In-Hospital Management of Acute Heart Failure

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KEYWORDS

• Acute heart failure • Heart failure with reduced ejection fraction
• Heart failure with preserved ejection fraction • Guideline-directed medical therapy
• Cardiorenal syndrome • Cardiogenic shock

KEY POINTS

- Patients with decompensated heart failure with evidence of both congestion and poor perfusion have worse outcomes than those with congestion alone.
- Natriuretic peptide levels may assist in diagnosis but are not a substitute for a comprehensive history and physical examination.
- Cardiology consultation should be sought early for patients admitted to the intensive care unit or who fail to improve despite initiation of guideline-directed medical therapy.
- Diuresis should target resolution of symptoms and signs of congestion; diuresis should not be guided by serial natriuretic peptide measurements.
- Guideline-directed medical therapy should be initiated in the hospital and not be routinely stopped; patients discharged on optimal guideline-directed medical therapy have improved outcomes.

INTRODUCTION

Heart failure (HF) is a complex clinical syndrome with symptoms and signs that result from structural or functional impairment of ventricular filling or ejection of blood. The lifetime risk of HF in the United States is high, ranging from 20% to 45% after 45 years of age,[1] and the prevalence of HF is projected to increase by 46% from 2012 to 2030, affecting over 8 million adults in the United States.[2]

An increase in overall HF cases is accompanied by an increase in HF hospitalizations. From 1979 to 2004, the number of HF hospitalizations in the United States has tripled,[3] and in 2017, there were nearly 1.2 million hospitalizations,[4] representing a 26% increase in HF hospitalizations over the prior 5 years. Besides the impact on

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Abbreviations	
ARNI	angiotensin receptor-neprilysin inhibitor
BB	beta blocker
BNP	B-type natriuretic peptide
DOSE	diuretic optimization strategies evaluation
ECG	electrocardiogram
ED	Emergency department
GDMT	guideline-directed medical therapy
HF	heart failure
HFimpEF	HF with improved EF
HFpEF	HF with preserved EF
HFrEF	HF with reduced ejection fraction
IVC	inferior vena cava
LV	left ventricular
MCS	mechanical circulatory support
MRA	mineralocorticoid antagonist
NT-proBNP	N-terminal pro-BNP
SGLT2i	sodium-glucose cotransporter-2 inhibitor

quality of life, an HF hospitalization also carries significant risk of future morbidity and mortality with a 30-day readmission rate of 56%[5] and 1-year mortality of 22%.[6]

The inpatient management of acute HF requires special considerations. Key components of effective management during the HF hospitalization include appropriate diagnostic evaluation, triage and risk stratification, early implementation of guideline-directed medical therapy (GDMT), adequate diuresis, management of cardiogenic shock, and appropriate discharge planning, all of which will be covered in this article.

NEWLY DIAGNOSED HEART FAILURE

Etiologies

The most common underlying etiologies of all subtypes of HF include ischemic heart disease, hypertension, and valvular heart disease. Other less frequent etiologies include familial or genetic cardiomyopathies; amyloidosis; cardiotoxicity with cancer or other treatments or substance use disorders such as alcohol, cocaine, or methamphetamine; tachycardia, right ventricular pacing or stress-induced cardiomyopathies; peripartum cardiomyopathy; myocarditis; autoimmune causes, sarcoidosis; iron overload, including hemochromatosis; and thyroid disease and other endocrine, metabolic, and nutritional causes (**Table 1**).[7] Generally, all etiologies of HF can result in either HF with reduced ejection fraction (HFrEF), defined as ejection fraction (EF) 40% or less, or HF with preserved EF (HFpEF), defined as EF 50% or higher, but there are certain classic presentations. For example, HFpEF is commonly associated with comorbidities such as hypertension, coronary artery disease (CAD), diabetes mellitus, obesity, anemia, chronic kidney disease, atrial fibrillation, and chronic obstructive pulmonary disease.[8–11] Infiltrative cardiomyopathies such as amyloidosis typically present with preserved or mildly reduced EF (HFmrEF, defined as EF 41%–49%). HFrEF, on the other hand, is a more common presentation from myocardial infarction, ischemic heart disease, substance use, tachycardia, stress, peripartum cardiomyopathy, and myocarditis.

Patients with HF with improved EF (HFimpEF, defined as prior EF 40% or less that improves to EF 50% or more) may have any of the etiologies of HFrEF noted earlier and may recover function with resolution of the insult (such as with revascularization, abstinence from substance use, control of heart rate or treatment of arrhythmia). Such

Table 1
Etiologies of heart failure

Ischemic heart disease	Tachycardia
Hypertension	Right ventricular pacing
Valvular heart disease	Stress-induced cardiomyopathies
Familial or genetic cardiomyopathies	Peripartum cardiomyopathy
Amyloidosis	Myocarditis
Cardiotoxicity with cancer or other treatments	Autoimmune diseases
Iron overload and hemochromatosis	Sarcoidosis
Thyroid disease and other endocrine, metabolic, and nutritional causes	Substance use disorders (eg, alcohol, cocaine, or methamphetamine)

patients may also have improvement in EF because they exhibit a strong favorable response to GDMT.

