Management of Acute Decompensated Heart Failure

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Relevant Financial Disclosure(s)

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- I have nothing to disclose.

Objectives

- Briefly describe the epidemiology of heart failure
- Discuss Definitions
- Review hemodynamic profile of decompensated heart failure patients.
- Review various pharmacologic agents used to manage decompensated HF based on updated guideline.
- Briefly discuss advanced HF

Epidemiology of HF

- ≈ 6.7 million Americans ≥20 years of age adults in US are affected with HF.
- 50% as HF with reduced ejection fraction (HFrEF) and 50% as HF with preserved EF (HFpEF).
- Approximately 10% with advanced HF
- Cost: $31 billion in 2012, $70 billion by 2030
- At least 1.2 million hospitalizations every year
- 25% of patients hospitalized are readmitted within 30 days of discharge
- High mortality: 1year- 10% and 5 year- >50%
- In 2020, HF was the underlying cause in 85855 deaths
- After cancer, it is the leading cause of death

American Heart Association 2023

Definition

- HF is a complex clinical syndrome that results from any structural or functional impairment of ventricular filling or ejection of blood.
- The cardinal manifestations of HF are dyspnea and fatigue, which may limit exercise tolerance, and fluid retention, which may lead to pulmonary and/or splanchnic congestion and/or peripheral edema.

2022 AHA/ACC/HFSA Guideline for the Management HF

Classification of HF by Left Ventricular Ejection Fraction (LVEF)

Type of HF according to LVEF	Criteria
HFrEF (HF with reduced EF)	• LVEF <40%
HFimpEF (HF with improved EF)	• Previous LVEF ≤40% and a follow-up measurement of LVEF >40%
HFmrEF (HF with mildly reduced EF)	• LVEF 41%–49% • Evidence of spontaneous or provokable increased LV filling pressures (e.g., elevated natriuretic peptide, noninvasive and invasive hemodynamic measurement)
HFpEF (HF with preserved EF)	• LVEF ≥50% • Evidence of spontaneous or provokable increased LV filling pressures (e.g., elevated natriuretic peptide, noninvasive and invasive hemodynamic measurement)

2022 AHA/ACC/HFSA Guideline for the Management HF

Acute Decompensated HF

- Acute decompensated heart failure (ADHF) is a clinical syndrome of new or worsening signs and symptoms of HF that often lead to hospitalization or an ER visit.
- ~70% with ADHF are admitted due to worsening chronic HF, up to 15-20% present with HF for the first time, and ~5% are admitted for advanced or end-stage HF.
- Can occur with either reduced or preserved EF
- Can lead to a systemic disorder affecting all vital organs
- Mechanisms of dysfunction:
  - Congestion and hypoperfusion

Factors That Can Contribute to Worsening HF

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

Basic Diagnostic Studies

- EKG
- CXR
- CBC, CMP, mag, BNP, TSH, uric acid
- Lactic acid if signs of hypoperfusion
- Echocardiography: Recommended for patients with new HF and for patients with prior history of HF with suspected change in cardiac function

Assessment

- Initial triage includes clinical assessment of the hemodynamic profile for severity of congestion and adequacy of perfusion
- Most patients admitted with HF have clinical evidence of congestion without apparent hypoperfusion
- Hospitalization for HF is a sentinel event that signals worsening prognosis and the need to restore hemodynamic compensation but also provides key opportunities to redirect the disease trajectory
- Address factors that contributed to worsening HF
- Establishment of optimal volume status is a major goal, and patients with residual congestion merit careful consideration for further intervention before and after discharge, because they face higher risk for re-hospitalization and death

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

Clinical Evidence of Congestion

Symptoms

Dyspnea on minimal exertion, Orthopnea

Paroxysmal nocturnal dyspnea

Nocturnal cough

Bendopnea

Abdominal swelling

Early satiety

Anorexia, nausea

Right upper quadrant pain

Peripheral swelling

Rapid weight gain

Signs

- Elevated jugular venous pressure
- Rales
- Pleural effusion
- Third heart sound
- Murmurs of mitral and/or tricuspid regurgitation
- Hepatomegaly
- Ascites
- Pre-sacral, scrotal, or perineal edema
- Peripheral edema

Hypoperfusion

- Cold sweaty extremities
- Oliguria
- Mental confusion
- Dizziness
- Diminished pulse pressure
- Organ dysfunction

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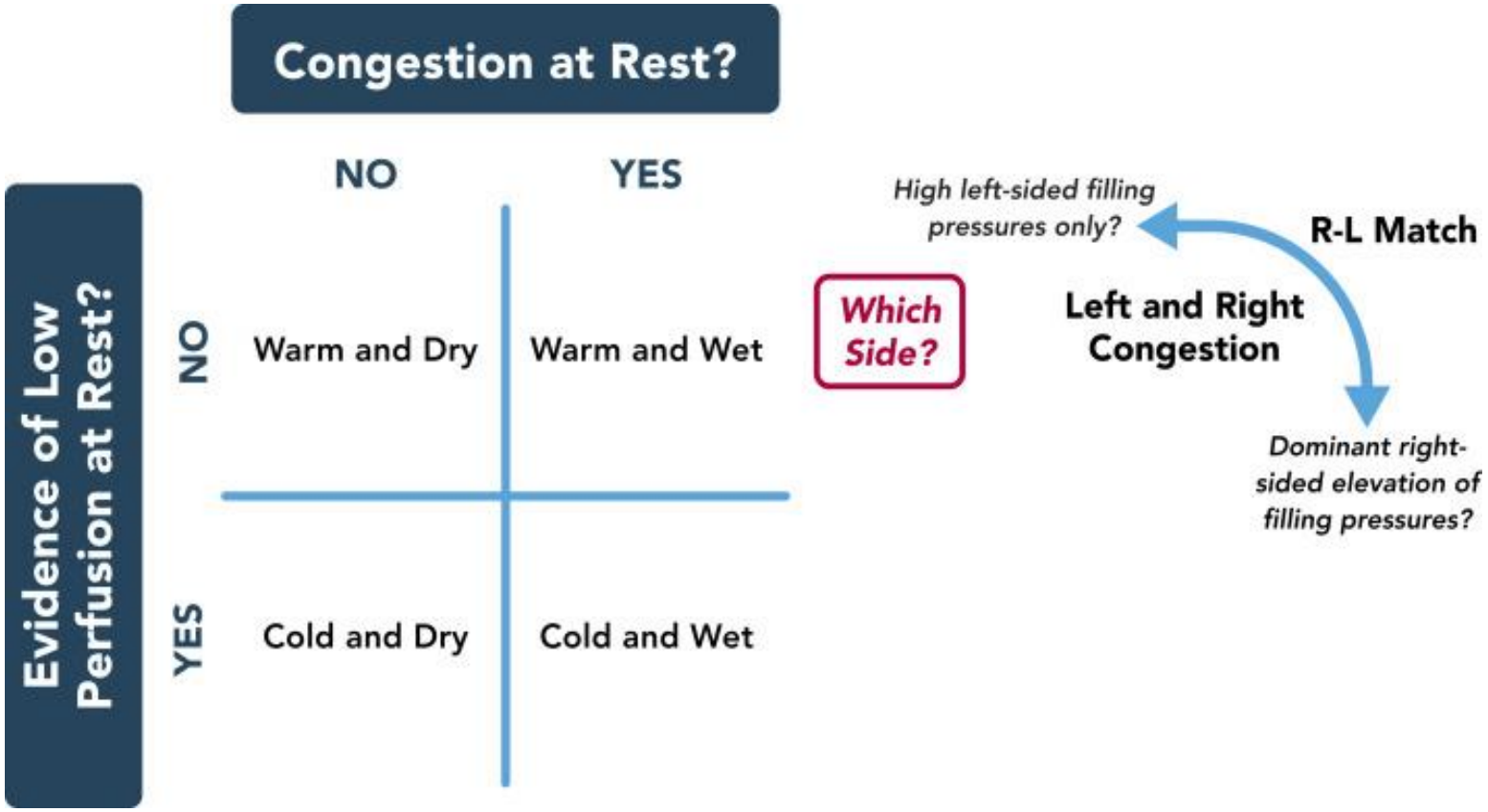
Hemodynamic Profile



Hollenberg SM, Stevenson LW, Ahmad T, et al. 2019

Risk Assessment During Hospitalization

- Chronic history prior to admission
- Class IV symptoms
- Nonadherence to medications or salt/fluid restriction
- Progressively higher risk with higher admission BNP
- Elevated serum creatinine or low clearance
- High BUN
- Low spot urine Na after 1st IV diuretic dose
- Diuretic resistance with high outpatient doses
- Hemodynamic profile of “cold and wet” at admission
- Low systolic blood pressure
- Hyponatremia
- No RAS therapy
- No beta blocker therapy
- Unexpected in-hospital events such as Resuscitation or Intubation or IV inotropic therapy even for a short period

