7A **Clinical Trajectories in Patients With HF**



The clinical trajectory during hospitalization reflects responsiveness to therapy. Three main in-hospital trajectories have been defined according to changes in patient symptoms, clinical signs, laboratory markers, hemodynamics, and complications: 1) *improving toward target*; 2) *initial response, then stalled*; or 3) *not improved/worsening*. This figure combines the 3 trajectories into 1 graphic. BP = blood pressure; HR = heart rate; IV = intravenous.

2.5. Optimizing Neurohormonal Modulators During Hospitalization for HFrEF

The ACC/AHA/HFSA guidelines for HF were updated in 2022. The 2021 ECDP on optimization of GDMT for chronic outpatient HFrEF[9] and the 2023 ECDP on management of chronic HFpEF[10] outline strategies for initiation and titration of GDMT in HF. The bases of evidence for these chronic HF therapies have been derived primarily from stable patients in outpatient settings without acute decompensation, usually already on 1 or more recommended medications. There are limited data comparing the order of addition of therapies, particularly for patients with de novo HFrEF. Hospitalization for HF identifies a group at higher risk for mortality, which ranges from 20% to 35%[59,61] at 1 year and up to 50% at 2 years, compared with <10% at 1 year and <20% at 2 years for many recent outpatient HF trials. This section will focus primarily on new evidence for initiation and titration of recommended outpatient medications during HF hospitalization. As shown in **Figure 1**, there is new emphasis on the systematic introduction of all 4 major classes of therapy for HFrEF after stabilization, along with diuretic agents as needed to relieve and prevent congestion. In addition to a reduction in events in the first months after discharge, in-hospital initiation of these agents is linked to higher likelihood that they will be prescribed 12 months after discharge.[57,61]

As discussed earlier, initiation of SGLT inhibitors is now recommended in all outpatients with HF, and newer evidence indicates that they enhance decongestion during hospitalization.[62] Mineralocorticoid receptor antagonists are recommended to decrease hospitalizations and mortality for outpatients with HFrEF and serum creatinine <2.5 mg/dL in men and ≤2.0 mg/dL in women with stable potassium handling and anticipated access to frequent monitoring of electrolytes after discharge. Although the benefit of MRAs has been clearly established for outpatients with Stages B and C HFrEF, no specific benefit has been demonstrated from initiation during hospitalization. A randomized blinded trial showed that 100 mg of spironolactone compared to 0 to 25 mg spironolactone for 96 hours after admission did not increase acute diuresis, relieve clinical congestion, or decrease natriuretic peptide levels in hospital, but was shown to be safe in the hospital setting,[63] where it can be initiated early in the absence of contraindications.

Since 2000, the major reduction in HF hospitalization and mortality and increased remission to improved LVEF has been attributed to beta-blockers and RAS inhibitors, both of which were shown in early studies to improve outcomes when initiated during hospitalization.[57,64] On top of these pillars, the addition of neprilysin inhibition to RAS inhibition with the angiotensin receptor antagonist valsartan has further decreased hospitalizations and mortality compared with enalapril over 2 years in the outpatient setting.[16] After stabilization during hospitalization, sacubitril/valsartan further decreased natriuretic peptide levels compared with enalapril during 8 to 12 weeks, with a trend for early reductions in hospitalization and mortality.[65,66]

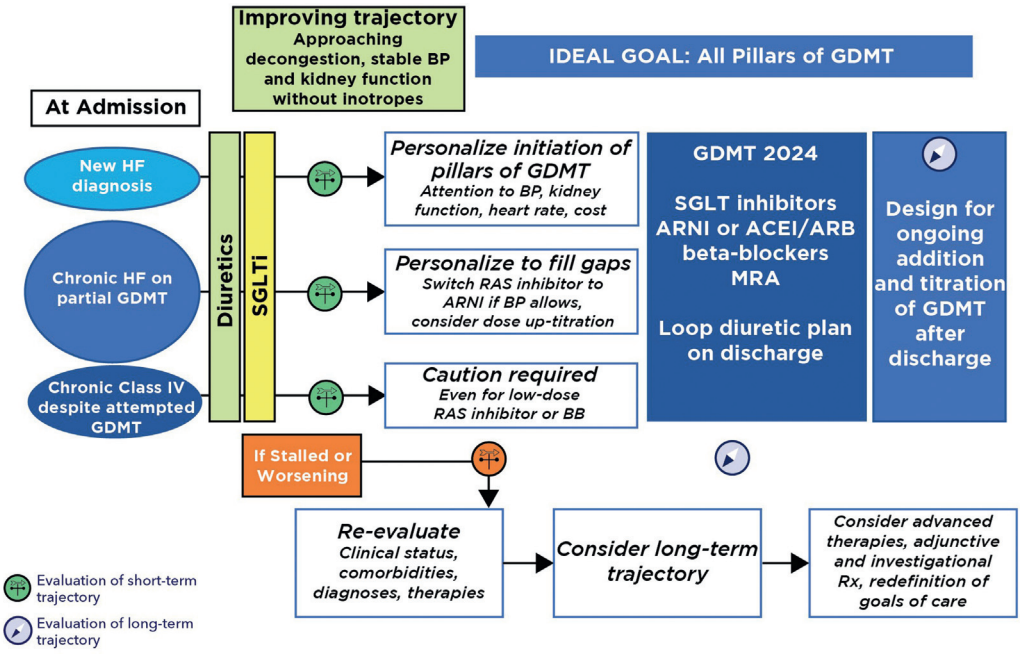
Neuromodulation through inhibition of the RAS, neprilysin, and the beta-adrenergic systems has potent effects on the circulation and requires careful titration compared with the standard dose regimens for SGLT inhibitors and MRAs, which have little or no effect on reducing blood pressure or cardiac output. General criteria for stability in hospital for neurohormonal modulation have included SBP of at least 100 mm Hg without IV inotropic therapy in the past 24 hours or an increase in IV diuretic agents or vasodilators in the past 6 hours. The steps to optimization of neurohormonal modulation depend upon the in-hospital course and the patient position along the HF journey (**Figure 7B**).

