



Congestive Heart Failure in Primary Care



Mohammed Hamed



2026



Outline



1

Define heart failure and classify its stages and phenotypes according to current guidelines.



2

Recognise the clinical presentation of heart failure and apply an evidence-based diagnostic approach in primary care.



3

Initiate and optimise guideline-directed medical therapy for HFrEF and understand the management principles of HFpEF.



4

Identify patients who require specialist referral for advanced therapies, device therapy, or multidisciplinary heart failure care.



5

Implement strategies for heart failure prevention, long-term monitoring, and management of common comorbidities in primary care.



Clinical Scenario

- Mr Greg is a 70-year-old retired man with a history of long-standing hypertension, obesity, OSA, COPD and permanent atrial fibrillation. Over the past few years, he has experienced recurrent hospital admissions for worsening heart failure and is currently functioning at **NYHA Class IV**. Review of his previous echocardiograms revealed that as early as **2010**, he had **preserved left ventricular ejection fraction, mild diastolic dysfunction (impaired relaxation), and left atrial enlargement**, while remaining asymptomatic and in sinus rhythm. Seven years later, he developed **symptomatic heart failure with preserved ejection fraction (HFpEF)**, followed by **permanent atrial fibrillation** and **pulmonary hypertension**. His most recent echocardiogram demonstrates preserved systolic function but **severe diastolic dysfunction**, illustrating the gradual progression from **Stage B (Pre-HF)** to advanced symptomatic HFpEF.



Definition of Heart Failure

Heart failure is a **clinical syndrome** caused by structural or functional cardiac abnormalities resulting in:



- **Elevated intracardiac filling pressures**



- **Inadequate cardiac output**



- **Or both**, at rest or during exercise
-

Elevated filling pressures may be identified by:



- Natriuretic peptide levels



- Echocardiography



- Cardiac catheterisation

1. COMMON SYMPTOMS



Common Symptoms

- Breathlessness
- Reduced exercise tolerance
- Fatigue
- Orthopnoea
- Ankle swelling



Less Common Symptoms

- Nocturnal cough
- Wheeze
- Abdominal bloating
- Loss of appetite
- Dizziness
- Syncope



Clinical Signs

- Raised jugular venous pressure
- Pulmonary crackles
- Peripheral oedema
- Tachycardia
- Third heart sound (S3)
- Laterally displaced apex beat
- Weight gain or cachexia
- Oliguria

2. RISK FACTORS AND ASSOCIATED CONDITIONS



Previous myocardial infarction



Coronary artery disease



Diabetes mellitus



Chronic kidney disease



Alcohol misuse



Cardiotoxic chemotherapy



Family history of cardiomyopathy



Family history of sudden cardiac death

3. ACUTE AND CHRONIC PRESENTATIONS



Chronic Heart Failure

- Established diagnosis of heart failure
- Gradual onset or progression of symptoms



Decompensated Heart Failure

Characterised by worsening:

- Congestion
- Symptom burden
- Cardiac output



Acute Heart Failure

May require hospital admission because of:

- Significant congestion
- Need for intravenous diuretics
- Vasodilator or inotropic support
- High symptom burden
- Haemodynamic compromise
- Risk of clinical deterioration



Primary Care Assessment

The initial assessment should focus on:

- Heart failure symptoms and functional status
- Symptoms suggestive of myocardial ischaemia or angina
- Recent viral illness
- Neoplastic or endocrine disease
- Constitutional symptoms
- Past medical history
- Substance use history
- Family history of heart disease or sudden cardiac death
- Recent physical or emotional stressors



Clinical Priorities

- Assess symptom severity
- Identify the underlying cause
- Initiate appropriate investigations
- Commence GDMT promptly
- Arrange cardiology referral when indicated

1. CLASSIFICATION OF HEART FAILURE ACCORDING TO LEFT VENTRICULAR EJECTION FRACTION¹¹

Heart failure with reduced ejection fraction

- Symptoms with or without signs of heart failure
- LVEF $\leq 40\%$

Heart failure with mildly reduced ejection fraction

- Symptoms with or without signs of heart failure
- LVEF 41–49%
- The diagnosis of other structural heart disease, such as increased left atrial size or left ventricular hypertrophy, or echocardiographic evidence of impaired left ventricular filling, makes this diagnosis more likely

Heart failure with preserved ejection fraction

- Symptoms with or without signs of heart failure
- LVEF $\geq 50\%$
- Objective evidence of cardiac structural and/or functional abnormalities consistent with the presence of left ventricular diastolic dysfunction and raised left ventricular filling pressures, including raised levels of natriuretic peptides
- A greater number of abnormalities signifies a higher likelihood of heart failure with preserved ejection fraction

Abbreviation: LVEF = left ventricular ejection fraction.

2. NEW YORK HEART ASSOCIATION FUNCTIONAL CLASSIFICATION BASED ON SEVERITY OF HEART FAILURE SYMPTOMS AND PHYSICAL ACTIVITY^{1,2}

Class I

- No limitation of physical activity
- Ordinary physical activity does not cause undue breathlessness, fatigue or palpitations

Class II

- Slight limitation of physical activity
- Comfortable at rest but ordinary physical activity results in disproportionate breathlessness, fatigue or palpitations

Class III

- Marked limitation of physical activity, but remains comfortable at rest
- Less than ordinary activity is enough to result in disproportionate breathlessness, fatigue or palpitations

Class IV

- Unable to continue any physical activity without discomfort
- Symptoms at rest may be present
- If physical activity is undertaken, discomfort is increased

CAUSES OF HFrEF



HFrEF has a diverse range of underlying causes, making identification of the aetiology essential for **appropriate treatment and prognostication**.