Treatment goal

The central themes of care for patients hospitalized for decompensated HF are:

- Decongestion
- Optimization of the therapies recommended for HF
- Maintain perfusion

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Decongestion Strategy

- Intravenous loop diuretic therapy provides the most rapid and effective treatment for signs and symptoms of congestion leading to hospitalization for HF.
- Titration to achieve effective diuresis may require doubling of initial doses, adding a thiazide diuretic, or adding an MRA that has diuretic effects in addition to its cardiovascular benefits.
- A major goal of therapy is resolution of the signs and symptoms of congestion before discharge, as persistent congestion scored at discharge has been associated with higher rates of rehospitalizations and mortality
- Therapy with diuretics and other guideline-directed medications should be titrated with a goal to resolve clinical evidence of congestion to reduce symptoms and rehospitalizations

Initial Diuretic Dosing

- Double outpatient diuretic dose in patients with HF and volume overload
- Either twice daily bolus IV dosing or bolus with continuous infusion can be used
- Consider dual diuretic therapy with thiazide if volume removal is suboptimal with loop diuretic

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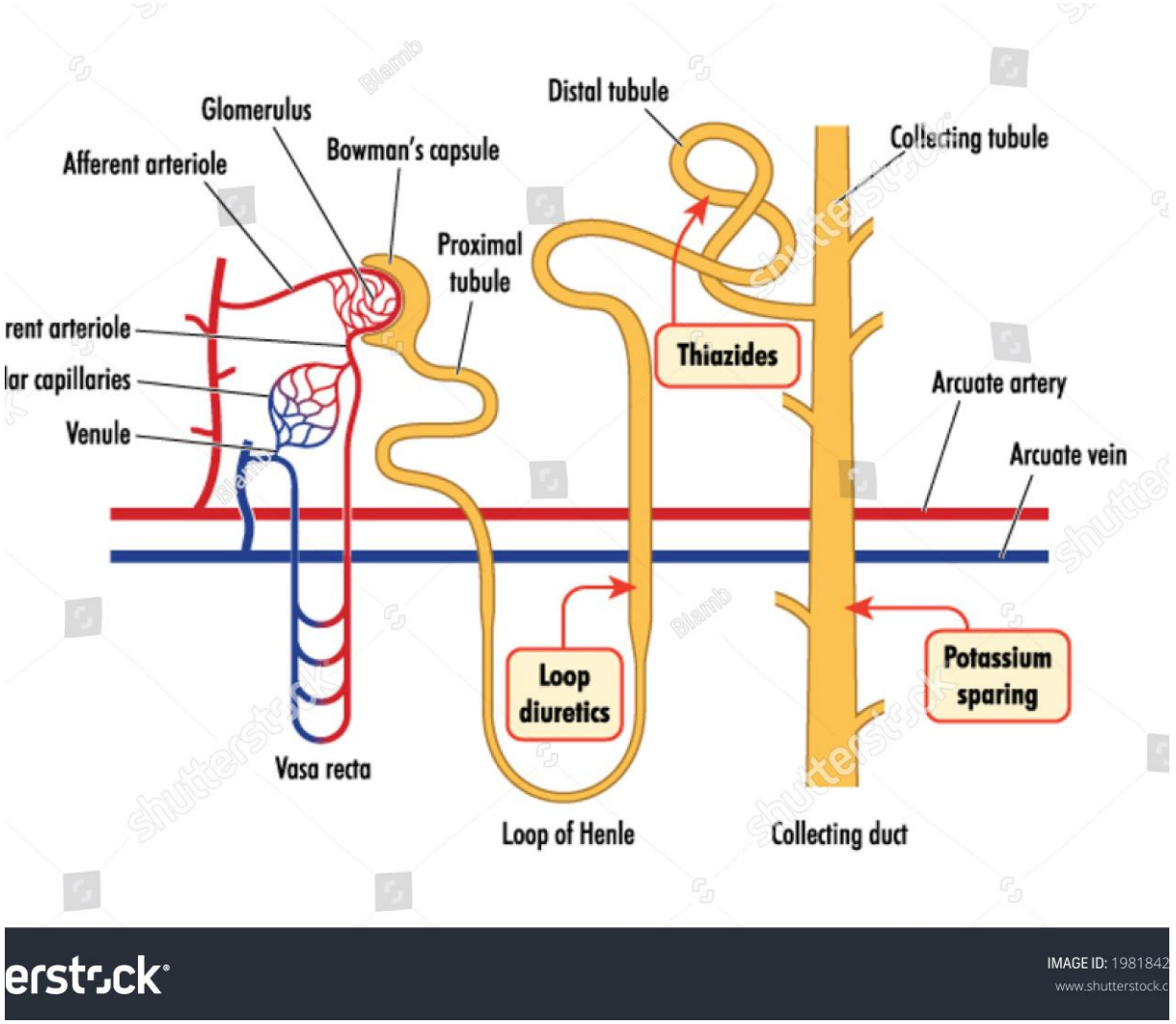
Diuretic

Class	Drug	Usual Inpatient Dosing (Maximum)	Usual Outpatient Dosing (Maximum)
Loop diuretics	Bumetanide	0.5–4 mg/hour IV QD-TID (5 mg/dose) *Or* 0.5–2 mg/hour IV infusion (4 mg/hour)	0.5–2 mg PO QD or BID (10 mg/day)
	Furosemide	40–160 mg IV QD-TID (200 mg/dose) *Or* 5–20 mg/hour IV infusion (40 mg/hour)	20–80 mg PO QD-BID (600 mg/day)
	Torsemide	N/A	10–40 mg PO QD-BID (200 mg/day)
Thiazide-type diuretics	Chlorothiazide	0.5–1 g IV QD-BID (2 g/day)	N/A
	Hydrochlorothiazide	25–50 mg PO QD-BID (100 mg/day)	25–50 mg PO QD (100 mg/day)
	Chlorthalidone	12.5–25 mg PO QD-BID (100 mg/day)	25–50 mg PO QD (100 mg/day)
	Metolazone	2.5–5 mg PO QD-BID (20 mg/day)	2.5–5 mg PO QD (20 mg/day)