In cases of HFpEF, it is important to exclude noncardiac sources of congestion such as pulmonary disease, kidney disease, liver disease, and obesity, and identify underlying cardiac conditions which may be reversible or warrant disease-directed therapy such as infiltrative disease (most commonly amyloidosis), hypertrophic cardiomyopathy, valvular heart disease, or pericardial constriction.

Initial Evaluation of Newly Diagnosed Heart Failure

Patients admitted to the hospital with a first episode of HF will require a more comprehensive evaluation into etiology than those admitted with a known cardiomyopathy and worsening HF (see **Table 1** for etiologies of HF and **Fig. 1** for a summary of the initial evaluation).[7] However, it is important to recognize that even patients with existing cardiomyopathy may develop a new insult. For instance, a patient with a known ischemic cardiomyopathy may develop new valvular disease as a precipitant. Thus, it is important to review common precipitants regardless of the known underlying etiology when caring for a patient with decompensated HF.

DIAGNOSIS OF DECOMPENSATED HEART FAILURE

The universal definition of HF provides a straightforward approach to determine if a patient's presentation is consistent with HF.[12] The universal definition of HF requires symptoms and/or signs of HF caused by structural/functional cardiac abnormalities *and* at least one of: (1) elevated natriuretic peptides *or* (2) objective evidence of cardiogenic pulmonary or systemic congestion.

History/Physical Examination

Decompensated HF presents most commonly with symptoms and signs of congestion from increased filling pressures, whether left-sided, right-sided, or both, as well as poor perfusion. Symptoms may include weight gain, exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, leg swelling, abdominal bloating, fatigue, and presyncope.

Physical examination findings can include tachycardia, hypotension, peripheral edema, ascites, elevated jugular venous distention, hepatojugular reflex, S3 gallop, and increase in murmurs of mitral and/or tricuspid regurgitation. Notably, rales (crackles) are absent in more than 80% of patients with chronically elevated filling pressures due to compensation of the pulmonary lymphatics.[13] Thus, the absence of rales should not be considered as lack of evidence for decompensated HF.

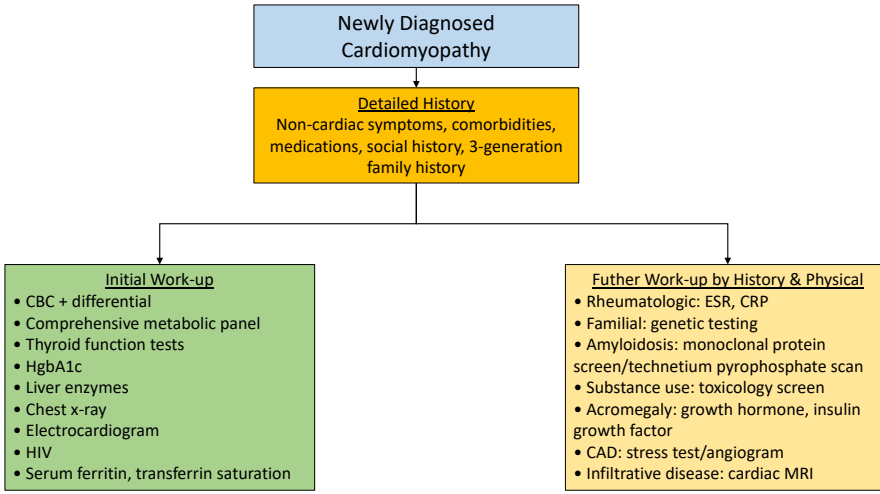

Fig. 1. Initial evaluation of a newly diagnosed cardiomyopathy. CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; H&P, history and physical; HIV, human immunodeficiency virus; HgbA1c, glycosylated hemoglobin.

Another important component of the history is determination of the precipitant for decompensation; **Table 2** describes common precipitants of heart failure. Not every patient requires exhaustive testing; rather, each patient should have a focused evaluation to uncover possible causes for exacerbation of heart failure for which additional evaluation may be warranted. Identification of heart failure precipitants is not only useful for preventive efforts, but it also may impact prognosis. When the identified trigger is infection, worsening kidney disease, or acute coronary syndrome, patients tend to have worse in-hospital[14] and 90-day survival.[15]

Table 2
Common precipitants of decompensated heart failure

F	Forgot medications • Nonadherence with medications • Taking drugs that worsen heart failure such as NSAIDs, steroids, thiazolidinediones, nondihydropyridine CCBs
A	Arrhythmias Anemia
I	Ischemia Infarction Infection
L	Lifestyle • Salt, excessive fluid intake • Alcohol, illicit substances • Obesity
U	Upregulation of cardiac output • Pregnancy, hyperthyroidism
R	Renal failure
E	Embolism (pulmonary)

Abbreviations: CCB, calcium-channel blockers; NSAID, non-steroidal anti-inflammatory drugs.