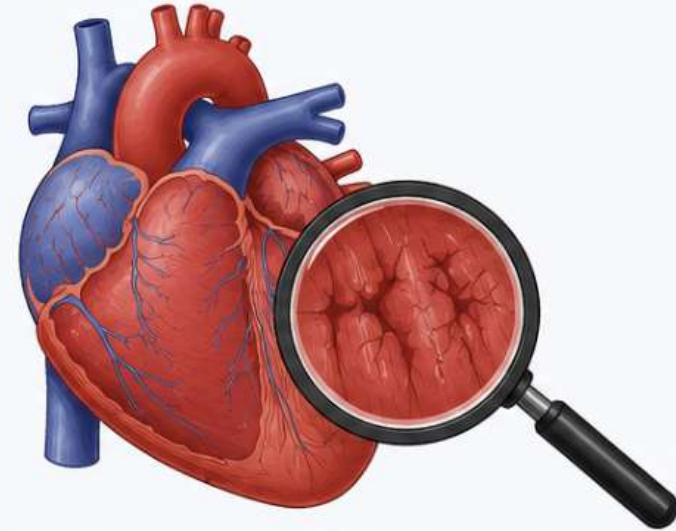
The most common pathological substrate is **myocardial dysfunction**, which is typically systolic, although many patients also have concomitant diastolic dysfunction.



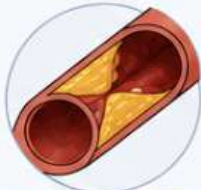
Disease involving the:

- Cardiac valves
- Endocardium
- Pericardium
- Cardiac rhythm and conduction system

may also contribute to HFrEF.



COMMON CAUSES



Ischaemic heart disease

Coronary artery disease leading to reduced blood flow and myocardial dysfunction.



Myocardial infarction

Irreversible myocardial injury and scarring resulting in systolic dysfunction.



Hypertension

Chronic pressure overload leading to left ventricular hypertrophy and dysfunction.



Valvular heart disease

Valvular abnormalities causing volume or pressure overload and ventricular dysfunction.



COMMON CAUSES OF ACUTE DECOMPENSATION

- Myocardial ischaemia or infarction
- Arrhythmias
- Infection
- Anaemia
- Thyroid disease
- Acute hypertension or Takotsubo cardiomyopathy
- Acute kidney injury
- Mechanical complications (e.g. acute valvular regurgitation or ventricular septal rupture)
- Aggravating medications
- Non-adherence to treatment



KEY MESSAGE: Even patients with mild symptoms remain at significant risk of hospitalisation and mortality. Early diagnosis, optimisation of GDMT, and regular follow-up are fundamental components of heart failure management in primary care.



Common Causes of Acute Decompensation



- Myocardial ischaemia or infarction



- Arrhythmias



- Infection



- Anaemia



- Thyroid disease



- Acute hypertension or Takotsubo cardiomyopathy



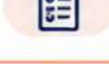
- Acute kidney injury



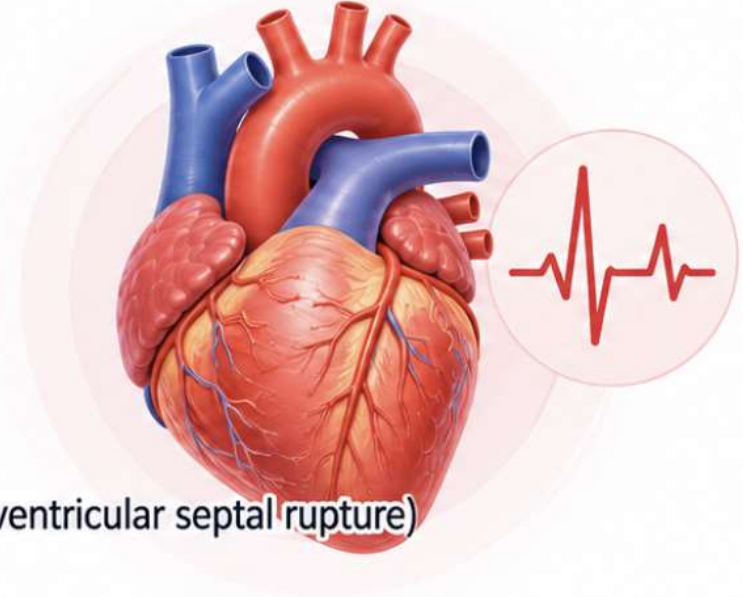
- Mechanical complications (e.g. acute valvular regurgitation or ventricular septal rupture)



- Aggravating medications



- Non-adherence to treatment



Key Message: Even patients with mild symptoms remain at significant risk of hospitalisation and mortality. Early diagnosis, optimisation of GDMT, and regular follow-up are fundamental components of heart failure management in primary care.

CAUSES OF HFrEF



HFrEF has a diverse range of underlying causes, making identification of the aetiology essential for **appropriate treatment and prognostication**.



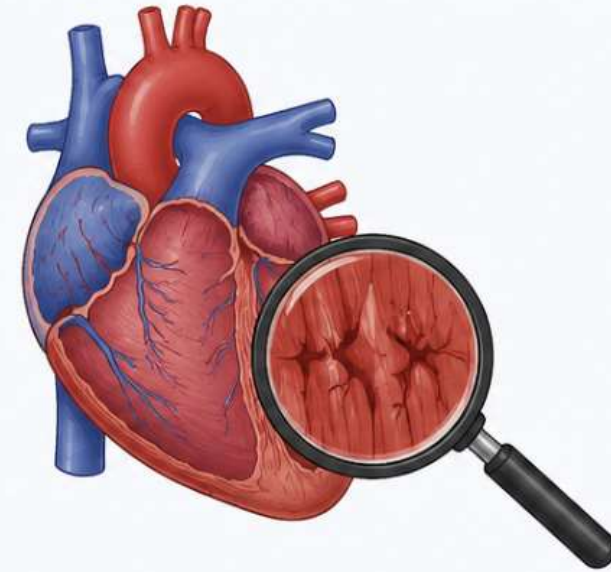
The most common pathological substrate is **myocardial dysfunction**, which is typically systolic, although many patients also have concomitant diastolic dysfunction.