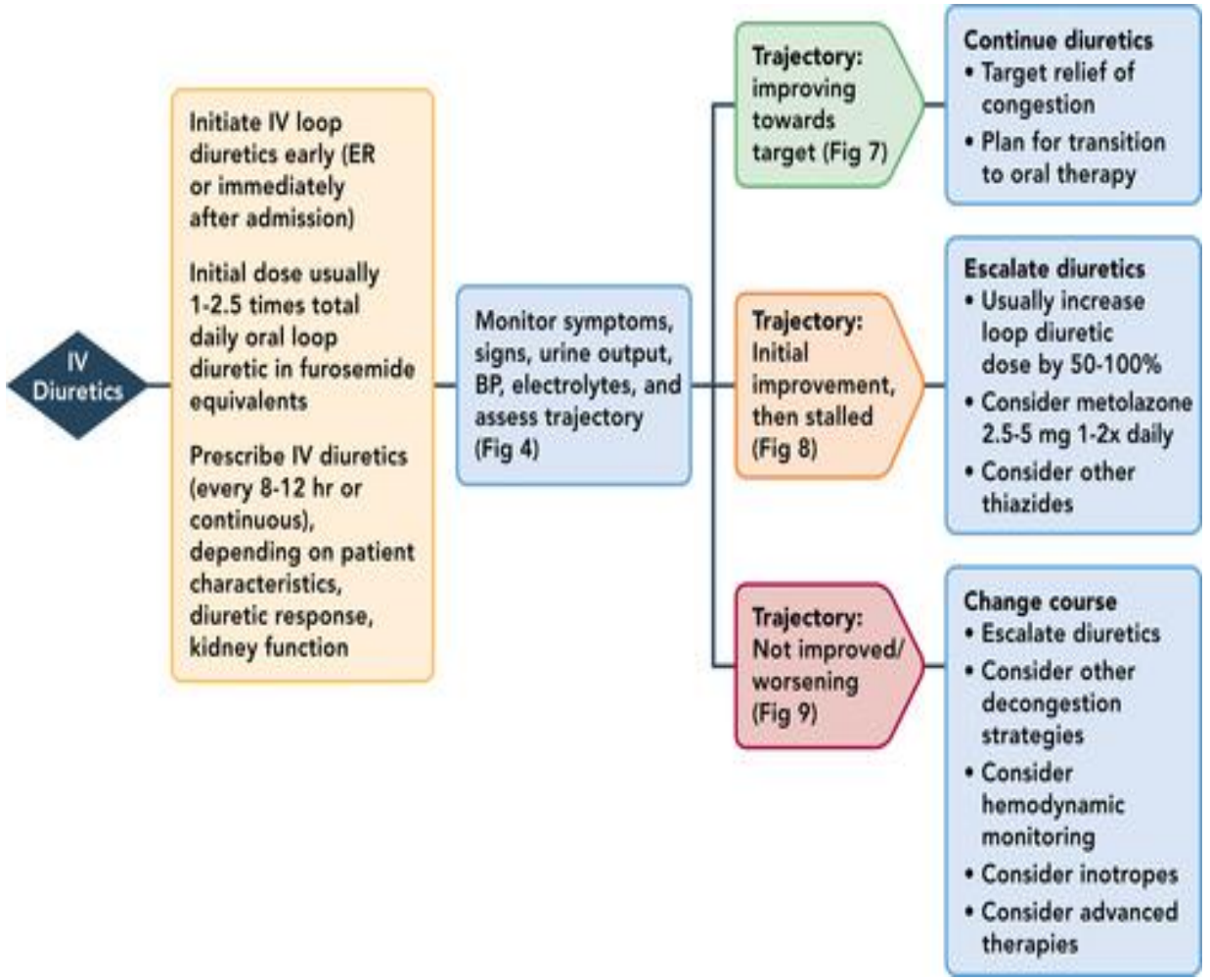
Diuretics

- Bumetanide, furosemide, and torsemide inhibit reabsorption of sodium or chloride at the loop of Henle
- Thiazide and thiazide-like diuretics act in the distal convoluting tubule
- Potassium sparing diuretics (e.g., spironolactone) in the collecting duct



Diuretic Therapy in Different Clinical Trajectory

- Three main trajectories according to changes in patient s/s, labs, images if done, presence or absence of complications, assessment and treatment of comorbidities, and treatment alignment with goals of care

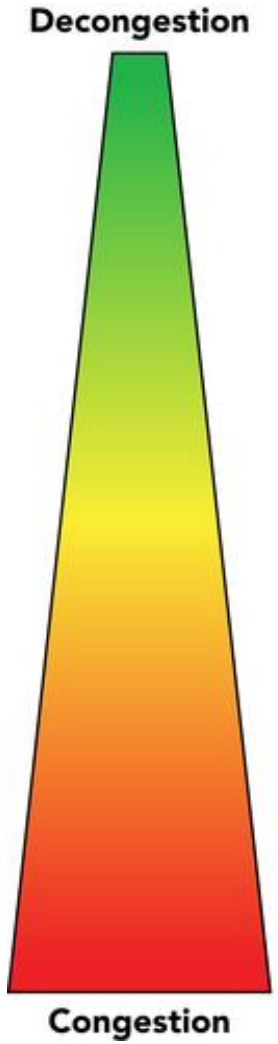


Monitoring

- Goal urine output 3-5 L/day
- BMP and magnesium level at least twice a day
- Adequate replacement of potassium and magnesium
- Daily weight- standing weight preferred
- Strict I/O
- Central venous pressure if central line present

Targets for Decongestion

- The usual goal is for complete decongestion, with absence of signs and clinical symptoms of elevated resting filling pressures.
- Rates of rehospitalization and death are consistently lower in patients rendered free of clinical congestion by the time of discharge



Freedom from clinical congestion

No peripheral edema

No rales

No dyspnea on minimal exertion

No hepatomegaly or congestive GI symptoms

No orthopnea or bendopnea

Jugular venous pressure ≤6-8 mm Hg

No hepatojugular reflex

Common reasons for Residual Congestion

Low cardiac output state
Dominant right heart failure
Advanced renal disease
Symptomatic hypotension
Limitations to patient engagement in self-care

Lack of improvement in signs/symptoms of HF

Lack of decrease in natriuretic peptide levels

Lack of decrease in weight

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Maintenance and Optimization of GDMT

- Hospitalization for HFrEF is a critical opportunity to continue, initiate, and further optimize guideline directed medical therapy (GDMT)
- Continuation of oral GDMT during hospitalization for HF has been shown in registries to lower risk of post discharge death and readmission compared with discontinuation
- In patients experiencing mild decrease of renal function or asymptomatic reduction of blood pressure during HF hospitalization, diuresis and other GDMT should not routinely be discontinued
- In patients with HFrEF, GDMT should be initiated during hospitalization after clinical stability is achieved
- In patients with HFrEF, if discontinuation of GDMT is necessary during hospitalization, it should be reinitiated and further optimized as soon as possible

Guideline Directed Medical Therapy for HFrEF

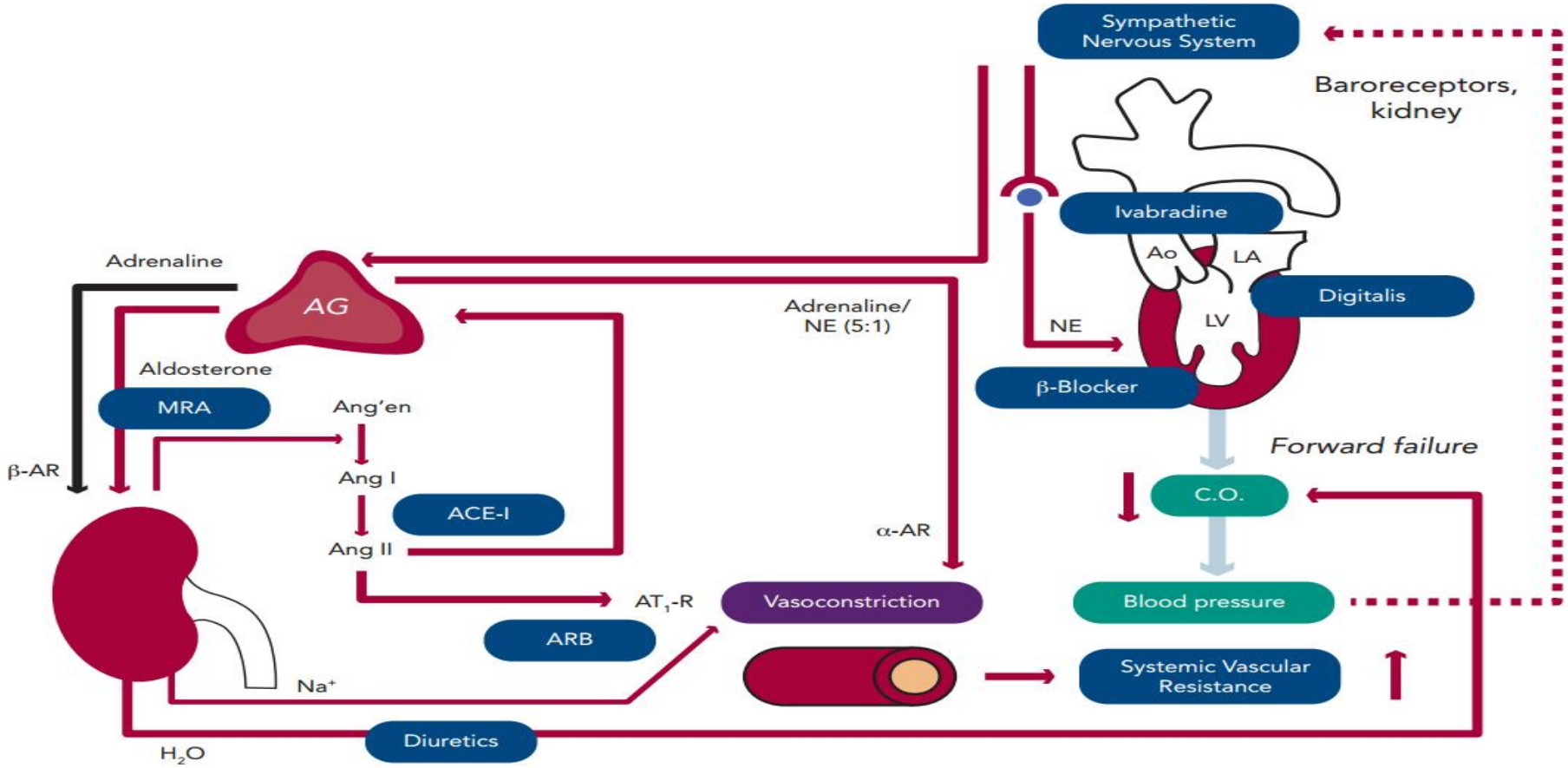
Class 1 Recommendation

- Renin-Angiotensin System Inhibitors
  - Angiotensin receptor-neprilysin inhibitor (ARNI)
  - Angiotensin converting enzyme (ACE) inhibitors
  - Angiotensin receptor blockers (ARB)
- Beta-blockers
- Mineralocorticoid receptor antagonist (MRA)
- Sodium–glucose cotransporter 2 (SGLT2) inhibitor