2.5.1. New Presentation of HFrEF

A new diagnosis of HFrEF may be present in a third or more of patients hospitalized with HF.[67-69] Compared with decompensation of chronic HF, new-onset (or "de novo") HF usually presents at a younger age and with fewer comorbidities; congestive symptoms are similar, but rales are somewhat more common.[68,70] Rates of discharge with residual congestion, early readmission, and mortality at 1 and 6 months are lower for new-onset HF, excluding the few patients presenting initially with cardiogenic shock. The better outcomes may reflect not only the earlier stage of disease but also the imminent impact of therapies to improve ventricular remodeling and ejection fraction.

Ideally, patients with new HF would receive all "pillars" of HF therapy during the initial hospitalization, including SGLT inhibitors, a diuretic plan, and the 3 neurohormonal modulators. However, combining multiple therapies rapidly may require distribution in a "spending function,"[71] due to effects on blood pressure. Although 20% to 40% of patients admitted with new-onset HF may be on ACE inhibitors/ARBs or beta-blockers for other reasons,[67,72] most patients with newly diagnosed HF are not on neurohormonal modulators. Assuming that SGLT inhibitors and MRAs are started early, there is little information to specify the order of addition for other neurohormonal modulator classes after stabilization. Sacubitril/valsartan provides inhibition of neprilysin and the RAS. The PIONEER-HF trial included 34% of patients with a new HF diagnosis (mean SBP 118 mm Hg),[67] and showed substantial benefit for sacubitril/valsartan over enalapril for

FIGURE 7B **Titration of GDMT in HFrEF**



Titration of GDMT in HFrEF by initial presentation and trajectory. Patients with decompensated HFrEF should be diuresed and started on SGLT inhibitor unless contraindicated or cost prohibitive. Those with an improving trajectory (denoted by the green weathervane icon) should have optimization of GDMT. Patients may have a new HF diagnosis, in which case initiation of all 4 pillars of GDMT should be attempted. Patients with chronic HF on partial GDMT should have personalized therapy to fill in gaps, considering a switch from an ACE inhibitor/ARB to ARNI if appropriate. Caution is required for patients with chronic Class IV HF with decompensated HF; these patients may not tolerate even low doses of beta blockers and RAS, although an attempt at titration may be made. Patients whose short-term trajectory is stalled or worsening (denoted by the orange weathervane icon) should have re-evaluation of comorbidities and consideration of other diagnoses. The long-term trajectory (denoted by the compass icon) should be reevaluated, with consideration of goals of care, candidacy for advanced therapies, and experimental treatments. The ideal goal is initiation of all 4 pillars of GDMT for HFrEF in the hospital on a baseline of diuretic therapy. A plan for ongoing addition and titration of GDMT after discharge should be fashioned as well. ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor/neprilysin inhibitor; BB = beta-blocker; BP = blood pressure; d/c = discharge; GDMT = guideline-directed medical therapy; MRA = mineralocorticoid antagonist; RAS = renin-angiotensin system; SGLT = sodium-glucose cotransporter.

reductions in both N-terminal pro-B-type natriuretic peptide and the cumulative incidence of rehospitalizations or cardiovascular death by 8[67] and 12 weeks.[65] For some patients with less robust blood pressure, the first step may be a low dose of an ARB, with a potential switch to sacubitril/valsartan before discharge, once a low dose of beta-blocker has been tolerated[62] (usually starting with metoprolol 6.25 mg twice per day or carvedilol 3.125 mg twice per day). Neurohormonal therapy is sometimes begun with a beta-blocker, particularly in young patients with resting tachycardia and presumed high sympathetic activation, because these patients may derive the greatest benefit if beta-blockade can be tolerated. There is no contemporary trial of initiation sequence, but the historical CIBIS III trial in outpatients demonstrated no difference in overall outcomes whether beta-blockers or ACE inhibitors were started during the first 6 months,[73] whereas a small trial showed a higher achieved beta-blocker dose, lower heart rate, and greater increase in LVEF at 12 months when beta-blockers were started first.[74]

The usual strategy aims for addition of all neurohormonal modulator classes before any up-titration. The European TRANSITION trial demonstrated that similar majorities of patients were on 49/51 mg to 97/103 mg twice-daily doses of sacubitril/valsartan at 10 weeks, whether initiated shortly before or very early after discharge, with higher doses achieved in patients with *de novo* heart failure, a history of hypertension, and higher starting doses.[75] Extensive experience in the United States indicates that prescription of recommended medications at discharge increases the likelihood that they will be successfully maintained for chronic therapy, and 4-pillar GDMT has been shown to decrease recurrent HF events, reverse ventricular remodeling, and extend meaningful survival.[57,61]