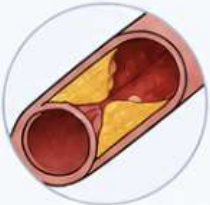
Disease involving the:

- Cardiac valves
- Endocardium
- Pericardium
- Cardiac rhythm and conduction system

may also contribute to HFrEF.



COMMON CAUSES



Ischaemic heart disease

Coronary artery disease leading to reduced blood flow and myocardial dysfunction.



Myocardial infarction

Irreversible myocardial injury and scarring resulting in systolic dysfunction.



Hypertension

Chronic pressure overload leading to left ventricular hypertrophy and dysfunction.



Valvular heart disease

Valvular abnormalities causing volume or pressure overload and ventricular dysfunction.



KEY MESSAGE: Identifying the underlying cause of HFrEF is fundamental to guiding investigations, risk stratification, treatment selection, and prognosis.

CAUSES	PRESENTATIONS AND ASSOCIATED FEATURES	SPECIFIC INVESTIGATIONS TO CONSIDER
 Coronary artery disease	• Myocardial infarction • Angina • Arrhythmias (e.g. ventricular tachycardia) • Complete heart block	• Glycated haemoglobin levels • Lipid levels • Imaging stress test (stress echocardiogram, myocardial perfusion scan) • Referral to cardiology for further evaluation
 Hypertension	• Malignant hypertension • Acute pulmonary oedema	• 24-hour ambulatory blood pressure • Renal artery imaging • Plasma metanephrines • Renin and aldosterone levels
 Valvular disease	• Primary valve disease • Secondary valve disease (e.g. functional mitral regurgitation or tricuspid regurgitation)	• Echocardiography (transthoracic, transoesophageal)
 Arrhythmias and pacing	• Atrial tachyarrhythmias • Ventricular arrhythmias • Right ventricular pacing	• Extended rhythm monitoring (e.g. Holter or HeartBug monitors) • Device check
 Cardiomyopathies	• Dilated, hypertrophic, restrictive, arrhythmogenic or peripartum presentations • Stress-related • Takotsubo syndrome • Toxins: alcohol, cocaine, iron, copper	• Depends on aetiology: referral to cardiology for further investigation; toxicology; liver function tests • ECG features: high or low voltage conduction, repolarisation changes, long QTc
 Congenital heart disease	• Congenitally corrected or repaired transposition of the great arteries • Shunt lesions • Repaired Tetralogy of Fallot • Ebstein's anomaly	• Referral to cardiology for further investigation
 Infective	• Viral myocarditis • Chagas disease • HIV infection	• Serology • Referral to cardiology for further investigation
 Drug-induced	• Anthracyclines • Trastuzumab • Vascular endothelial growth factor inhibitors • Immune checkpoint inhibitors • Proteasome inhibitors • Rapidly accelerated fibrosarcoma/mitogen-activated protein-kinase kinase inhibitors	• Referral to cardiology for further investigation

CAUSES	PRESENTATIONS AND ASSOCIATED FEATURES	SPECIFIC INVESTIGATIONS TO CONSIDER
 Infiltrative	• Amyloid • Sarcoidosis • Neoplastic	• Serum electrophoresis • Serum free light chains • Bence Jones proteins • Bone scintigraphy • Serum ACE (sarcoidosis) • Referral to cardiology for further investigation
 Storage disorders	• Haemochromatosis • Fabry disease • Glycogen storage diseases	• Iron studies • Alpha-galactosidase A • Genetic testing • Referral to cardiology for further investigation
 Endomyocardial disease	• Radiotherapy • Endomyocardial fibrosis or eosinophilia • Carcinoid	• 24-hour urine 5-hydroxyindoleacetic acid • Referral to cardiology for further investigation
 Pericardial disease	• Calcification • Infiltrative	• Chest CT • Referral to cardiology for further investigation
 Metabolic	• Thyroid or endocrine disease • Nutritional disease (thiamine, vitamin B1, selenium deficiencies) • Autoimmune disease	• Thyroid function tests • Plasma metanephrines • Renin and aldosterone • Cortisol • Specific plasma nutrients • Antinuclear antibodies and antineutrophil cytoplasmic antibodies • Rheumatology review
 Neuromuscular disease	• Friedreich's ataxia • Muscular dystrophy	• Nerve conduction studies • Genetic tests • Creatine kinase levels • Referral to neurology

INVESTIGATIONS FOR SUSPECTED HFrEF



INITIAL PRIMARY CARE WORKUP



Laboratory Investigations

- Full blood count (FBC)
- Urea, electrolytes and creatinine (UEC)
- Liver function tests (LFTs)
- Thyroid function tests (TFTs)
- B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP)



Clinical Pearl: In the non-acute setting, heart failure is unlikely if:

- "BNP <35 pg/mL, or"
- "NT-proBNP ≤125 pg/mL"

SPECIALIST CARDIOLOGY INVESTIGATIONS

Further investigations are guided by the suspected aetiology and disease severity and may include:



- CT coronary angiography



- Cardiac magnetic resonance imaging (MRI)



- Invasive coronary angiography



- Left and right heart catheterisation



- Cardiopulmonary exercise testing



Key Message: BNP/NT-proBNP testing, ECG, chest X-ray, and transthoracic echocardiography form the cornerstone of the initial primary care evaluation of suspected HFrEF, with specialist investigations directed by the underlying cause and severity of disease.