2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure

Pathophysiology

Figure 1: Pathophysiological Changes in Patients with Heart Failure, Highlighting the Haemodynamic Alterations and Neuroendocrine Activation as well as the Pharmacological Targets

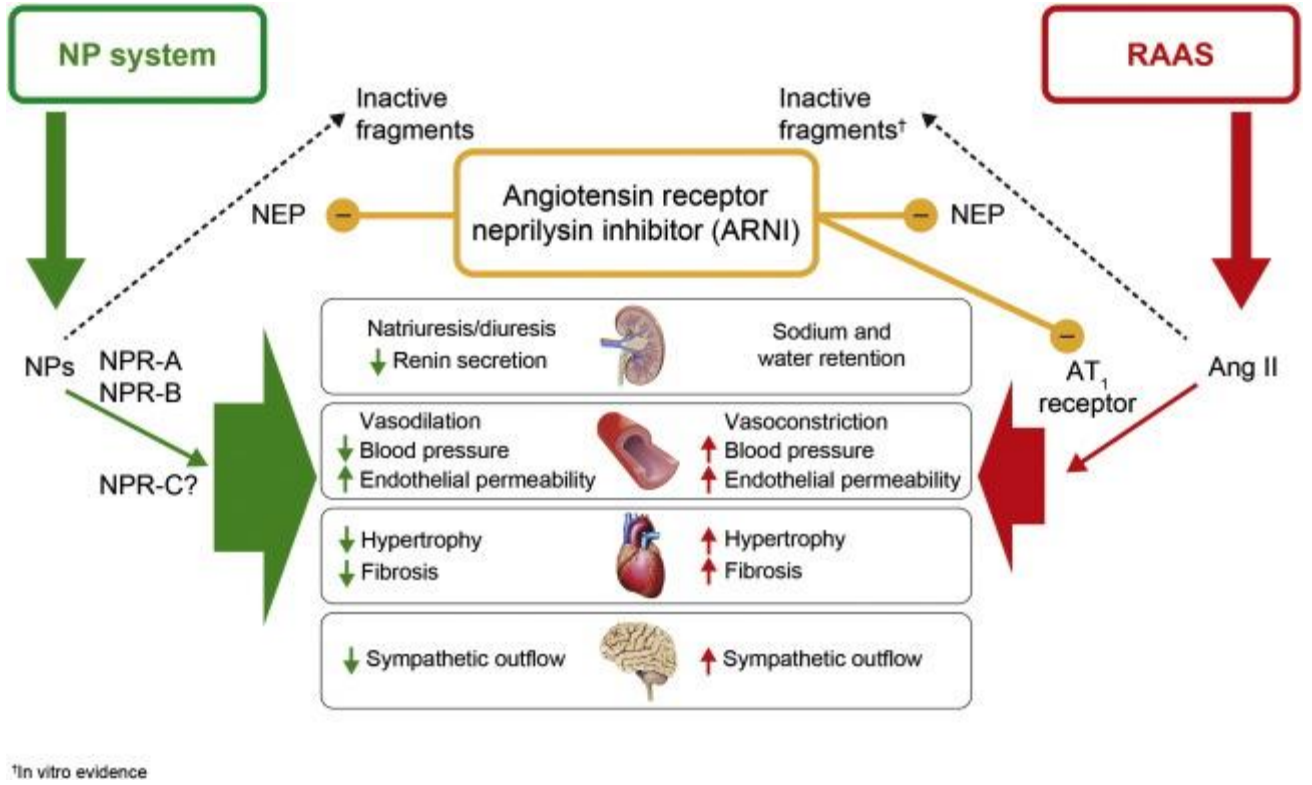


ACE = angiotensin-converting enzyme; AG = adrenal gland; Ang = angiotensin; Ang'en = Angiotensinogen; AO = aorta; AR = adrenergic receptor; ARB = AT1-receptor blockers; C.O. = cardiac output; H_2O = water; LA = left atrial; LV = left ventricle; MRA = mineralocorticoid antagonist; Na^+ = sodium; NE = norepinephrine; SVR = systemic vascular resistance.

C. Maack & M. Bohm (2019)

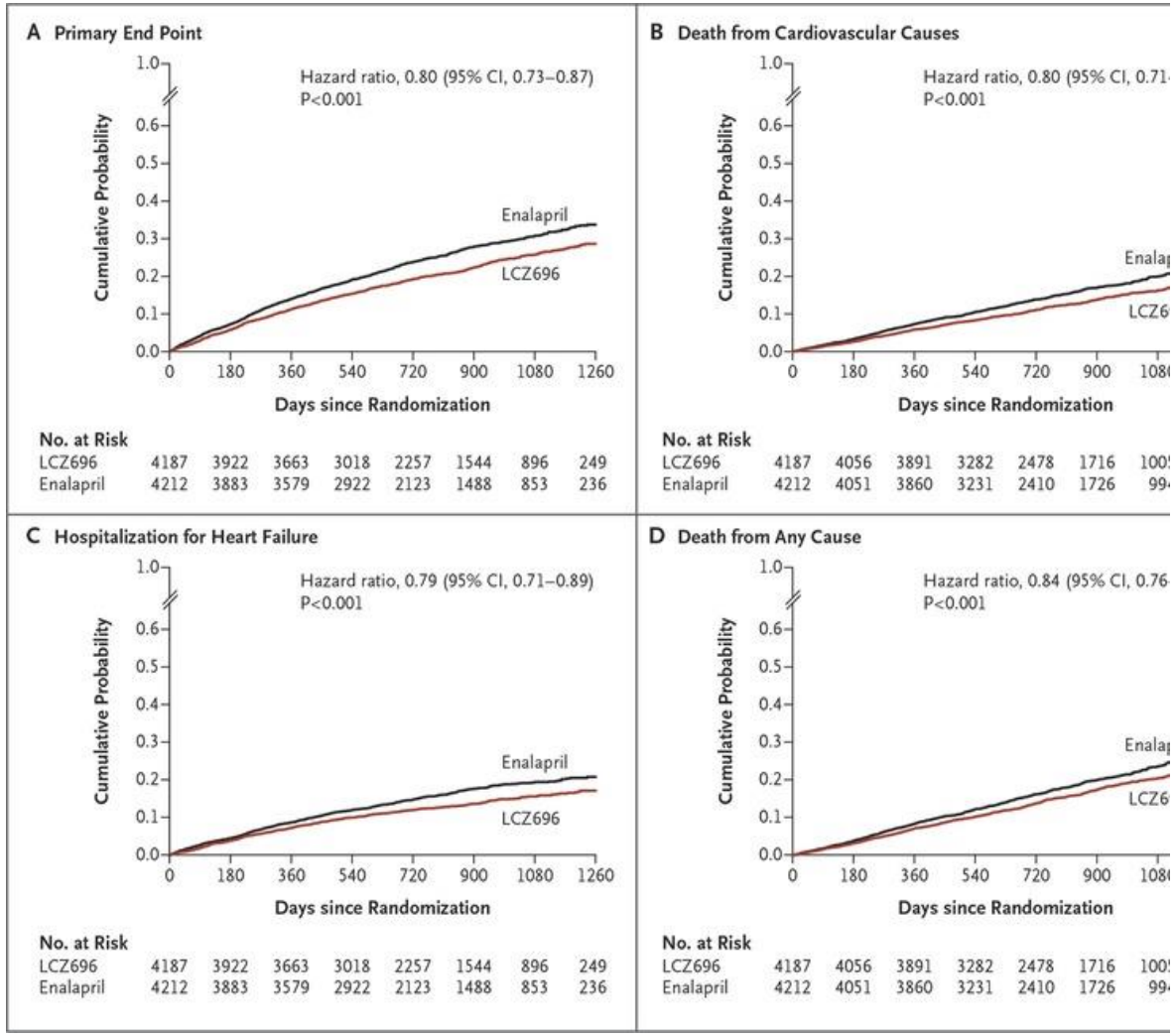
Angiotensin Receptor-Neprilysin Inhibitor (ARNI)

Sacubitril/valsartan (Enresto) is the first in class ARNI that was developed to increase natriuretic peptide levels by preventing their degradation and antagonize the RAAS by blocking type 1 angiotensin receptor.