2.5.2. Chronic HF on Partial GDMT

Most HF hospitalizations occur in patients with exacerbations of HF that have been previously diagnosed and partially treated, as seen in 66% of patients in the PIONEER-HF trial. Vigorous quality improvement initiatives have consistently been highly successful for increasing the use of recommended therapies for HFrEF between admission and discharge, with patient eligibility rates varying with comorbidities.[76] From the Get With the Guidelines-Heart Failure Registry data[61] for 50,170 Medicare patients hospitalized from 2017 to 2020 with HFrEF and diabetes, patients were deemed eligible for a mean number of 3.9 medications and were receiving 2.1 ± 1.3 at admission and 3.0 ± 1.0 medications at discharge, for a net gain of 0.9 medications over a hospital stay averaging 5.6 ± 5.3 days. Of the 70% of patients deemed eligible for ARNIs/ACE inhibitors/ARBs, 59% received them on admission and 91% on discharge. For MRAs, 50% of patients were considered eligible, with 26% on MRAs at admission and 56% at discharge.[61] Omission of MRAs in some medically eligible patients may reflect perceived obstacles to the recommended frequent monitoring of electrolytes and kidney function.

Once the trajectory check indicates favorable progression, those patients hospitalized with chronic HFrEF, as for new HFrEF, should be targeted for addition of the remaining GDMT components as recommended. For patients already on RAS inhibitors but not beta-blockers, a beta-blocker will generally be initiated at a low dose if not contraindicated. For patients with HFrEF without ARNI therapy or contraindications, initiation should be considered if financially feasible. Additional effects of ARNI, such as increased levels of natriuretic peptides, may decrease intravascular pressures more than ACE inhibitors/ARBs, so optimal initiation may be when patients are at the mid-to-upper range of normal filling pressures. Previous IV inotropic therapy (used in 7.7% of patients enrolled in PIONEER-HF) should have been successfully discontinued at least 24 hours before starting sacubitril/valsartan. Patients already on an ARB can be transitioned directly to ARNI, whereas ACE inhibitors should be discontinued for at least 36 hours before initiating ARNI. For patients in whom ARNI is not feasible, optimization of GDMT may include increased dosages of RAS inhibitors. In patients for whom kidney function limits the use of any RAS inhibitor, hydralazine and nitrates can be considered. Outpatients who self-reported as Black or African-American had remarkable improvements in quality of life, hospitalization, and survival with the addition of hydralazine/nitrate combinations to the central pillars of therapy, but attention should be paid to blood pressure and the frequent side effect of nausea, which may appear early or months after initiation of hydralazine.

2.5.3. Chronic NYHA Functional Class IV HF With Prior GDMT

The prior discussions apply to patients with a trajectory of improvement toward the goal of decongestion and stabilization (**Figure 5**). Some patients whose hospital course is stalled or worsening may nonetheless improve with further intervention and reach a favorable trajectory. Whereas SGLT inhibitors are likely to be widely tolerated, and MRA tolerated in patients with adequate kidney function, the addition of ARNIs/ACE inhibitors/ARBs or beta-blockers is generally not attempted in patients in stalled or worsening trajectories. In some patients, decreased dosages or discontinuation of these classes of medications may be required to achieve adequate blood pressure and kidney function. Neurohormonal antagonists are usually discontinued in patients for whom IV inotropic support is used to increase cardiac output or pressors to increase blood pressure.

The highest-risk contemporary population for RAS inhibitor therapy is exemplified in the LIFE trial, the purpose of which was to determine the tolerability and efficacy of sacubitril/valsartan therapy vs valsartan therapy in patients with recent Class IV symptoms of advanced HF (**Table 7A**). Enrollment required Class IV symptoms at screening or within the previous 3 months, with a minimum of 3 months of guideline-directed therapy with RAS inhibitors, beta-blockers, and MRAs or intolerance to such therapy. Inclusion criteria required 1 additional high-risk feature, most commonly recent inotropic therapy, 1 or more other HF hospitalizations in the past 6 months, or LVEF <0.25.

In the LIFE trial, the mean duration of HF before enrollment was 6 years, with average LVEF 0.20. By design, the inclusion criteria defined a population with more severe chronic illness than previous trials, with a lower limit of SBP of 95 mm Hg, and patients could be receiving inotropic therapy. The trial included a run-in with sacubitril/valsartan, before which an ACE inhibitor or ARB had been taken by 72% of patients, with the remainder deemed intolerant. The run-in period for all enrolled patients was 3 to 7 days with the lowest dose (24-26 mg of sacubitril/valsartan), which 18% of patients did not tolerate. For the patients randomized after the run-in period, there was no trend seen for benefit of sacubitril/valsartan to reduce N-terminal pro–B-type natriuretic peptide levels, readmission, or death compared to valsartan.

2.5.4. Populations, Blood Pressure, and Dosing for Sacubitril/Valsartan

Individualization of RAS inhibition should be guided by the clinical trajectory before and during the hospitalization. To guide timing and dosing for initiation of sacubitril/valsartan, the patient profile should be compared to those represented in the randomized ARNI trials, as

shown in Table 7A. These trials in HFrEF define a spectrum extending from primarily Class II outpatient HF to late stages of chronic HF with Class IV symptoms and other high-risk features despite previously attempted recommended therapies (Table 7A). Symptomatic hypotension is more common with sacubitril/valsartan than with the other RAS inhibitors, particularly in combination with beta-blockers. Inclusion in the PARADIGM trial required tolerance of a beta-blocker and an ACE inhibitor equivalent of at least 10 mg enalapril for 4 weeks before the trial and a successful run-in period of 2 weeks of enalapril 10 mg twice daily and then 200 mg increasing to 400 mg sacubitril/valsartan. After the exclusion of 20% of patients during the run-in phase, symptomatic hypotension still occurred with both sacubitril/valsartan and with enalapril, particularly in patients with baseline SBP <110 mm Hg (25.5% with sacubitril/valsartan and 13.7% with enalapril), but sacubitril/valsartan showed benefit over enalapril in all blood pressure groups.[77]