INITIAL INVESTIGATIONS



- 12-lead ECG



- Chest X-ray



- Transthoracic echocardiography



ECG FINDINGS SUGGESTIVE OF HEART FAILURE

- Atrial fibrillation
- ST-segment abnormalities
- Pathological Q waves
- Left ventricular hypertrophy
- Broad QRS complexes



CHEST X-RAY

May demonstrate:

- Pulmonary oedema
- Cardiomegaly
- Pulmonary congestion



Important: A normal chest X-ray does not exclude heart failure.

ADDITIONAL INVESTIGATIONS



- Twenty-four-hour ambulatory blood pressure monitoring may be considered when hypertension is suspected.



Indications for Cardiology Referral



Refer All Patients With:

- Suspected new-onset heart failure (where possible)
- Persistently reduced LVEF $\leq 30\%$ despite guideline-directed medical therapy (GDMT)
- Uncertain aetiology or contributing cardiac pathology
- Chest pain or suspected myocardial ischaemia



High-Risk Features Requiring Specialist Review

- Persistent NYHA Class III–IV symptoms
- Previous inotrope use
- Elevated serum creatinine
- Atrial fibrillation
- Palpitations or syncope suspicious for ventricular arrhythmia
- Recurrent implantable cardioverter-defibrillator (ICD) shocks
- Multiple heart failure hospitalisations
- Clinical deterioration despite treatment



Key Message: Early cardiology referral facilitates accurate diagnosis, optimisation of GDMT, timely consideration of device therapy, and improved long-term outcomes in patients with HFrEF.



THE HEART FAILURE TRAJECTORY

Heart failure exists along a clinical continuum:



AT RISK OF HEART FAILURE

Patients with risk factors but no structural heart disease or symptoms.



PRE-HEART FAILURE

Structural heart changes or abnormal function but no symptoms.



SYMPTOMATIC HEART FAILURE

Development of symptoms (e.g. dyspnoea, fatigue, fluid retention) that impact quality of life.



ADVANCED HEART FAILURE

Severe symptoms at rest or with minimal exertion; frequent hospitalisations.



Early identification provides an opportunity to prevent progression to clinically significant disease.



CLINICAL COURSE AND PROGNOSIS

The trajectory of heart failure varies according to its underlying cause.

POTENTIAL FOR RECOVERY

Patients with the following conditions may demonstrate substantial improvement or recovery:



Viral myocarditis



Stress (Takotsubo) cardiomyopathy



Peripartum cardiomyopathy



Tachycardia-induced cardiomyopathy



Some patients may experience significant improvement or normalisation of LVEF following optimal GDMT and device therapy.



PROGRESSIVE DISEASE

Others may develop progressive heart failure requiring:



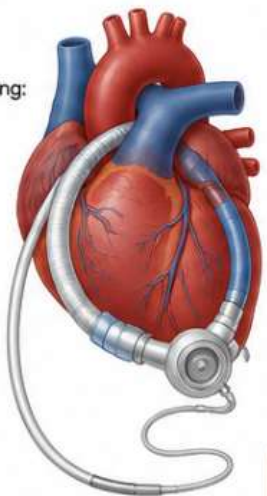
Advanced heart failure therapies



Left ventricular assist devices (LVADs)



Cardiac transplantation



PROGNOSIS

Despite advances in therapy, heart failure remains associated with significant mortality:



Approximately

20%

mortality at one year



Approximately

53%

mortality at five years



KEY MESSAGE: Heart failure is a dynamic disease. Early diagnosis, optimisation of GDMT, and longitudinal primary care management are essential to prevent progression and improve long-term outcomes.