PARADIGM-HF

- Prospective Comparison of ARNI With ACEI to Determine Impact on Global Mortality and Morbidity in Heart Failure
- Randomized double-blind: 4187 (LCZ696 200 mg BID) and 4212 (Enalapril 10 mg BID)
- Primary outcome: Composite of death from CV causes or hospitalization for HF
- The trial was stopped early, after a median follow-up of 27 months, because the boundary for an overwhelming benefit with LCZ696 had been crossed.



McMurray et al (2014)

Sacubitril/Valsartan

Initial dose	Target dose	Adverse Effects	Monitoring
24/26- 49/51 mg BID	97/103 mg BID	• Hypotension • Hyperkalemia • S Cr increase • Dizziness • Cough • Angioedema	• Blood pressure • Electrolytes • Renal function

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LCZ696 (Entresto) in Advanced Heart Failure (LIFE) Trial

- <1% of the patients in PARADIGM-HF had NYHA class IV HF
- LIFE tested the hypothesis that Sacubitril/Valsartan is superior to valsartan in lowering NT-proBNP in patients with advanced HF
- Sample size: 335 (167 sac/val, 168 Val)

Result:

- Neither treatment with Sacubitril/Valsartan or Valsartan decreased the median NT-proBNP
- No reduction in death or hospitalization
- Small increase in nonlife threatening hyperkalemia

Contraindications and Cautions for Sacubitril/Valsartan

Contraindications	Cautions
Within 36 hours of ACEI use	Renal impairment: • Mild-to-moderate (eGFR 30-59 mL/ min/1.73 m^2): no starting dose adjustment required • Severe* (eGFR <30 mL/min/ 1.73 m^2): reduce starting dose to 24/26 mg twice daily; double the dose every 2–4 weeks to target maintenance dose of 97/103 mg twice daily, as tolerated
History of angioedema with or without an ACEI or ARB	Hepatic impairment: • Mild (Child-Pugh A): no starting dose adjustment required • Moderate (Child-Pugh B): reduce starting dose to 24/26 mg twice daily; double the dose every 2–4 weeks to target maintenance dose of 97/103 mg twice daily, as tolerated
Pregnancy	Renal artery stenosis
Lactation (no data)	Systolic blood pressure <100 mm Hg
Severe hepatic impairment (Child-Pugh C)	Volume depletion

ACE inhibitors/ARB

- ACE inhibitors or ARB if unable to start Entresto.

Sodium–glucose cotransporter 2 (SGLT2) inhibitor

- In patients with symptomatic chronic HFrEF, SGLT2i are recommended to reduce hospitalization for HF and cardiovascular mortality, irrespective of the presence of type 2 diabetes
- The exact mechanism of action remains uncertain
- Addition of SGLT2 inhibitors leads to:
  - Osmotic diuresis and natriuresis
  - Reduction in arterial pressure and stiffness
  - Reduction of preload and afterload blunting of cardiac stress/injury
  - Less hypertrophy and fibrosis
  - Decreased myocardial remodeling

2021 Update to the 2017 ACC Expert Consensus Decision Pathway for Optimization of Heart Failure Treatment

DAPA-HF Trial

Dapagliflozin in Patients with Heart Failure and Reduced Ejection Fraction (DAPA-HF)

- Patients with established HF and a reduced EF (40% or less) regardless of the presence or absence of type 2 diabetes
- Randomized placebo-controlled trial
- 4744 HF patients randomly assigned to Dapagliflozin 10 mg or placebo
- Primary outcome: Worsening HF or CV death
- Findings: CV death, hospitalization for HF, or urgent HF visit occurred in 16.3% of the dapagliflozin group compared with 21.2% of the placebo group ($p < 0.001$).

EMPEROR-Reduced

EMPagliflozin outcomE tRial in Patients With chrOnic heaRt Failure With Reduced Ejection Fraction (EMPEROR-Reduced)

- Design: Randomized double-blind
- Sample size: 3,730 patients with chronic HFrEF (empagliflozin versus placebo)
- Empagliflozin significantly reduced the CV death or HF hospitalization in adults with and without diabetes (19.4% vs 24.7%; hazard ratio: 0.75; 95% CI: 0.65 to 0.86).
- Empagliflozin slowed the decline in the eGFR over time
- Meta-analysis of DAPA-HF and EMPEROR-Reduced showed both agents reduced hospitalization, all-cause death, CV death, and renal outcomes

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SGLT2 inhibitors

	Dapagliflozin (Farxiga)	Empagliflozin (Jardiance)
	Dapagliflozin (Farxiga)	Empagliflozin (Jardiance)
Dose	10 mg	10 mg
Indication	LVEF <40%, NYHA class II-IV	LVEF <40%, NYHA class II-IV
Contraindication	• Type 1 diabetes • H/o hypersensitivity to SGLT2i • Lactation • ESRD	• Type 1 diabetes • H/o hypersensitivity to SGLT2i • Lactation • ESRD
Cautions	• eGFR <30 (Dapa), eGFR <20 (Empa) • Pregnancy • Increased risk of mycotic genital infection • Discontinue at least 3 days before a planned surgery to prevent post-op ketoacidosis. • If A1c well at baseline or known h/o hypoglycemia- may need to wean sulfonylurea or glinide if they are on it and may need to consider decreasing insulin by 20% when starting the therapy. • Consider reducing diuretic as it can cause volume depletion.	

Beta Blockers

- HFrEF stimulates the RAAS and sympathetic system in order to compensate for the reduced EF.
- This activation may accelerate ventricular remodeling.
- Beta blockers prevent ventricular remodeling promoted by the stimulated RAAS and sympathetic system
- Only three beta blockers approved for HF treatment

Beta Blockers

Beta-Blockers	Starting Dose	Target Dose	Adverse Effects	Contraindication
Bisoprolol (Selective β_1 receptor)	1.25 mg once daily	10 mg once daily	• Hypotension • First-degree heart block • Edema • Dizziness • Abdominal pain/diarrhea	• Severe bradycardia • Second-or third-degree heart block in the absence of a pacemaker • Cardiogenic shock • Decompensated HFrEF • Sick sinus syndrome
Carvedilol (β_1, β_2, α_1, receptors)	3.125 mg twice daily	25 mg BID for weight <85 kg and 50 mg BID for weight ≥85 kg		
Metoprolol succinate (Selective β_1 receptor)	12.5–25 mg daily	200 mg daily		

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Aldosterone Antagonist/Mineralocorticoid-receptor antagonists (MRA)

- Aldosterone is an endogenous steroid hormone that increases sodium retention and facilitates magnesium/potassium loss.
- It may ultimately cause myocardial fibrosis, vascular injury, direct vascular damage, and baroreceptor dysfunction leading to the development and progression of HFrEF

MRA

MRA	Starting Dose	Target Dose	Adverse Effects	Contraindication	Monitoring
Eplerenone	25 mg daily	50 mg daily	• Hyperkalemia • Diarrhea • Impaired renal function • Dizziness • Fatigue • Gynecomastia with spironolactone	eGFR <30 mL/min/1.73 m2 or serum potassium >5.0 mEq/L	BMP 1 week, then 4 weeks, then every 6 months after initiating or intensifying MRA, with more frequent testing for clinical instability.
Spironolactone	12.5–25 mg daily	25–50 mg daily			

- Spironolactone, which is chemically similar to progesterone, increases peripheral estradiol formation, potentially leading to adverse events, including gynecomastia, vaginal bleeding, or menstrual irregularities
- These adverse events are not seen with eplerenone because it is selective to the aldosterone receptor

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Vasodilators

- Hydralazine and Isordil shown to reduce mortality in African-Americans patients with NYHA class III–IV HF*r*EF when added to the standard GDMT
- Recommended to reduce morbidity and mortality in patients with current or prior symptomatic HF*r*EF who cannot be given an ACE inhibitor or ARB.
- Both hydralazine and isosorbide dinitrate have vasodilatory effects.
- Isosorbide dinitrate causes a release of nitric oxide that relaxes vascular smooth muscle, affecting both arteries and veins.
- Hydralazine works to selectively relax arterial smooth muscle and may minimize nitrate tolerance

HF GDMT

- It cannot be assumed that oral GDMT will be initiated or optimized after hospitalization for HFrEF.