Imaging

Guideline-based imaging in patients who present with HF symptoms and signs include chest radiography, electrocardiography, and echocardiogram.[7] Chest radiography can demonstrate evidence of pleural effusions or pulmonary edema, with distention of pulmonary veins, enlargement of hilar structures, and Kerley B lines. An electrocardiogram (ECG) is useful to identify evidence of some precipitants such as ischemia or arrhythmias.

An echocardiogram identifies abnormalities of myocardium, heart valves, and pericardium and can thus provide clues to the etiology of HF. Ventricular dilation may offer insight into the chronicity of the HF process, and regional wall motion abnormalities may suggest CAD. Similarly, echocardiographic documentation of increased left ventricular (LV) wall thickness may aid in the diagnosis of hypertrophic cardiomyopathy or amyloidosis. The tricuspid valve regurgitant gradient, coupled with inferior vena cava diameter and its response during respiration, provides estimates of systolic pulmonary artery pressure and central venous pressure, which is useful to guide diuresis.

Although a formal transthoracic echocardiogram can provide the most accurate and in-depth information, point-of-care ultrasonography has become a useful modality to assess volume status in these patients. A dilated inferior vena cava (IVC) with an expiratory diameter greater than 2.0 cm or IVC collapsibility index of lesser than 30% can be suggestive of decompensated heart failure.[16]

Echocardiography importantly offers evaluation of valvular function, specifically the degree of mitral regurgitation and candidacy for transcatheter edge-to-edge repair. However as mitral regurgitation is a dynamic lesion sensitive to volume and afterload, it is essential that ECG be performed when the patient is free from congestion and medically optimized with maximum-tolerated dosages of GDMT.[7,17]

Laboratory Evaluation

Guideline-based laboratory evaluation for patients hospitalized with HF includes complete blood count (CBC), urinalysis, serum electrolytes, blood urea nitrogen, serum creatinine, glucose, and liver function tests.[7] Cardiac enzymes, specifically troponin, can be elevated in the setting of decompensated HF from subendocardial ischemia or increased myocardial demand CAD and should be interpreted in the context of the clinical presentation. Finally, arterial blood gas and lactate can be instrumental in early recognition of cardiogenic shock.

In patients presenting with dyspnea, measurement of natriuretic peptides (B-type natriuretic peptide [BNP] or its precursor, N-terminal pro-BNP [NT-proBNP]), are useful to support or exclude a diagnosis of HF.[7] For patients presenting to the emergency department (ED) with dyspnea, the diagnostic accuracy of BNP at a cutoff of 100 pg/mL is 83% and the negative predictive value BNP lesser than 50 pg/mL is 96%.[18] For NT-proBNP, age-related cut-points of 450, 900, and 1800 pg/mL for ages lesser than 50, 50 to 75, and greater than 75, respectively, yield 90% sensitivity and 84% specificity for acute HF. An age-independent cut-point of 300 pg/mL has 98% negative predictive value to exclude acute HF.[19] However, it is important to remember that patients with HFpEF or obesity may have normal natriuretic peptide levels despite other evidence of HF.[7]

In patients hospitalized for HF, measurement of BNP or NT-proBNP levels at admission is also recommended to establish prognosis.[7] In 1 study, patients with admission BNP levels at or above 5000 pg/mL had an associated in-hospital mortality of 8.5%, while those patients with BNP measurements under 100 pg/mL had an in-hospital mortality of 1.9%.[20]

Laboratory evaluation for HF should always be interpreted in the context of the clinical presentation. Therefore, the pretest probability for HF should be estimated independent of the natriuretic peptide level to guide next steps, which may include pursuing other noncardiac diagnostic avenues or proceeding with HF management. Although an elevated BNP is associated with worse prognosis, the evidence for treatment guidance using serial BNP or NT-proBNP measurements remains insufficient and this practice should not be pursued.[7]

TRIAGE OF DECOMPENSATED HEART FAILURE

Assessing Hemodynamic Profiles

Patients with HF can be stratified based on their hemodynamic profiles, defined as the presence or absence of congestion (wet or dry) and adequate perfusion (warm or cold; **Fig. 2**).[21] When applied prospectively, compared with patients with HF who were warm and dry, those who are warm and wet or cold and wet have a 1.8-fold and 2.5-fold, increased risk of death or transplantation at 18 months, respectively.[21] Thus, these clinical profiles offer insight into potential need for admission, higher level of care, and/or cardiology consultation.

Criteria for Inpatient Admission

Patients with HF may follow one of 3 general paths after initial therapy in the ED.[22] The first group is comprised of low-risk patients who respond to initial therapy, returning quickly to their baseline without any high-risk features such as hypotension, kidney dysfunction, or unstable arrhythmias. These patients are often eligible for discharge from the ED. The second group constitutes high-risk patients who develop worsening clinical status despite therapy with continued symptoms, worsening kidney function, hypotension, or an elevated troponin—this group warrants hospitalization and inpatient care. The third group consists of patients with intermediate risk, who have a partial response to therapy; that is, symptoms improve partially without high-risk features. Because this group has an incomplete response, they require continued treatment and observation either in an ED or observation setting. A summary of criteria for admission is shown in **Box 1**.