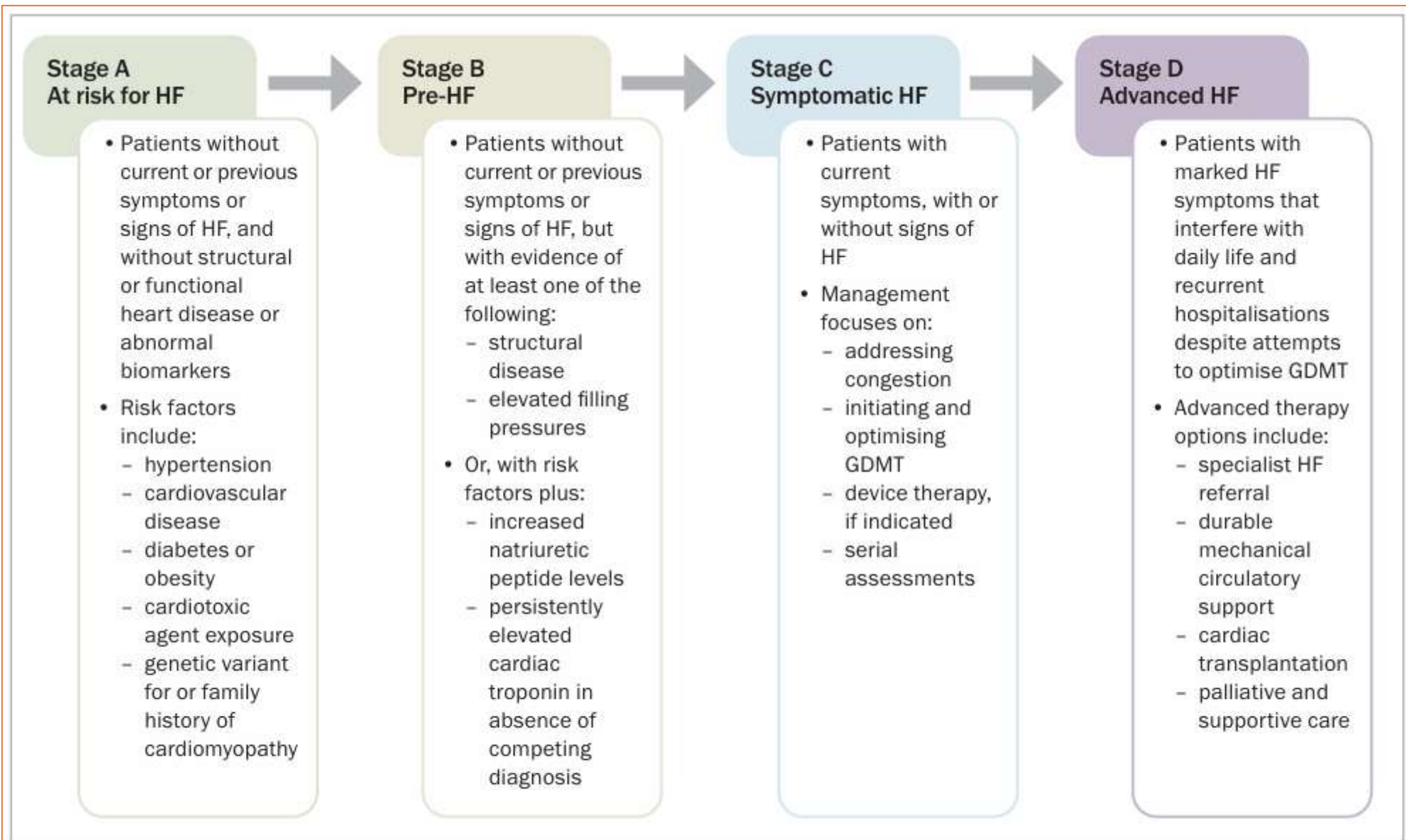


Figure. American College of Cardiology and American Heart Association stages of heart failure.²

Abbreviations: GDMT = guideline-directed medical therapy; HF = heart failure.



Prevention of Heart Failure

Management of Modifiable Risk Factors

For patients at risk of heart failure, primary prevention is essential to prevent disease progression.

Key strategies include:

- ✓ Optimal blood pressure control
- ✓ Good glycaemic control in patients with diabetes
- ✓ Consider SGLT2 inhibitors in patients with diabetes and high cardiovascular risk
- ✓ Regular physical activity
- ✓ Achieving and maintaining a healthy weight
- ✓ Smoking cessation
- ✓ Adherence to healthy dietary patterns, including:
 - Wholegrain diets
 - Plant-based diets
 - Mediterranean diets



Optimal blood pressure control



Good glycaemic control in patients with diabetes



Consider SGLT2 inhibitors in patients with diabetes and high cardiovascular risk



Regular physical activity



Achieving and maintaining a healthy weight



Smoking cessation



Adherence to healthy dietary patterns, including:



Wholegrain diets



Plant-based diets



Mediterranean diets



Clinical Pearl: Hypertension is one of the most important modifiable risk factors for developing symptomatic heart failure.



GOALS OF GUIDELINE-DIRECTED MEDICAL THERAPY (GDMT)

Beyond relieving congestion, the aims of treatment are to:



Reduce mortality



Prevent recurrent heart failure hospitalisations



Improve symptoms and functional capacity



Improve quality of life



Slow disease progression



TREAT THE UNDERLYING CAUSE

Management should include identifying and treating reversible causes when present, including:



Atrial fibrillation and tachyarrhythmias



Coronary artery disease requiring revascularisation



Specific cardiomyopathies
(e.g. transthyretin cardiac amyloidosis)



THE FOUR PILLARS OF HFrEF THERAPY

Guideline-directed medical therapy consists of four foundational therapies:

1

**ARNI (preferred) or
ACE inhibitor/ARB**



2

**Evidence-based
Beta Blocker**



3

**Mineralocorticoid
Receptor Antagonist (MRA)**



4

SGLT2 Inhibitor



TREATMENT PRINCIPLES

- Initiate all four therapies as early as possible.
- Rapid up-titration is preferred when tolerated.
- Simultaneous initiation is favoured over traditional stepwise escalation.
- Continue optimisation to maximally tolerated doses.



Clinical Pearl: SGLT2 inhibitors improve outcomes in HFrEF regardless of diabetes status.



MEDICATIONS TO AVOID IN HFrEF

Avoid the following medications unless specifically recommended by a cardiologist:



Verapamil



Diltiazem



Thiazolidinediones (glitazones)



Saxagliptin



Alogliptin



NSAIDs



Flecainide



MEDICATIONS THAT MAY WORSEN HEART FAILURE

Use with caution:



Tricyclic antidepressants



Corticosteroids



Tyrosine kinase inhibitors



Clozapine



Minoxidil



KEY MESSAGE: Early initiation of the four pillars of GDMT, alongside aggressive management of modifiable risk factors and treatment of reversible causes, remains the cornerstone of preventing progression and improving survival in HFrEF.



OPTIMISING PHARMACOTHERAPY IN HFrEF

Goals of Treatment



Initiate and rapidly optimise guideline-directed medical therapy (GDMT).



Arrange early post-discharge assessment and close follow-up.



Commence more than one therapy simultaneously when appropriate.



Optimise volume status to improve symptoms.



Address clinical, psychosocial and financial barriers to treatment adherence.

Practical Approach to GDMT

Treatment should be individualised according to:



Volume status



Comorbidities



Renal function



Electrolyte stability



Heart rate



Blood pressure



CLINICAL PEARL: It is not necessary to achieve target doses of one medication class before commencing another.