At the late stage of the HFrEF spectrum, the LIFE trial portrays the patient who is not receiving intensive care but is nonetheless at high risk for intolerance of any RAS inhibitor. As shown in Table 7A, patients in the LIFE trial had long duration of HF, frequent hospitalizations, poor quality of life, high diuretic doses, noncontinuation of previous RAS inhibitor therapy, and lower blood pressures and kidney function than the typical patient hospitalized with HF.[78] Failure to tolerate the 24/26 mg dose for 3 to 7 days during run-in was usually due to hypotension and was more common in patients who had discontinued an ACE inhibitor/ARB before enrollment, had lower blood pressure, required insulin, or had moderate-severe valvular regurgitation.[79] After randomization, SBP was ≤85 mm Hg with sacubitril/valsartan in 17% of patients and in 12% of patients on valsartan alone. Within the next 6 months, 26% of patients in the sacubitril/valsartan group and 21% in the valsartan group had at least 1 week of inotropic infusion, 8% in the sacubitril/

TABLE 7A Sacubitril Valsartan Populations and Dosing

Name of Trial	PARADIGM n = 10,513 Run-in Randomized n = 8,442	PIONEER-HF No Run-in Randomized n = 881	LIFE Passed Run-in n = 372*	LIFE Failed Run-in n = 73
Patient setting	Stable outpatient	Hospital median 68 h after admission	In or out of hospital	In or out of hospital
LVEF	0.30 ± 0.06	0.24 (0.18-0.30)	0.20 ± 0.07	0.21 ± 0.06
Heart failure diagnosis	4 wks stable dose beta-blocker and ACE inhibitor	34% first diagnosis of HF	≥3 mo 4.8 y	≥3 mo 5.1 y
NYHA functional class II/III/IV %	71/23/0.7	23/64/9	Required class IV in past 3 mo on GDMT† NYHA at run-in 10/31/58 IV	Required class IV in past 3 mo on GDMT† NYHA at run-in 5/30/64 IV
KCCQ Overall Score	73 ± 19		53 ± 23	46 ± 23
NT-proBNP pg/mL	1,631	2,883	3,210	6,377
Previous diuretic agent	80%	60%	93%	95%
Baseline equivalent furosemide/d	48 mg		128 ± 121 mg	157 ± 124 mg
Recent ACE inhibitor/ARB	100%	47%	53%†	27%†
Recent beta-blocker	93%	60%	81%†	64%†
Recent inotropic therapy	0	7.7%	20%	30%
SBP, mm Hg	122 ± 15	122 ± 18	113 ± 15	109 ± 14
Creatinine mg/dL	1.13 ± 0.03	1.28 (1.07-1.51)	1.35 ± 0.4	1.55 ± 0.5
Starting dose of ARNI	4 -6 wks run-in, 49/51 and then 97/103 twice daily	24/26 mg if SBP <100-120 mm Hg 49/51 mg if SBP >120 mm Hg	24/26 mg if on 0-10 mg equivalent lisinopril or eGFR <30 49/51 mg if higher ACE inhibitor/ARB	Run-in 24/26 mg twice daily for 3-7 days
Comparator	Enalapril 10 mg twice daily	Enalapril 2.5 mg twice daily or enalapril 5 mg daily	Valsartan 40 mg or 80 mg twice daily	No comparator for open label run-in. 78% discontinuations for hypotension

*335 of the 372 patients in LIFE who passed run-in were randomized.
†Inclusion criteria required to have been on GDMT: ACE inhibitor/ARB, beta-blocker, and MRA or intolerant.

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor/neprilysin inhibitor; eGFR = estimated glomerular filtration rate; KCCQ = Kansas City Cardiomyopathy Questionnaire; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-B-type natriuretic peptide; SBP = systolic blood pressure.

valsartan group and 5% in the valsartan group died, and transplant/left ventricular assist device implantation was done in 9% in the sacubitril/valsartan group and in 8% in the valsartan group. This trial, sponsored by the National Heart, Lung, and Blood Institute, was conducted in academic centers, the majority offering heart transplant and mechanical circulatory support, and as such, this trial is less representative of hospitalized HF than exemplified in PIONEER-HF.

In an analysis of almost 100,000 patients discharged with HFrEF between 2006 and 2018 in Get With The Guidelines-Heart Failure registry,[61,65,80] the actionable criteria for sacubitril/valsartan initiation (which excludes the trial requirement for elevated natriuretic peptide levels) applied to about 70% of patients hospitalized with HFrEF. Typical of patients hospitalized with HF, mortality at 1 year was 36.7% in this overall cohort, highlighting the importance of early optimization of contemporary therapies for HFrEF to improve survival for hospitalized patients.

2.6. Transitions of Care

Figure 10 has been revised from the original document with minor changes aimed at improving clarity and applicability.