CHAMP-HF (Change the Management of Patients with Heart Failure) registry data in 2018:

- Only 73%, 66%, and 33% of eligible patients with HFrEF were prescribed ACEi-ARB-ARNi, beta blockers, and MRA therapy, respectively

Claims Data 2016:

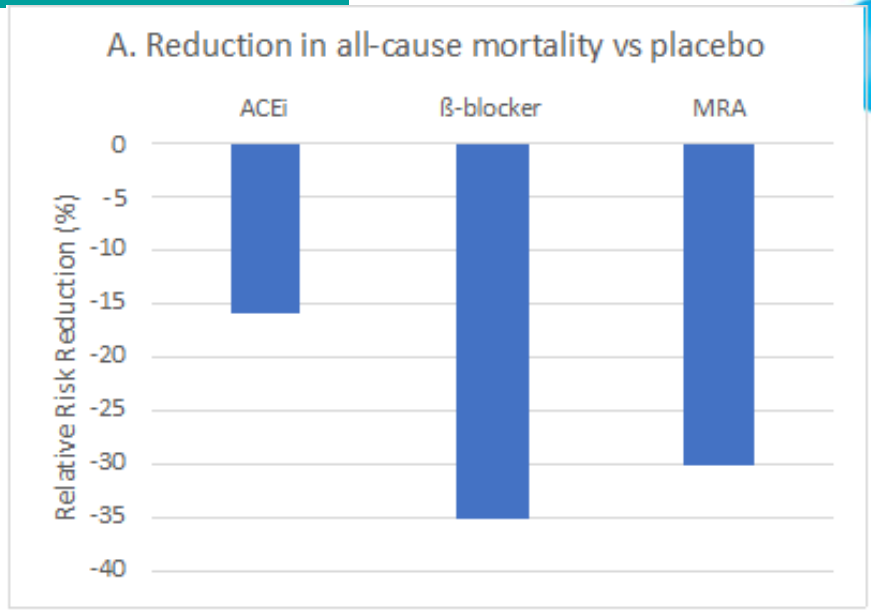
- 42% of patients are not prescribed any GDMT within 30 days post-index hospitalization

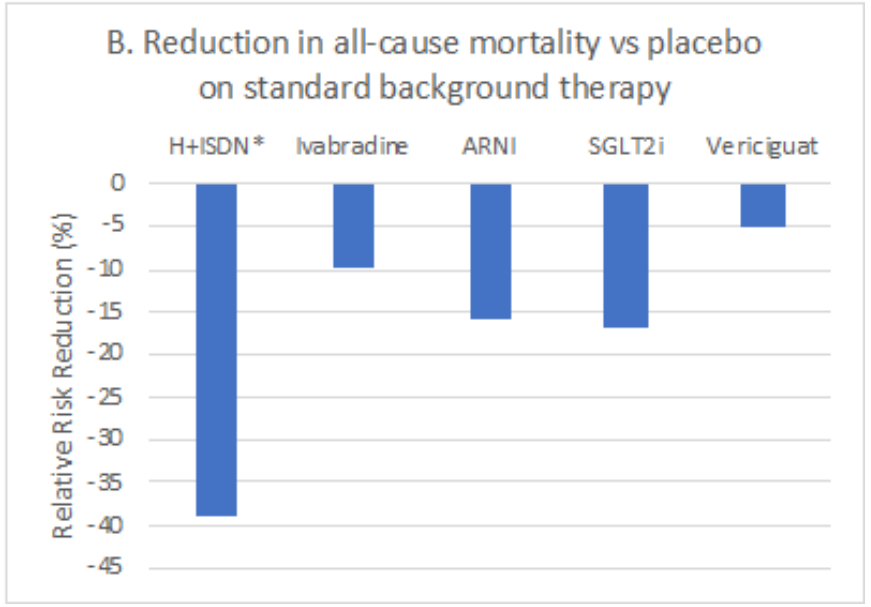
Real World analysis of guideline-based HF therapy data in 2020:

- Approximately 45% are prescribed either no oral GDMT or monotherapy within 1-year after hospitalization.
- Most patients with HFrEF have no changes made to oral GDMT over 12 months despite being discharged on suboptimal doses or no GDMT

GDMT and All Cause Mortality

Mortality reduction with GDMT for HFrEF as reported on different trials



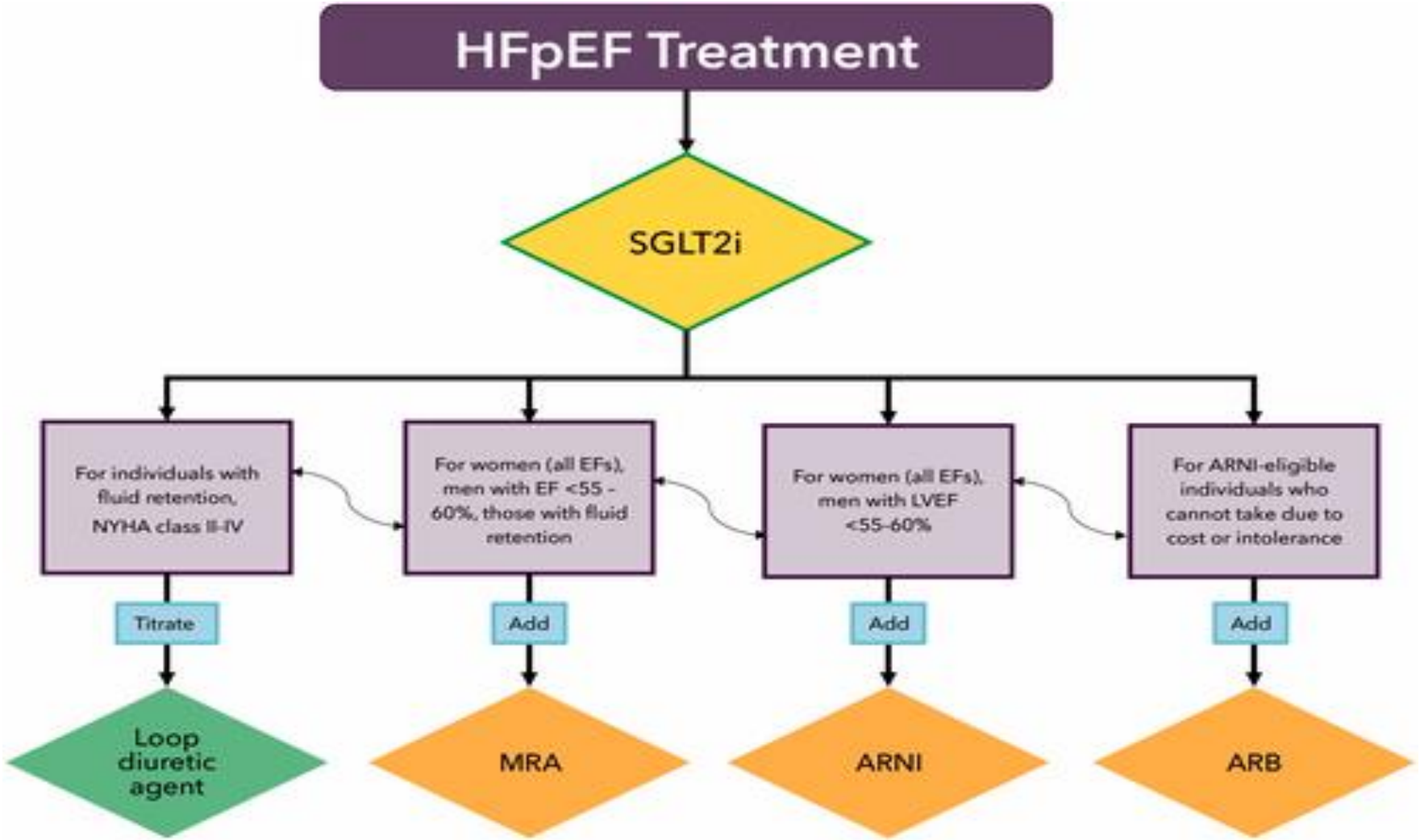


Lee et al. (2020

Heart Failure with Preserved EF

- Diuretic for decongestion
- Hypertension control
- SGLT2i can be beneficial in decreasing HF hospitalizations and cardiovascular mortality
- Management of AF can be useful to improve symptoms
- In selected patients with HFpEF, MRAs, ARB or ARNI may be considered to decrease hospitalizations, particularly among patients with LVEF on the lower end of this spectrum
- Management of comorbidities

Heart Failure with Preserved EF



2023 ACC Expert Consensus Decision Pathway on Management of Heart Failure With Preserved Ejection Fraction

Cold & Wet- Cardiogenic Shock

Definition: State in which ineffective cardiac output (CO) due to a primary cardiac dysfunction results in inadequate end-organ perfusion.