KEY TAKE-HOME POINTS



GDMT improves survival, reduces hospitalisations, and enhances quality of life.



Early initiation and rapid up-titration are essential.



Treat the underlying cause and manage modifiable risk factors.



Avoid medications that may worsen heart failure.



Individualise therapy based on patient characteristics and monitor regularly.

Initiating the Four Pillars

- 1 ARNI (preferred), ACE inhibitor or ARB**
- Start with a low dose and up-titrate as tolerated.



- 2 Evidence-based beta blocker**

- Bisoprolol
- Carvedilol
- Metoprolol succinate
- Nebivolol



- 3 Mineralocorticoid receptor antagonist**

- Spironolactone
- Eplerenone



- 4 SGLT2 inhibitor**
- Dapagliflozin or Empagliflozin



Treatment Principles



Initiate therapies
as early as possible.



Rapid up-titration
is recommended
when tolerated.



Follow-up
every 1–2 weeks
during optimisation.



Once stable,
review every 3–6 months.



Key Message: Patients whose LVEF improves to >40% should continue GDMT indefinitely.



LVEF >40%
Continue GDMT
indefinitely

Management of Congestion



Fluid Overload Present

- Commence loop diuretics (e.g. furosemide).
- Review every 1–2 weeks to assess:
 - Symptoms
 - Weight
 - Volume status
 - Renal function
 - Electrolytes



If congestion persists:

- Consider adding a thiazide diuretic (e.g. hydrochlorothiazide) when appropriate.
- Failure of oral diuretic therapy may require hospital referral for intravenous treatment.



Important: GDMT can be commenced before complete euvolaemia has been achieved.



Hydralazine and Isosorbide Dinitrate

May be considered in patients with:

- LVEF $\leq 35\%$, or
- LVEF $< 45\%$ with a dilated left ventricle
- Persistent NYHA Class III–IV symptoms despite GDMT



Emerging Therapies



Digoxin

- Emerging evidence suggests reduced mortality and heart failure hospitalisation in selected patients.
- Use cautiously in:
 - Elderly patients
 - Females
 - Frail individuals
 - Patients with hypokalaemia
 - Patients in sinus rhythm



Omecamtiv Mecarbil

- Selective cardiac myosin activator.
- May reduce cardiovascular death and heart failure hospitalisation.
- Appears most beneficial in patients with severely reduced LVEF.
- Currently unavailable in Australia.



Key Message: Early initiation of the four pillars of GDMT, aggressive optimisation within the first few weeks, and close longitudinal follow-up are fundamental to improving outcomes in HFrEF.



Additional Pharmacological Options



Ivabradine

Consider in patients with:

- LVEF $\leq 35\%$
- Persistent symptoms despite optimal GDMT
- Sinus rhythm
- Resting heart rate > 70 bpm despite maximally tolerated beta-blocker therapy



Dosing

- < 75 years: 5 mg twice daily
- ≥ 75 years: 2.5 mg twice daily
- Review heart rate after 2–4 weeks and up-titrate when appropriate.



Vericiguat

May be considered in patients with:

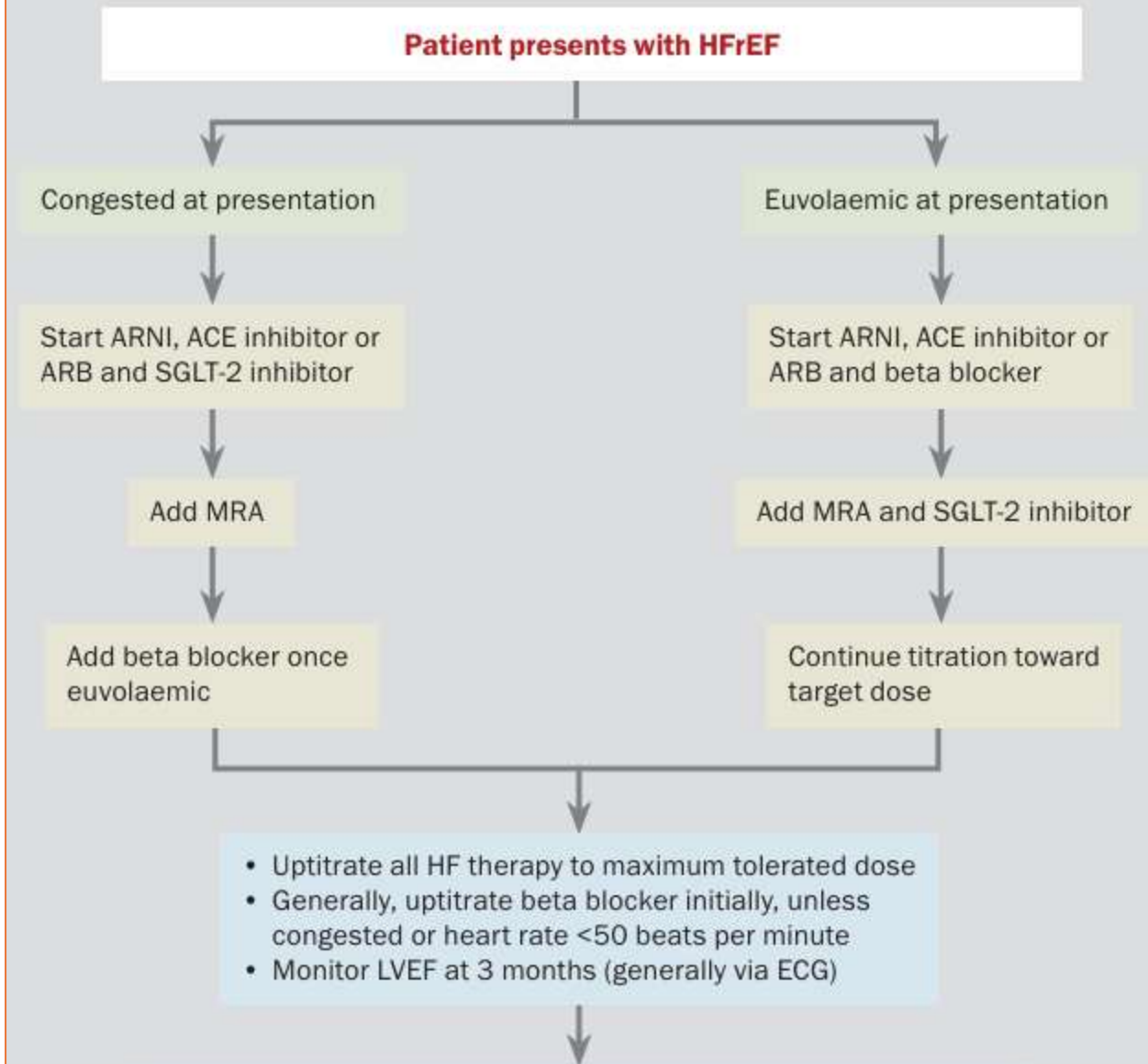
- HFrEF
- Recent heart failure decompensation
- NYHA Class II–IV symptoms despite optimal GDMT