Decision-Making on Telemetry Versus Intensive Care Unit Care

Patients with HF are usually admitted to a nonintensive care unit setting on telemetry monitoring. However, patients with significant hemodynamic derangement or those

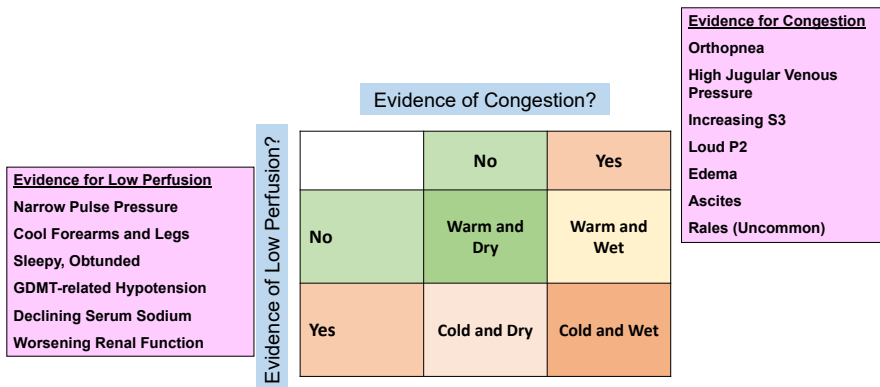

Fig. 2. Hemodynamic profiles of HF.

Box 1
Criteria for heart failure admission

- Critical illness at presentation: hypoxia, hypotension, and cardiogenic shock
- New diagnosis of HF
- Marked congestion with incomplete response to initial therapy
- Evidence for ischemia, uncontrolled arrhythmias, or infection
- Significant laboratory abnormalities: renal dysfunction, hyponatremia, and hypokalemia/hyperkalemia
- Poorly managed comorbidities such as chronic obstructive lung disease or diabetes

requiring intravenous infusion of vasoactive agents and/or ventilatory support (invasive or noninvasive positive-pressure ventilator) may benefit from higher levels of care. Similarly, transfer to an intensive care unit for closer monitoring and more aggressive intervention may be warranted if patients develop hypotension, respiratory distress unresponsive to diuretics, worsening kidney function, and/or hemodynamically significant tachy-arrhythmias or brady-arrhythmias.

Indications for Cardiology Consultation

Cardiology consultation at presentation is indicated for any patient with HF being admitted to an intensive care unit. For patients admitted to a nonintensive care unit setting, cardiology consultation may be warranted if there is evidence of a *cold and wet* hemodynamic profile.[21,23] Such patients are at high risk of developing incipient cardiogenic shock and may require pulmonary artery catheterization, inotropic support, and consideration of advanced HF therapies such as transplantation or mechanical circulatory support.[7] Patients who develop ischemia as evidenced by refractory symptoms, persistent or worsening ECG changes, or rising troponin also warrant cardiology consultation. Indications for cardiology consultation are listed in **Box 2** while a useful mnemonic comprising the cardinal features of advanced HF, "I NEED HELP",[24] is shown in **Table 3**.

Regardless of whether cardiology consultation is sought at the outset or during HF hospitalization, it is essential that clinicians pause to perform a *trajectory check* for their patients. Clinicians should consider the following: (1) are heart failure symptoms and signs of congestion improving, (2) is there evidence of adequate perfusion, and (3)

Box 2
Indications for cardiology consultation

- Admission to the intensive care unit
- *Cold and wet* hemodynamic profile
- Ischemic symptoms, especially if accompanied by ECG changes and elevated troponin
- Severe valvular disease
- Atrial fibrillation with rapid ventricular response; ventricular tachycardia
- Persistent congestion unresponsive to high-dose diuretic therapy
- Worsening kidney or liver function

Table 3
Identification of patients with advanced heart failure

I	Inotropic support, previous or ongoing
N	NYHA Class III/VI or elevated Natriuretic peptides
E	End-organ dysfunction of kidney or liver
E	Ejection fraction <20%
D	Defibrillator shocks for ventricular arrhythmias
H	Hospitalizations, i.e., one or more in the prior 12 mo
E	Edema/Escalating diuretics
L	Low blood pressure, e.g., systolic pressure under 90 mm Hg
P	Prognostic medications, i.e., inability to titrate or need to decrease GDMT due to low blood pressure or kidney dysfunction

are patients being initiated on and tolerating GDMT? For patients who worsen despite appropriate efforts, cardiology consultation is warranted.