FIGURE 10 Education for Patients, Families, and Caregivers

EDUCATION FOR PATIENTS, FAMILIES, AND CAREGIVERS

- ☐ Current medications
  - Indication
  - Dose/frequency
  - Potential side effects
  - Potential adherence barriers
- ☐ Activity level
- ☐ Dietary sodium restriction _____mg/day
- ☐ Fluid restriction ☐ Yes _____L/day or ☐ No
- ☐ Daily weight monitoring
  - Has scale ☐ Yes ☐ No
  - Records daily weights ☐ 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

  _____
- ☐ Diuretic management plan
- ☐ Plans for continuation of care
  - Cardiology specialty clinic follow-up appointment _____/_____/_____

L = liter; ICD = implantable cardioverter-defibrillator.

FIGURE 11 Model Focused Discharge Handoff

FOCUSED DISCHARGE HANDOFF

Name_____ Age_____ MRN_____ Admission Date___/___/___Discharge Date___/___/___

HF TYPE: ☐ HFrEF ☐ HFpEF (≥50) ☐ HFmrEF (41-49) Last LVEF_____

HF ETIOLOGY: ☐ Ischemic ☐ Non-isch ☐ Other_____

Arrhythmia history ☐ VT ☐ AF ☐ Other Device type_____

Triggers for Hospitalization

_____

_____

CONDITION AT DISCHARGE: ☐ Discharged to Rehab or other facility

D/C BP___/___ HR ___ Rhythm ☐ Sinus ☐ AF ☐ paced **Weight** At D/C ___lbs Est. target weight ___lbs

Residual congestion at D/C ☐ **Y** ☐ **N** **Edema 0-4+**_____ **JVP**_____ **Rales** ☐ **Y** ☐ **N** **Ascites** ☐ **Y** ☐ **N**

If still wet, limited by ☐ Dominant R heart failure ☐ Kidney function ☐ Hypotension ☐ Other

Kidney Function Discharge BUN/Cr_____ eGFR_____ Worst CR in hospital_____ Baseline_____

Biomarkers Admission BNP/NT-proBNP _____ Discharge BNP/NT-proBNP (if known)_____

Comorbidites: _____ Labs to Check ☐ **K+** ☐ **Cr** ☐ **BNP** ☐ **TSH** ☐ **Iron** ☐ **Other** _____

Psychosocial Factors _____

Other hospital events ☐ code ☐ sepsis ☐ dialysis ☐ intubation IV inotropes used? ☐ **Y** ☐ **N**

Code Status ☐ Full code ☐ Full code but discussed ☐ DNR/DNI ☐ DNI only ☐ *POLST* ☐ *Needs discussion*

DISCHARGE HF MEDICATIONS:

Diuretic Loop_____dose/freq_____. Metolazone dose/freq_____ Other: dose/freq_____

In-hospital effective loop dose ___mg IV ☐ Daily ☐ BID ☐ TID ☐ Drip at ___mg/hr Metolazone used? ☐ **Y** ☐ **N**

Triggers for rescue dose: If _____lbs up, or_____(sentinel symptom)

Rescue dose _____orally and/or metolazone _____for_____days before recheck

K+ replacement_____meQ/day Plan for more K+ with rescue dose? ☐ **Y** ☐ **N**

GUIDELINE DIRECTED MEDICAL THERAPY:

RAS meds ACEI_____mg/day ARB_____mg/day ARNI _____mg/day Dose ↓in hospital? ☐ **Y** ☐ **N**

If not given or dose ↓, why? ☐ Hypotension ☐ Dizziness ☐ Kidney fx ☐ Cough

If no ARNI, why? ☐ Hypotension ☐ Cost ☐ Hyperkalemia ☐ Angioedema

Beta-blocker_____mg/day Dose ↓ in hospital? ☐ **Y** ☐ **N**

If not given or dose ↓, why? ☐ Hypotension ☐ bradycardia

MRA ☐ **Y** ☐ **N** If not, why not? ☐ Hypotension ☐ Kidney function ☐ Hyperkalemia

SGLTi ☐ **Y** ☐ **N** If not, why not? ☐ Kidney function ☐ DKA ☐ UTI/GMI ☐ Cost

PLAN

☐ **Start** ☐ **Titrate**
☐ △ **ARNI**

☐ Start ☐ Titrate

☐ Start ☐ Titrate
☐ Start ☐ Continue

Other HF meds Hydral/nitrates ☐ **Y** ☐ **N** Ivabradine ☐ **Y** ☐ **N** Digoxin ☐ **Y** ☐ **N** Inotropes ☐ **Y** ☐ **N**

- Anticoagulation *for* ☐ AF ☐ DVT/PE ☐ Mech valve ☐ LV thrombus ***with*** ☐ DOAC ☐ Warfarin
- Antiplatelet *for* ☐ ACS ☐ PCI ☐ Stroke/TIA ***with*** ☐ ASA ☐ Clopidogrel ☐ Prasugrel ☐ Ticagrelor
- Antiarrhythmic ☐ Amiodarone ☐ Dofetilide ☐ Sotalol ☐ Other _____

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

FOLLOW-UP: Follow-up team_____ Appt 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_____