Dosing

- Start: 2.5 mg daily
- Double every two weeks
- Target dose: 10 mg daily

MANAGEMENT ALGORITHM FOR HEART FAILURE WITH REDUCED EJECTION FRACTION



-
- ```
graph TD; A["• Uptitrate all HF therapy to maximum tolerated dose
• Generally, uptitrate beta blocker initially, unless congested or heart rate <50 beats per minute
• Monitor LVEF at 3 months (generally via ECG)"] --> B["• ICD if LVEF ≤35% after 3 months
• CRT if QRS ≥130 ms (left bundle branch block preferred ≥150 ms)"]; B --> C["• Also consider ivabradine if applicable
• If sinus rhythm ≥70 bpm + LVEF ≤35% on maximally tolerated beta blocker"]; C --> D["Additional treatment options for persistent HFrEF
• Consider nitrates + hydralazine if ARNI, ACE inhibitor or ARB is contraindicated or not tolerated
• Consider nitrates ± hydralazine ± digoxin if symptoms are refractory
• Consider vericiguat if recent hospitalisation and high risk of readmission
• Consider intravenous ferric carboxymaltose if ferritin ≤299 ng/mL and transferrin saturation <20%
• For advanced or refractory HF, refer patient to advanced HF centre for mechanical circulatory support or heart transplant"]; style A fill:#e0f0ff; style B fill:#e0f0ff; style C fill:#e0f0ff; style D fill:#d0c0e0;
```
- Uptitrate all HF therapy to maximum tolerated dose
  - Generally, uptitrate beta blocker initially, unless congested or heart rate <50 beats per minute
  - Monitor LVEF at 3 months (generally via ECG)

- ICD if LVEF ≤35% after 3 months
- CRT if QRS ≥130 ms (left bundle branch block preferred ≥150 ms)

- Also consider ivabradine if applicable
- If sinus rhythm ≥70 bpm + LVEF ≤35% on maximally tolerated beta blocker

#### **Additional treatment options for persistent HFrEF**

- Consider nitrates + hydralazine if ARNI, ACE inhibitor or ARB is contraindicated or not tolerated
- Consider nitrates ± hydralazine ± digoxin if symptoms are refractory
- Consider vericiguat if recent hospitalisation and high risk of readmission
- Consider intravenous ferric carboxymaltose if ferritin ≤299 ng/mL and transferrin saturation <20%
- For advanced or refractory HF, refer patient to advanced HF centre for mechanical circulatory support or heart transplant

Abbreviations: ARB = angiotensin receptor blocker; ARNI = angiotensin receptor-neprilysin inhibitor; CRT = cardiac resynchronisation therapy; HF = heart failure; HFrEF = heart failure with reduced ejection fraction; ICD = implantable cardioverter defibrillator; LVEF = left ventricular ejection fraction; MRA = mineralocorticoid receptor antagonist; SGLT-2 = sodium-glucose cotransporter-2.





## Precautions and Clinical Considerations



### ARNI (Sacubitril/Valsartan)

- Do not commence within **36 hours** of stopping an ACE inhibitor.
- Use with caution in patients with:
  -  eGFR <30 mL/min/1.73 m<sup>2</sup>
  -  Systolic blood pressure <100 mmHg
- Monitor:
  -  Blood pressure
  -  Renal function
  -  Serum potassium



### SGLT2 Inhibitors

- Use in type 1 diabetes remains **off-label** because of the risk of diabetic ketoacidosis.
- Counsel patients regarding:
  -  Genital mycotic infections
  -  Euglycaemic diabetic ketoacidosis
  -  Sick-day management
  -  Fasting and perioperative medication withholding recommendations
  -  Maintaining adequate hydration



## Beta Blockers

- Do not initiate during:
  - Acute decompensated heart failure
  - Significant bradyarrhythmias or conduction disease
- Beta blockers may be commenced or recommended once the patient is:
  - Clinically stable
  - Euvolaemic



## Ivabradine

- Avoid in patients with:
  - Acute decompensated heart failure
  - Significant conduction disease
  - Bradyarrhythmias
- Appropriate only for patients in:
  - Sinus rhythm
  - Persistent resting heart rate >70 bpm despite maximally tolerated beta-blocker therapy



**Key Message:** Careful patient selection, close monitoring of renal function, electrolytes, blood pressure, and volume status are essential when initiating and optimising GDMT in patients with HFrEF.