Palliative care consultation also may be useful during an HF hospitalization, especially for patients with high-risk features and/or contraindications to advanced therapies.[25] A *goals of care* conversation during hospitalization offers the advantage of communicating with the patient and caregivers at a time when they may be more receptive to and available for discussion. Whenever possible, the patient's primary care provider should be included in these meetings because that provider often knows the patient best and shared decision-making and input allows for continuity of care in the outpatient setting.

MANAGEMENT

Approach to Diuretics

The 2022 AHA/ACC/HFSA guideline for management of HF recommends that patients admitted with decompensated HF should receive prompt treatment with intravenous loop diuretics.[7] For a diuretic-naïve patient, an initial dose of intravenous furosemide 40 mg is a reasonable starting point. For a patient with chronic HF on outpatient loop diuretic therapy, the diuretic optimization strategies evaluation (DOSE) study provides insight into diuretic dosing and administration.[26] In DOSE, patients were randomized to an administration route (bolus dosing every 12 hours or continuous infusion) and a dosing strategy (low-dose or high-dose).

There were no differences in the primary endpoint of patient-reported improvement of symptoms, or the primary safety endpoint of change in serum creatinine from baseline to 72 hours between the bolus and continuous infusion groups or the low-dose and high-dose groups. However, patients in the high-dose group had significant improvement in several secondary endpoints, including decreased dyspnea, decreased body weight, and increased fluid loss at 72 hours.[26] Therefore, in hospitalized patients with acute decompensated heart failure (ADHF) on outpatient furosemide, initiation of high-dose furosemide with a daily intravenous dose that is equal to 2.5 times their daily outpatient oral dose is ideal, using either bolus or continuous infusion.

For patients with inadequate diuresis, defined as less than 3 L of urine output daily,[27] serial doubling of intravenous loop diuretic doses by bolus or infusion and/or sequential nephron blockade with addition of a thiazide diuretics should be considered (**Table 4**).[7] Addition of acetazolamide, a carbonic anhydrase inhibitor, to furosemide therapy, improved objective measurement of natriuresis and relief of congestion at

Table 4
Diuretic options in decompensated heart failure

	Initial Daily Dose	Maximum Total Daily Dose	Comments
Loop diuretics			
Furosemide	20–40 mg IV	600 mg	No clear evidence for benefit of one loop diuretic over another
Bumetanide	0.5–1 mg IV	10 mg	
Torsemide	10–20 mg PO	200 mg	Only available orally; no benefit of torsemide vs furosemide in 12-mo all-cause mortality among patients discharged after hospitalization for heart failure[64]
Thiazide diuretics			
Chlorothiazide	250–500 mg IV	1000 mg	May be prescribed 30 min before loop diuretic to augment diuresis; requires at least twice-daily monitoring of potassium due to risks of severe hypokalemia
Metolazone	2.5–5 mg PO	20 mg	
Mineralocorticoid antagonists			
Spironolactone	12.5–25 mg PO	100 mg	Useful to maintain potassium levels during diuresis with loop ± thiazide diuretics
Eplerenone	25–50 mg PO	100 mg	
Carbonic anhydrase inhibitor			
Acetazolamide	250–500 mg IV	1000 mg	May serve to augment diuresis[28] and mitigate diuretic-induced metabolic alkalosis; effective when used for up to 48 h

discharge though there was nonsignificant increase all-cause mortality and rehospitalizations at 3 months in the group receiving acetazolamide.[28]

Targets for the Diuretic Response

The endpoint for diuresis is resolution of patient symptoms and signs of HF. However, before discharge, the clinical signs of congestion have usually resolved in only 50% to 70% of patients.[29,30] This gap is likely because symptoms of congestion usually improve before the signs of congestion have fully resolved. Therefore, if diuresis is guided solely by symptom relief, it may be stopped prematurely leading to inappropriate discharge and risk of readmission. Clinicians should therefore ensure that signs of congestion such as jugular venous distention, dyspnea at rest, and orthopnea or edema have normalized/resolved before discharge. When this objective, physical examination-guided evidence of euvolemia is achieved, the treating clinician can designate the patient's *dry weight.*

Diuresis should not be halted solely based on a rising creatinine. Modest increases in serum creatinine levels are not linked to worse outcomes as long as the rise in creatinine is transient,[31,32] accompanied with successful decongestion,[33,34] or occurs after initiation of renin-angiotensin-aldosterone system antagonists.[35] In fact, more aggressive diuresis as demonstrated by the presence of hemoconcentration[36] or by targeting at least 3 to 5 L of urine output every 24 hours[27] is associated with better kidney function and improved postdischarge outcomes.