Other clinicans_____ ☐ Research Study_____

Additional support needed for optimal care:_____

ACE = angiotensin-converting enzyme; ACS = acute coronary syndrome; AF = atrial fibrillation; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor/neprilysin inhibitor; ASA = acetylsalicylic acid; BID = twice a day; BNP/NT-proBNP = B-type natriuretic peptide/N-terminal pro–B-type natriuretic peptide; BP = blood pressure; BUN = blood urea nitrogen; CR = creatinine ratio; D/C = discharge; DKA = diabetic ketoacidosis; DNI = do not intubate; DNR = do not resuscitate; DOAC = direct oral anticoagulant; DVT/PE = deep vein thrombosis/pulmonary embolism; eGFR = estimated glomerular filtration rate; GMI = glomerular macrophage index; HF = heart failure; HFmrEF = heart failure with mid-range ejection fraction; HFpEF = heart failure with preserved ejection fraction; HFrEF = heart failure with reduced ejection fraction; HR = heart rate; IV = intravenous; JVP = jugular venous pressure; K+ = potassium; lbs = pounds; LV = left ventricular; LVEF = left ventricular ejection fraction; mg = milligram; MRA = mineralocorticoid antagonist; MRN = medical record number; PCI = percutaneous coronary intervention; POLST = physician orders for life sustaining treatment; proBNP = pro-B-type natriuretic peptide; PT = physical therapy; RAS = renin-angiotensin system; SGLT = sodium-glucose cotransporter; TIA = transient ischemic attack; TID = 3 times a day; UTI = urinary tract infection; VT = ventricular tachycardia.

FIGURE 12 Checklist for Communication to Continuing Care Providers

CHECKLIST FOR 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
  - ARNI/ACEI/ARB
  - Beta blockers
  - Aldosterone mineralocorticoid antagonists
  - SGLT inhibitor
  - Ivabradine
  - Hydralazine/isosorbide
- ☐ Adjustment of diuretics
- ☐ 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 OR AF
- ☐ Recommendations for when to assess response to therapy and in what venue (in person vs telehealth)
- ☐ COVID, Pneumovax and Influenza vaccination

FOLLOW-UP RELATED TO COMORBIDITIES

- ☐ Kidney function
- ☐ Diabetes
- ☐ Sleep-disordered breathing
- ☐ Depression
- ☐ Anemia
- ☐ IV iron given?
- ☐ 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

Advanced care plan ☐ Y ☐ N
Goals of care changed during hospitalization ☐ Y ☐ N

ACE = angiotensin converting enzyme; AF = atrial fibrillation; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor/neprilysin inhibitor; EP = electrophysiology; GDMT = guideline-directed medical therapy; IV = intravenous; SGLT = sodium-glucose cotransporter.

FIGURE 13 Checklist for Follow-Up Phone Call

CHECKLIST FOR FOLLOW-UP PHONE CALL

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 alternatives such as frozen fruit, etc
Follow-up	When is your follow-up appointment? and in what venue (in person, telehealth)? _____ If going in person, do you have a way to get there? _____	NO follow-up appt or no way to get there	
Physical Activity			Consider cardiac rehabilitation

FIGURE 14 First Post-Discharge Visit Checklist

FIRST POST-DISCHARGE VISIT CHECKLIST

☐ **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
  - AF
  - COPD
  - OSA
  - Others

☐ **Goals of Care changed**

☐ **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-proBNP
- 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?
- ARNI/ACEI/ARB
  - Dose optimized?
  - Eligible to switch to ARNI?
- Mineralocorticoid antagonist
  - Dose optimized?
- SGLT inhibitor
- Diuretic agents
  - Dose adjustment?
- Ivabradine? (Consider initiation if heart rate remains elevated despite beta-blocker optimization)

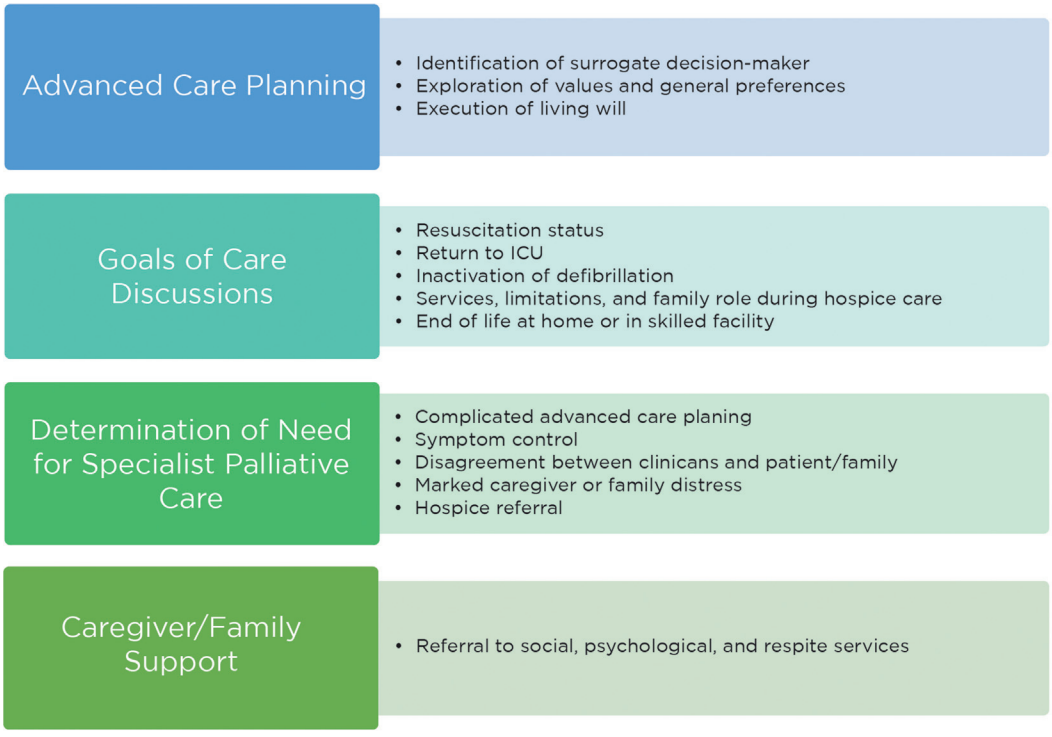
☐ **Interventional therapies (if applicable)**
- Revascularization
- CRT
- ICD
- AV node or AF ablation
- Valvular intervention
- Remote monitoring