- Clinically presents as hypotension with evidence of organ hypo-perfusion, altered mental status
  - Cold, clammy skin and/or extremities (mottling)
  - Oliguria (< 0.5 ml/kg/hr or < 30 ml/hr)
  - Respiratory distress in the form of pulmonary congestion
  - Organ dysfunction

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Medical Management

- Goal is to restore Cardiac Output and reverse end-organ dysfunction
- Hemodynamic Evaluation
- Inotropes
- Epinephrine
- Vasopressors

Inotropes

- Milrinone and Dobutamine are the two commonly used inotropes
- Both increase cardiac output by increasing the intracellular level of cyclic adenosine monophosphate (cAMP)
- Dobutamine increases cAMP indirectly through adrenergic agonism.
- Milrinone, a phosphodiesterase inhibitor, directly blocks cAMP breakdown

Inotropes

- Indicated in the presence of acute or chronic hemodynamic compromise with end organ dysfunction.
- Bridge to mechanical circulatory support (MCS)
- Bridge to transplant
- Palliative

Cold & Dry

- Evidence of hypoperfusion
- Might be volume depleted
- Management: Cardiogenic shock management as described above
- May need gentle hydration

Medical Management Failure

- Persistent hypotension & hypo-perfusion despite use of 2 or more inotropic and/or vasopressor agents
- Rising lactic acid
- Evolving organ dysfunction
- May Need short term mechanical circulatory support (MCS)

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Preparing for Hospital Discharge

- Many patients hospitalized with HF are discharged too early
- The risk of readmission for HF has been linked to shorter lengths of stay, which may lead to incomplete decongestion, lack of appropriate titration of GDMT, and incomplete translation of plans to post-discharge care.
- For patients requiring diuretic treatment during hospitalization for HF, the discharge regimen should include a plan for adjustment of diuretics to decrease rehospitalizations
- Verifying the effectiveness of oral diuretic therapy prior to discharge, generally requires at least 24 hours of observation after discontinuation of IV diuretics

Discharge- Diuretic

- Maintenance diuretic dosing
  - Torsemide and bumetanide are more reliably absorbed than furosemide and may be considered when daily furosemide doses are high.
  - Kidney dysfunction may lead clinicians to underdose diuretics, despite evidence that transient worsening of creatinine during effective decongestion does not confer long-term decrements in kidney function
- A rescue dosing plan may need to be included in the intended discharge regimen.

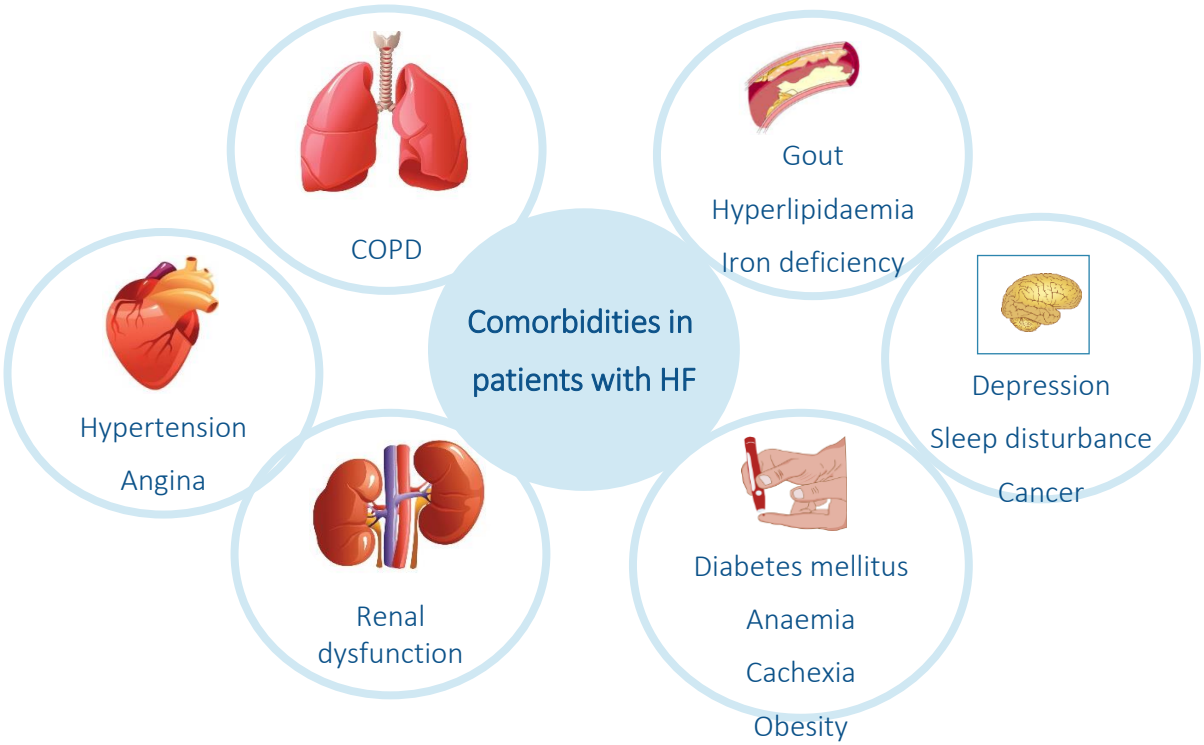
Drugs to Avoid or Used with Caution

- NSAIDs and COX-2 inhibitors
- Thiazolidinediones
- Saxagliptin, Alogliptin
- Flecainide and Disopyramide: Negative inotrope, proarrhythmic effects
- Dronedarone: Negative inotrope
- PDE-3 inhibitors: Cilostazol and Anagrelide
- Trimethoprim-sulfamethoxazole: Use with caution in patients who are taking ACE inhibitor, ARB, ARNI, or MRA
- Cardiotoxic chemotherapy agents: Anthracyclines, high dose cyclophosphamide, trastuzumab and bevacizumab
- Doxazosin: Beta-1-receptor stimulation with increases in renin and aldosterone

Comorbidities in HF

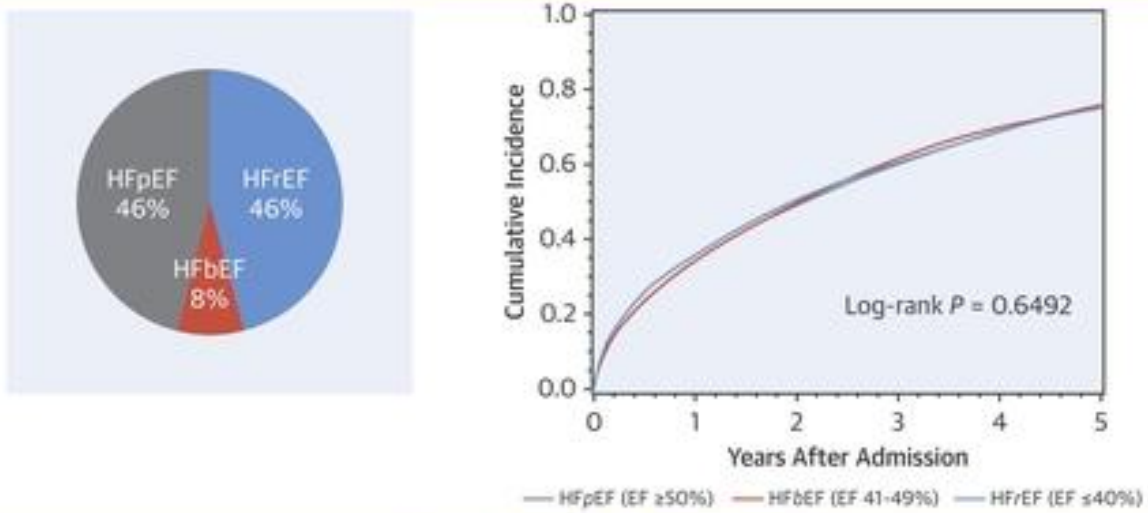
Why comorbidities are relevant in HF:

- Comorbidities may affect the use of treatments for HF
- Most comorbidities are associated with worse clinical status and are predictors of poor prognosis in HF



Heart Failure Hospitalization and Mortality

CENTRAL ILLUSTRATION: 5-Year Outcomes in Patients Hospitalized With HF With Preserved, Borderline, and Reduced EF



Outcomes – 5-Year Event Rates (%)					
	Mortality	Readmission	CV Readmission	HF Readmission	Mortality/Readmission
HFrEF	75.3	82.2	63.9	48.5	96.4
HFbEF	75.7	85.7	63.3	45.2	97.2
HFpEF	75.7	84.0	58.9	40.5	97.3

Shah, K.S. et al. J Am Coll Cardiol. 2017;70(20):2476-86.