Guideline-Directed Medical Therapy

In patients with HFrEF, the combination of an angiotensin receptor-neprilysin inhibitor (ARNI; sacubitril/valsartan), an evidence-based beta-blocker (BB; bisoprolol, carvedilol, or sustained-release metoprolol), a mineralocorticoid antagonist (MRA; spironolactone or eplerenone), and a sodium-glucose cotransporter-2 inhibitor (SGLT2i; dapagliflozin or empagliflozin) improves quality of life, decreases HF hospitalizations, and improves survival.[7] Therefore, these agents should be initiated or continued during hospitalization and/or restarted at the time of hospital discharge. Starting and target dosages for GDMT in heart failure are shown in **Table 5**. In patients with HFpEF, SGLT2i therapy should be initiated to reduce the risk of cardiovascular death and HF hospitalizations.[37,38] Addition of an MRA should also be considered to further reduce the risk of HF hospitalization,[7] with evidence of benefit from spironolactone[39] and finerenone.[40]

There is strong evidence for the initiation, titration, and continuation of these agents in HF, and hospitalization offers a critical opportunity to optimize GDMT. Initiation and continuation of GDMT during HF hospitalization is safe and reduces the risk of subsequent death and readmission.[41–44] Thus, in patients hospitalized with HF, preexisting GDMT should not be routinely discontinued, but rather optimized and/or reinitiated once clinical stability is achieved. GDMT should not be withheld for mild or transient reductions in blood pressure or mild deteriorations in kidney function. Specifically, withholding or reducing BB therapy should only be considered in patients with marked volume overload or marginally low cardiac output, or for whom recent increase in BB dosage is presumed to be the trigger for decompensation and HF hospitalization. In fact, true contraindications to GDMT are rare, such as bradycardia or high-degree atrioventricular block in the absence of pacemakers for nodal blocking agents, cardiogenic shock requiring inotropic support for BB, or angioedema for angiotensin converting-enzyme inhibitors (ACEI) or ARNI therapy.

An empirical roadmap for *de novo* initiation, resumption, and optimization of GDMT in HF is illustrated in **Fig. 3**. In this strategy, MRA therapy, which balances the potassium wasting of loop diuretics is prioritized, followed by agents that offer afterload

Table 5
Starting and target dosages for heart failure medications

Medication	Initial Dose	Target Dose
ARNI		
Sacubitril/valsartan	24/26 mg twice daily	97/103 mg twice daily
ACEI		
Enalapril	2.5 mg twice daily	10–20 mg twice daily
Lisinopril	2.5–5 mg daily	20–40 mg daily
Ramipiril	1.25–2.5 mg once daily	10 mg once daily
ARB		
Candesartan	4–8 mg once daily	32 mg once daily
Losartan	25–50 mg once daily	50–150 mg once daily
Valsartan	20–40 mg once daily	160 mg twice daily
BB		
Bisoprolol	1.25 mg once daily	10 mg once daily
Carvedilol	3.125 mg twice daily	25–50 mg twice daily
Metoprolol succinate	12.5–25 mg once daily	200 mg once daily
MRA		
Eplerenone	25–50 mg once daily	50 mg once daily
Finerenone[a]	10–20 mg once daily	20–40 mg once daily
Spironolactone	12.5–25 mg once daily	25 mg once daily
SGLT2 inhibitor		
Dapagliflozin	10 mg once daily	10 mg once daily
Empagliflozin	10 mg once daily	10 mg once daily

Abbreviations: ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker
[a] Not currently approved for the treatment of HF.
Adapted from.[7]

reduction and long-term benefits (ARNI, SGLT2i). These agents are safe to initiate in patients who are congested and well-perfused (warm and wet). For patients who are already on GDMT, it is unnecessary to stop GDMT during hospitalization in the absence of hypotension, shock, or absolute contraindications as outlined above. BB if stopped, should be restarted at a low dose before discharge once the patient is successfully decongested.

Additionally, measurement of serum ferritin and transferrin saturation levels can detect iron deficiency in patients, and treatment with iron replacement HF readmission.[45,46]

Vasodilator Therapy

Vasodilators can be used in acute HF to relieve symptoms of pulmonary congestion in selected patients, particularly those without hypertension (SBP >110 mm Hg). Although they may mitigate dyspnea and relieve pulmonary congestion, their benefits have not been shown to have durable effects for either rehospitalization or mortality benefit.[47] Thus, the role for directed vasodilators in acute decompensated HF remains uncertain.

Patients with hypertension, coronary ischemia, or significant mitral regurgitation may be suitable candidates for the use of intravenous nitroglycerin. However, tachyphylaxis may develop within 24 hours, and up to 20% of those with HF may develop

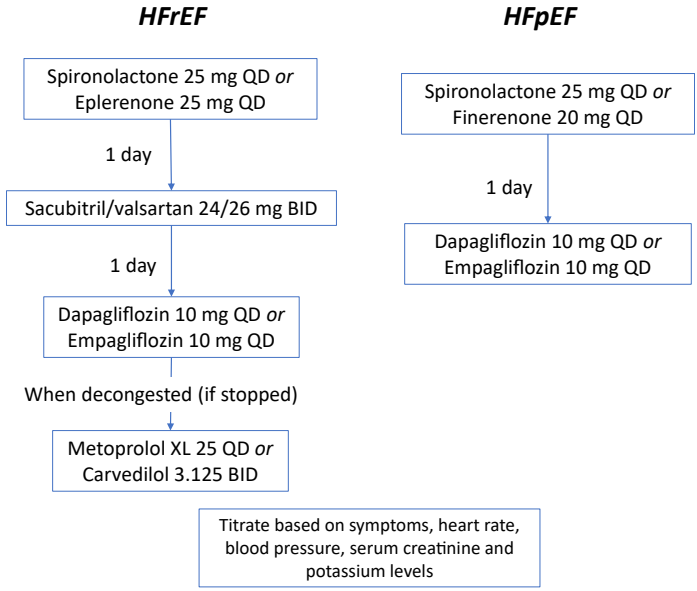

Fig. 3. Empirical roadmap for *de novo* initiation, resumption, and optimization of GDMT in HF.