☐ **Patient education**
- Importance of adherence
- Medication education
- Dietary education
- Activity education
- Smoking cessation
- Alcohol consumption
- Follow-up appointment scheduled
  - ☐ In-person
  - ☐ Telehealth

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

ACE = angiotensin-converting enzyme; AF = atrial fibrillation; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor/neprilysin inhibitor; AV = atrioventricular; BMI = body mass index; BNP = B-type natriuretic peptide; CKD = chronic kidney disease; COPD = chronic obstructive pulmonary disease; 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; SGLT = sodium-glucose cotransporter; TTE = transthoracic echocardiogram.

FIGURE 15 Common Aspects of Palliative Care During HF Hospitalization



This figure reflects recent data and publications, including:

1. A study demonstrating that a palliative care referral trigger tool decreased hospital readmission rates up to 90 days postdischarge while also increasing the completion of advanced directives.
2. The 2022 AHA/ACC/HFSA HF Guideline,[8] which advocates a need-based approach to palliative care, with emphasis on shared decision-making, caregiver and bereavement support, and hospice care.

HF = heart failure; ICU = intensive care unit.

Figure 11 has been revised from the original document to include SGLT inhibitors and a separate box to denote whether the outpatient plan is to start components of GDMT or titrate them as tolerated. Consideration of whether to switch from RAS inhibitors to ARNIs is also included in this box. An attempt has been made to streamline the form, and some additions have also been incorporated. Checkboxes for frequent laboratory tests are now included, and discharge to a rehabilitation or other facility is now an option.

It may be worth reiterating that this handoff form condenses the most useful information about a patient's baseline condition, HF treatment regimen, plans for titration of GDMT and barriers to that titration, rescue doses, and scheduled follow-up into a concise 1-page document. The initial completion of this form is intended to be a team effort, with contributions from different clinicians throughout the hospitalization, even before discharge. Completing the form at a follow-up visit or second admission is substantially easier, and the form should be equally useful.

Figure 12 has been revised from the original document to include SGLT inhibitors and consideration of vaccinations for COVID-19 as well as for pneumonia and influenza. Vaccination has been shown to result in marked improvement in mortality and hospitalization outcomes for patients with HF,[81-83] and all clinicians play a crucial role in influencing their patients to receive these important annual vaccinations.[84] The possibility of care using telehealth is also included.

Figure 13 has been slightly revised from the original document to include telehealth. In the previous version, a question about the patient's ability to attend the follow-up visit was included, whereas now, it is highlighted as an immediate cause for concern.

Figure 14 has been revised from the original document to include atrial fibrillation as a significant concomitant disease state. Additionally, SGLT inhibition has been

added to the medication section. The potential for telehealth follow-up is now included.

2.7. Palliative Care During HF Hospitalization

There is growing recognition of the importance of palliative care in the management of patients with HF and an emerging evidence base to support its routine incorporation.[85] A 2020 study demonstrated a significantly reduced hazard of multiple readmissions in a cohort who received palliative care consultations compared with usual care.[86] The AHA/ACC/HFSA HF guidelines advocate a need-based approach to palliative care, with emphasis on shared decision-making, caregiver and bereavement support, and hospice care.[8]

Figure 15 has been revised to include a more detailed description of aspects of palliative care during hospitalization, including advanced care planning, goals of care discussions, and caregiver/family support. In particular, the figure highlights specific areas to address in goals of care discussions and lists indications for specialist palliative care referral. A recent study demonstrated that a palliative care referral trigger tool decreased hospital readmission rates up to 90 days postdischarge while also increasing the completion of advance directives.[87]

PRESIDENT AND STAFF

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KEY WORDS ACC Expert Consensus Decision Pathway, diuretics, goals of care, heart failure, HFpEF, HFrEF, inpatient hospitalization, SGLT, trajectory

APPENDIX 1. AUTHOR RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (RELEVANT)—2024 ACC EXPERT CONSENSUS DECISION PATHWAY ON CLINICAL ASSESSMENT, MANAGEMENT, AND TRAJECTORY OF PATIENTS HOSPITALIZED WITH HEART FAILURE FOCUSED UPDATE