Implantable Hemodynamic Monitoring

- c

The CardioMEMS™ HF System measures pulmonary artery (PA) pressure which can indicate worsening HF.

Remote hemodynamic-guided management provides presymptomatic data to inform proactive treatment modifications to prevent heart failure hospitalizations.

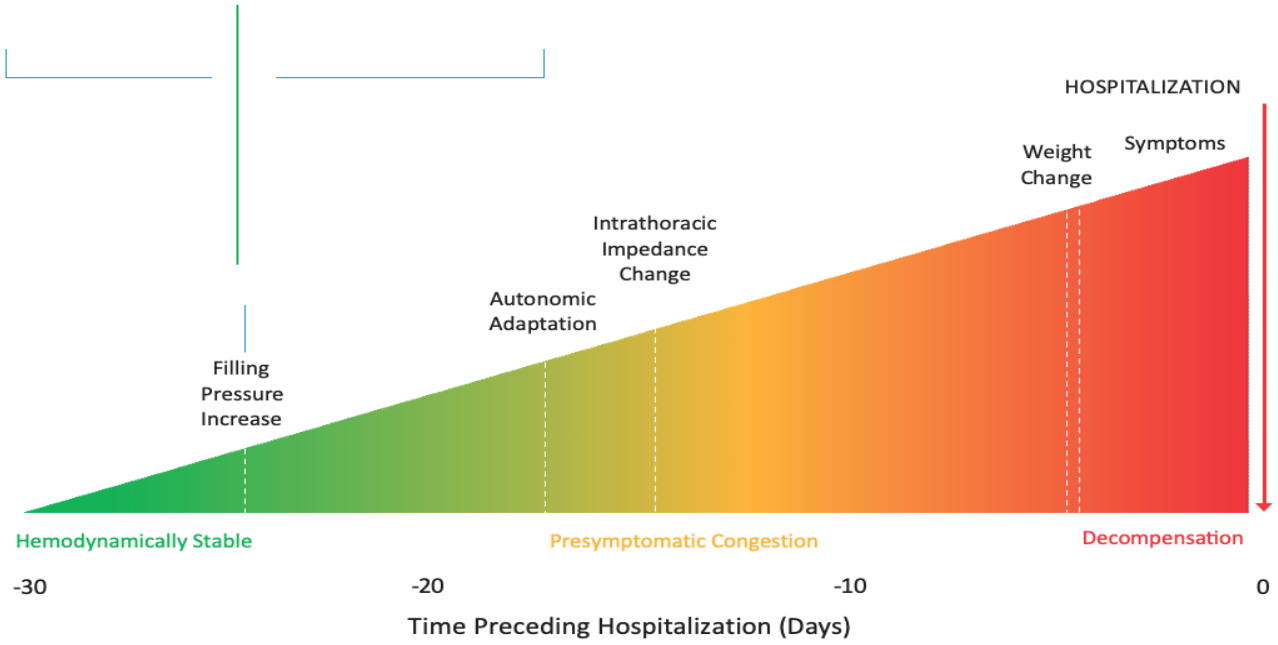
Adamson (2009.)

WEEKS EARLY

With the CardioMEMS™ PA Sensor, PA pressure data can indicate worsening heart failure symptoms weeks before they appear.[1]

SIGNS & SYMPTOMS

Care teams have traditionally had to rely on physical markers like fatigue, shortness of breath and weight gain.



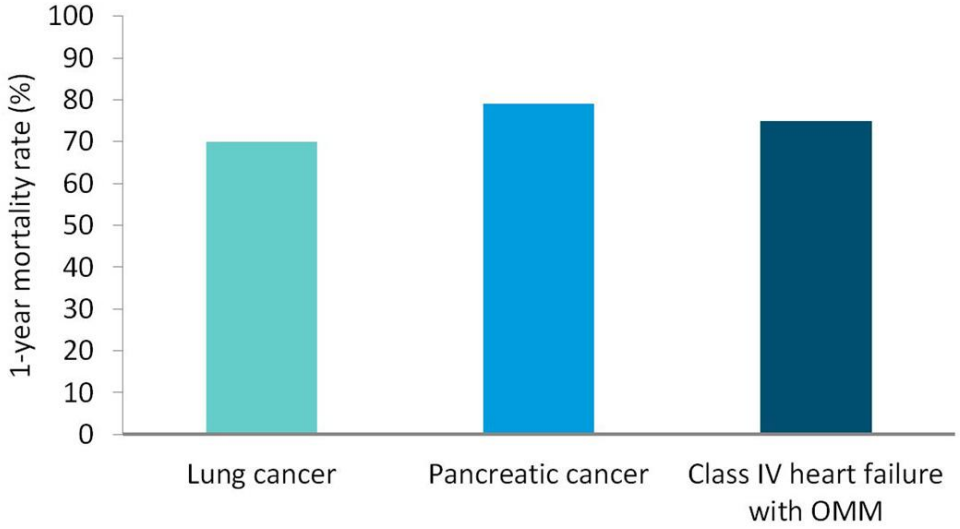
Advanced Heart Failure

- Some patients will progress and develop persistently severe symptoms despite maximum GDMT
- Described as “advanced”, “end-stage”, “refractory”
- Severe symptoms with dyspnea and/or fatigue at rest or with minimal exertion
- Episodes of fluid retention and/or reduced CO at rest

Mortality

Class IV heart failure mortality is similar to that of aggressive malignancies[1-3]

MORTALITY AT 1 YEAR



OMM = optimal medical management.

References: 1. Cetin K, Ettinger DS, Hei Y-J, et al. Survival by histological subtype in stage IV nonsmall cell lung cancer based on data from the Surveillance, Epidemiology and End Results Program. *Clin Epidemiol*. 2011;3:139-148. **2.** Wang Y, Schrag D, Brooks GA, et al. National trends in pancreatic cancer outcomes and pattern of care among Medicare beneficiaries, 2000-2010. *Cancer*. 2014;120(7):1050-1058. **3.** Lee DS, Austin PC, Rouleau JL, et al. Predicting mortality among patients hospitalized for heart failure: derivation and validation of a clinical model. *JAMA*. 2003;290(19):2581-2587.

Triggers For Referral to HF Specialist/Program

I-NEED-HELP.

- **I:** Intravenous inotropes
- **N:** New York Heart Association (NYHA) class IIIB/IV or persistently elevated natriuretic peptides
- **E:** End-organ dysfunction
- **E:** EF ≤35%
- **D:** Defibrillator shocks
- **H:** Hospitalizations >1
- **E:** Edema despite escalating diuretics
- **L:** Low systolic BP ≤90, high heart rate
- **P:** Prognostic medication; progressive intolerance or down-titration of GDMT.

Triggers For Referral to HF Specialist/Program HFpEF

When to Refer: Cardiovascular Specialist to Advanced Heart Failure Specialist

Acronym to assist in decision making for HF specialist referral: INHALE

I

In need of diagnosis

Lack of conventional HFpEF risk factors; exercise-intolerant-only phenotype

N

Nonresponsive to diuretic agents or medical therapy; Natriuretic peptides extremely high

Resistance to diuretic agents or medical therapy; progressive symptoms; NT-proBNP >3,000 pg/mL, BNP >1,000 pg/mL

H

Hospitalized frequently for HF

2 or more HF hospitalizations in the past year

A

Acute or chronic end-organ dysfunction

Worsening kidney or liver function; cardiac cachexia

L

Low blood pressure

Systolic blood pressure <100 mm Hg

E

Evidence of HFpEF mimics

Management of rare or unusual cardiomyopathies

2023 ACC Expert Consensus Decision Pathway on Management of Heart Failure With Preserved Ejection Fraction

OU Health

Advanced HF Treatment

Limited treatment option

- Heart transplantation
- Mechanical circulatory support (MCS)
    - Left ventricular assist device (LVAD)
    - Total artificial heart (TAH)
- Long-term inotropic therapy
- Palliative care

Conclusion

- Patients with HF carry poor prognosis
- Adequate decongestion and optimization of GDMT is key in reducing readmission and improving outcomes in hospitalized HF patients.
- HF GDMT shown to improve symptoms, cardiac function, and mortality. Therefore, every effort should be made to optimize GDMT
- Risk of mortality increases with each hospitalization
- Timely identification of advance HF and referral to HF specialist is essential to decrease mortality and morbidity in advanced HF patients.

Questions?

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