resistance to even high doses.[48] Sodium nitroprusside is approved for the treatment of acute decompensated HF; as a direct-acting vasodilator it has the potential for producing marked hypotension. Invasive hemodynamic blood pressure monitoring (eg, an arterial line) is often required with use, and nitroprusside is typically used in the intensive care setting. Albeit rare, extended duration infusions of nitroprusside have been associated with thiocyanate and cyanide toxicity, particularly in the setting of kidney and hepatic disease. Vasodilators such as nitroglycerine and nitroprusside are potentially of value in severely congested patients with hypertension or severe mitral regurgitation complicating LV dysfunction.[49]

Overall, there is no data that suggest that intravenous vasodilators improve outcomes in the patient hospitalized with HF; thus, use of intravenous vasodilators is limited to the relief of dyspnea in the hospitalized HF patient with intact or high blood pressure.[47]

Venous Thromboembolism Prophylaxis

HF has long been recognized as affording additional risk for venous thromboembolic disease. When patients are hospitalized for decompensated HF, or when patients with chronic stable HF are hospitalized for other reasons, they are at increased risk for venous thromboembolic disease. The risk may be associated with higher HF symptom burden.[50] The use of anticoagulation with subcutaneous low-molecular-weight heparin, unfractionated heparin, fondaparinux, or approved direct oral-acting anticoagulants is used for the prevention of clinically symptomatic deep vein thrombosis and pulmonary embolism.[51,52]

Shock

Cardiogenic shock describes a clinical condition of hypoperfusion that requires interventions including inotropes, pressors, mechanical support beyond volume

resuscitation to restore perfusion.[53] These patients typically present with relative hypotension, with the majority manifesting the classic shock phenotype of mean arterial blood pressure $\leq$60 mm Hg or systolic blood pressure $\leq$90 mm Hg and evidence of hypoperfusion with altered mental status, cool extremities, oliguria, and pulmonary edema. The laboratory findings may include impaired kidney function, elevated lactate, BNP, and/or liver enzymes. Invasive hemodynamics will demonstrate the classic elevating left-sided and/or right-sided filling pressures and depressed cardiac index that are associated with cardiogenic shock.

Although intravenous inotropic support is typically first-line therapy for optimization of cardiac index and perfusion, these agents carry risks such as arrhythmia and increased myocardial oxygen consumption and may offer insufficient circulatory support. Temporary mechanical circulatory support (MCS) devices, on the other hand, can provide significantly more hemodynamic support, such as increasing cardiac output and reducing afterload.[54]

There are multiple options for device implantation and selection of devices should be tailored for each patient and clinical condition. There are 3 circuit configurations for MCS devices—right atrium to systemic artery, left atrium to systemic artery, and left ventricle to systemic artery. Depending on the configuration, cardiac output can be augmented from 0.5 L/min up to 7 L/min (**Table 6**). There has been mixed evidence in support of the use of percutaneous temporary MCS devices.[55–57] There is no randomized data to support 1 temporary MCS device over another; therefore, the type of device selected should be individualized to balance the advantages of greater support at the cost of increase invasiveness with associated risks of bleeding, vascular injury, and thromboembolism. Nonetheless, employing these devices in a timely manner, with the aid of invasive hemodynamics to optimize perfusion, can be useful as a bridge to myocardial recovery or to advanced HF therapies such as durable MCS or heart transplantation.

DISCHARGE PLANNING

Transitioning to Discharge

Transitioning to discharge for patients admitted with acute HF represents a critical juncture in hospital management to achieve sustained treatment response and prevent morbidity and mortality associated with potential rehospitalization.[45] Some patients may be offered the use of indwelling urinary catheters during treatment, but their use is not routinely recommended and removal with trial of void should be conducted as soon as possible to avoid complication during hospitalization.[58] Continuous cardiac monitoring may often be employed in these patients for the detection of cardiac arrhythmias, and discontinuation of telemetry should be sought in those patients with normal or stable heart rhythms and in the absence of the use of inotropic agents.[59] The use of supplemental oxygen should be stopped when arterial oxygen is adequate with the patient breathing room air.[60] Before completion of hospitalization, clinicians must address these domains in patients with HF: (1) minimize congestion, (2) optimize GDMT, and (3) arrange for postdischarge follow-up.