Committee Member	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Steven M. Hollenberg, *Chair*	Emory University—Director, Cardiac Intensive Care	None	None	None	None	None	None
Lynne Warner Stevenson, *Vice Chair*	Vanderbilt University School of Medicine—Professor of Medicine	None	None	None	None	■ Abbott* ■ American Board of Internal Medicine ■ Cytokinetics* ■ Endotronix*	None
Tariq Ahmad	Yale University School of Medicine—Assistant Professor of Medicine	■ Amgen	■ Novartis†	None	None	None	None
Biykem Bozkurt	Baylor College of Medicine—Professor of Medicine	■ Bristol Myers Squibb ■ Bayer Health Care Pharmaceuticals ■ LAntheus Medical Imaging ■ Respicardia ■ scPharmaceuticals, Inc	None	None	■ Liva Nova, Anthem Trial (DSMB) ■ Abbott Guide HF Trial (CEC)	■ Novartis‡	None
Javed Butler	Baylor Scott and White Health—Professor of Medicine	■ Adrenomed ■ AstraZeneca ■ Bayer† ■ Berlin Cures ■ Boehringer Ingelheim† ■ Boston Scientific ■ CVRx ■ G3 Pharmaceuticals ■ Janssen† ■ Luitpold Pharmaceuticals ■ Medtronic ■ Merck† ■ Novartis† ■ Relypsa† ■ Roche ■ Sanofi Aventis ■ Stealth Peptide ■ Vifor† ■ ZS Pharma	Novartis†	None	■ Amgen (DSMB) ■ Bristol Myers Squibb (DSMB) ■ European Union†		None
Leslie L. Davis	University of North Carolina Chapel Hill—Associate Professor of Nursing	None	None	None	None	None	None
Mark H. Drazner	UT Southwestern Medical Center—Clinical Chief of Cardiology; Medical Director of the LVAD, and Cardiac Transplant Program; Professor of Medicine	None	None	None	None	None	None

Continued on the next page

APPENDIX 1. CONTINUED

Committee Member	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
James N. Kirkpatrick	University of Washington Medical Center—Professor of Medicine, Division of Cardiology and Department of Bioethics and Humanities; Chief of the Section of Cardiac Imaging; Director of Echocardiography; Ethics Committee Chair	None	None	None	None	None	None
Alanna A. Morris	Emory University School of Medicine—Associate Professor of Medicine, Director of Heart Failure Research	■ Abbott Laboratories ■ Boehringer Ingelheim Pharmaceuticals† ■ Cytokinetics ■ Merck† ■ Novo Nordisk ■ Regeneron†	None	None	■ ARRAY 797-301, Pfizer Inc‡ ■ ATTRIBUTE, Eidos Therapeutics‡ ■ Cardio TTRansform, Ionis Pharmaceuticals‡ ■ DAPA-HF, AstraZeneca‡ ■ DCM Consortium Precision Medicine, NHLBI/NHGR‡ ■ EMBARK, MyoKardia‡ ■ MANAGE, Boston Scientific‡ ■ METEORIC, Amgen Inc‡ ■ PARAGLIDE, Novartis Corporation‡ ■ REBIRTH, NHLBI‡ ■ TRANSFORM, NHLBI‡ ■ VICTOR, Merck Co., Inc‡	None	None
Robert Lee Page	University of Colorado—Professor of Medicine	None	None	None	None	None	None
Hasan Khalid Siddiqi	Vanderbilt University Medical Center—Assistant Professor	None	None	None	None	None	None
Alan B. Storrow	Vanderbilt University School of Medicine—Vice-Chairman for Research and Academic Affairs; Associate Professor of Emergency Medicine	■ Alere ■ Siemens†	None	None	None	None	None
John R. Teerlink	San Francisco Veterans Affairs Medical Center—Director of HF and Echo Lab	None	None	None	None	None	None

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*No financial benefit.

†Significant relationship.

‡Relationship with this company is limited to enrolling patients in clinical trials. This disclosure was entered under the Clinical Trial Enroller category in the ACC's disclosure system. To appear in this category, the author acknowledges that there is no direct or institutional relationship with the trial sponsor as defined in the ACC/AHA Disclosure Policy for Writing Committees.

CEC = Clinical Endpoint Committee; DSMB = Data and Safety Monitoring Board; LVAD = left ventricular assist device; NHGR = National Human Genome Research Institute; NHLBI = National Heart, Lung, and Blood Institute; UT = University of Texas.

APPENDIX 2. PEER REVIEWER RELATIONSHIPS WITH INDUSTRY AND OTHER ENTITIES (COMPREHENSIVE)—2024 ACC EXPERT CONSENSUS DECISION PATHWAY ON CLINICAL ASSESSMENT, MANAGEMENT, AND TRAJECTORY OF PATIENTS HOSPITALIZED WITH HEART FAILURE FOCUSED UPDATE

Peer Reviewer	Representation	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Rachel M.B. Barish	Official Reviewer—ACC CV Team Section Council (NP)	MedStar Heart and Vascular Institute—Infiltrative Cardiomyopathy Program Director	None	None	None	None	None	None
Nicole Martin Bhave	Official Reviewer—ACC SSOC	University of Michigan—Clinical Associate Professor	■ ACCF: clinical adjudication committee for vaccine-associated myocarditis	None	None	■ Rednvia, Inc.	None	None
Kristen de Almeida	Official Reviewer—ACC CV Team Section Council (PharmD)	Baptist Health South Florida—Cardiology Clinical Pharmacy Specialist	None	None	None	None	None	None
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ACC = American College of Cardiology; AHA = American Heart Association; DSMB = Data Safety Monitoring Board; NHLBI = National Heart, Lung, and Blood Institute; NIH = National Institutes of Health; SSOC = Solution Set Oversight Committee.

APPENDIX 3. ABBREVIATIONS

ACC = American College of Cardiology
ACE = angiotensin-converting enzyme
ARB = angiotensin receptor blocker
ARNI = angiotensin receptor-neprilysin inhibitor
ECDP = expert consensus decision pathway
GDMT = guideline-directed medical therapy
HaH = hospital at home
HF = heart failure
HFrEF = heart failure with reduced ejection fraction
RAS = renin-angiotensin system
SGLT = sodium-glucose cotransporter