Diuretic Dosage

Initial steps include de-escalation of intravenous diuretic therapy to the lowest dose that maintains euvolemia and conversion from intravenous therapy to oral therapy. When transitioning diuretics from intravenous to the oral route, an oft-cited bioavailability study established that intravenous furosemide is twice as potent as the oral

Table 6
Temporary mechanical circulatory support devices

	Impella RP	TandemHeart RA-PA	VA-ECMO	IABP	Impella (2.5, CP, 5.0, 5.5)	TandemHeart LA-FA
Flow	Max 4.0 L/min	Max 4.0 L/min	Max 7.0 L/min	0.5 L/min	2.5–5.0 L/min	Max 4.0 L/min
Mechanism	RA->PA	RA->PA	RA->Ao	Ao	LV->Ao	LA->Ao
LV Unloading				+	+ to +++	++
RV Unloading	+	+	++			
Cardiac Power	-	-	⇑⇑	⇑	⇑⇑	⇑⇑
Afterload	-	-	⇑⇑	⇓	⇓⇓	⇑
Coronary Perfusion	-	-	-	⇑	⇑	-

Abbreviations: FA, femoral artery; IABP, intra-aortic balloon pump; LA, left atrium; PA, pulmonary artery; RA, right atrium; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

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form.[61] Diuretic dosages should be selected at doses that reduce the risk for hypotension and hypovolemia in patients, particularly when initiating GDMT.[62]

Options for outpatient loop diuretics include furosemide, torsemide, and bumetanide at a ratio of potency of 4:2:1.[63] In the TRANSFORM-HF trial, patients hospitalized for HF were randomized to torsemide versus furosemide at discharge with no difference in all-cause mortality after 12 months.[64] Thus, there is no clear evidence of benefit of 1 loop diuretic over another, and choice of specific loop diuretic will be governed by availability and cost.

Sequential nephron blockade with thiazide diuretics at discharge should be reserved for those patients who are refractory to single-agent diuresis and in those cases reduced from daily therapy to intermittent therapy such as 3 times or once weekly.[65] Kidney function and electrolytes should be monitored closely at discharge and every 1 to 2 weeks after with each adjustment in diuretic dose or until stability is reached.[66]

Strategies to Prevent Readmissions

Strategies to prevent HF readmissions involve examining the causes for readmission and identification of prognostic variables.[67] The most effective method to prevent readmission is GDMT optimization as described earlier. Therapies that reduce HF hospitalizations should also considered to reduce future hospitalization for these patients at high-risk for readmission.[68] In fact, updated performance and quality measures for adults hospitalized with HF (left ventricular ejection fraction [LVEF] <40%) emphasize that patients should be prescribed GDMT at discharge.[41,42,69–72]

Follow-up Plan

Postdischarge follow-up for acute HF include education such as dietary sodium and water intake restrictions, follow-up visits for clinical assessment and medication management, and laboratory analysis as above.[73] Clinical assessment involves the use of congestion scores based on physical examination, body weight, New York Heart Association functional class, blood pressure, heart rate, and signs of hypoperfusion.[73] Natriuretic peptides are markers of congestion, and the change in concentration from hospitalization compared to before discharge may be checked to predict postdischarge outcomes.[74] The achievement of improved levels of natriuretic peptides 1-month posthospitalization portends a more favorable prognosis in patients with chronic HF.[75,76]

Follow-up for patients admitted with HF is recommended to occur with a visit in 1 to 2 weeks (optimally within 7 days) and/or a telephone follow-up within 3 days of hospital discharge.[77–80] Some patients may benefit from noninvasive home telemonitoring, invasive hemodynamic monitoring, or cardiac rehabilitation programs.[81,82] In addition to ambulatory monitoring, a care team including cardiologists, a general practitioner, and a nurse dedicated to chronic HF management for patients is necessary to avoid or decrease urgent care or emergency department visits and hospitalizations in these patients.[80,83,84]

SUMMARY

An HF hospitalization is a sentinel event; in the first 30 days following an admission for HF, up to 25% of patients will be readmitted. [85] In the first year following an admission for HF, almost 30% of patients will die.[86] Thus, an HF hospitalization is not just an opportunity to achieve decongestion and improve other symptoms, but it should be

viewed as a teachable moment to optimize GDMT and trigger a discussion around prognosis including eligibility for and interest in advanced HF therapies.

With the guidance in this review, patients may receive a comprehensive assessment of HF etiology and precipitants, decongestion and medication optimization, identification of high-risk features to trigger more advanced therapy at triage, and successful transitions of care to prevent future readmissions.

CLINICS CARE POINTS

- Patients with decompensated heart failure should be evaluated for evidence of congestion and poor perfusion to guide appropriate management strategies.
- Patient with decompensated heart failure with evidence of cardiogenic shock require evaluation for advanced heart failure therapies.
- In patients with decompensated heart failure, every effort should be made to initiate and optimize guideline-directed medical therapy.

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