## Monitoring and Managing Complications of GDMT



### Common Adverse Effects



Symptomatic hypotension



Bradycardia



Dizziness



Hypovolaemia



### Managing Symptomatic Hypotension

Hypotension occurs more commonly with ARNIs.



If systolic blood pressure is <100 mmHg, consider:



- Reducing the dose of sacubitril/valsartan



- Switching to an ACE inhibitor or ARB if required



- Reducing loop diuretic doses if the patient is not clinically congested



- Reassessing volume status and other contributing medications



#### Clinical Pearl:

Persistent hypotension, poor medication tolerance, or worsening symptoms should prompt specialist cardiology review.



## Monitoring During GDMT Optimisation



### At Follow-up Visits

Assess:



Symptoms of congestion



Blood pressure



Heart rate



Volume status



Medication tolerance



### Laboratory Monitoring

Monitor:



Serum potassium



Renal function



Electrolytes



### Timing

- Check blood pressure, renal function and electrolytes within 1–2 weeks of:
  - Initiating an ARNI, ACE inhibitor or ARB
  - Any dose escalation
- Ongoing monitoring should be individualised according to renal function and clinical stability.



**Aim:** Optimise GDMT safely to improve symptoms, reduce hospitalisations, and improve survival.

| Drug name                                               | Starting dose | Target dose  |
|---------------------------------------------------------|---------------|--------------|
| <b><i>ACE inhibitors</i></b>                            |               |              |
| Captopril                                               | 6.25 mg tds   | 50 mg tds    |
| Enalapril                                               | 2.5 mg bd     | 10–20 mg bd  |
| Lisinopril                                              | 2.5–5 mg od   | 25–35 mg od  |
| Ramipril                                                | 2.5 mg bd     | 5 mg bd      |
| Trandolapril                                            | 0.5 mg od     | 4 mg od      |
| <b><i>Angiotensin receptor–neprilysin inhibitor</i></b> |               |              |
| Sacubitril/valsartan                                    | 24/26 mg bd   | 97/103 mg bd |
| <b><i>Beta blockers</i></b>                             |               |              |
| Bisoprolol                                              | 1.25 mg od    | 10 mg od     |
| Carvedilol                                              | 3.125 mg bd   | 25–50 mg bd  |
| Metoprolol (controlled-release/extended-release)        | 23.75 mg od   | 190 mg od    |
| Nebivolol                                               | 1.25 mg od    | 10 mg od     |
| <b><i>Mineralocorticoid receptor antagonists</i></b>    |               |              |
| Eplerenone                                              | 25 mg od      | 50 mg od     |
| Spironolactone                                          | 25 mg od      | 50 mg od     |



|                                                         |             |           |
|---------------------------------------------------------|-------------|-----------|
| <b><i>Sodium-glucose cotransporter-2 inhibitors</i></b> |             |           |
| Dapagliflozin                                           | 10 mg od    | 10 mg od  |
| Empagliflozin                                           | 10 mg od    | 10 mg od  |
| <b><i>Angiotensin receptor blockers</i></b>             |             |           |
| Candesartan                                             | 4 mg od     | 32 mg od  |
| Losartan                                                | 50 mg od    | 150 mg od |
| Valsartan                                               | 40 mg bd    | 160 mg bd |
| <b><i>Drugs in other classes</i></b>                    |             |           |
| Hydralazine                                             | 37.5 mg tds | 75 mg tds |
| Isosorbide dinitrate                                    | 20 mg tds   | 40 mg tds |
| Ivabradine                                              | 5 mg bd     | 7.5 mg bd |
| Vericiguat                                              | 2.5 mg od   | 10 mg od  |



## Assessment of Recovery

- Repeat transthoracic echocardiography should usually be performed:
  - **3–6 months** after achieving target or maximally tolerated GDMT doses.



## The Purpose of Repeat Echocardiography

- Assess improvement in LVEF
- Evaluate ventricular remodelling
- Determine eligibility for:
  - >  Implantable cardioverter-defibrillator (ICD)
  - >  Cardiac resynchronisation therapy (CRT)
  - >  Advanced heart failure therapies



**Key Message:** Close follow-up during GDMT optimisation is essential. Early identification of adverse effects, regular monitoring of renal function and electrolytes, and timely reassessment of ventricular function are fundamental to improving outcomes in HFrEF.



## Non-Pharmacological Management

### Iron Optimisation

- Iron deficiency is common in patients with HFrEF and contributes to:
  - Reduced exercise capacity
  - Worsening symptoms
  - Impaired quality of life

### Benefits of Iron Replacement

- Improves:
  - Heart failure symptoms
  - Exercise capacity
  - Quality of life
- May reduce heart failure hospitalisations.
- No proven mortality benefit has been demonstrated to date.



## Screening

Screen all patients with HFrEF every **3–6 months** with:



Full blood count (FBC)



Serum ferritin concentration



Transferrin saturation (TSAT)



## Treatment

- **Intravenous iron replacement** is recommended for symptomatic patients with HFrEF and iron deficiency.



**Key Message:** Regular screening for iron deficiency should form part of routine HFrEF follow-up, as appropriate iron replacement can significantly improve symptoms, functional capacity, and quality of life.



# KEY POINTS



- **Iron** is an **essential micronutrient** for many cellular functions, including both haematopoietic and cellular metabolic processes.



- Iron deficiency is **common** in patients with heart failure and often occurs **independently of anaemia**, and is associated with poor clinical outcomes, including increased mortality.



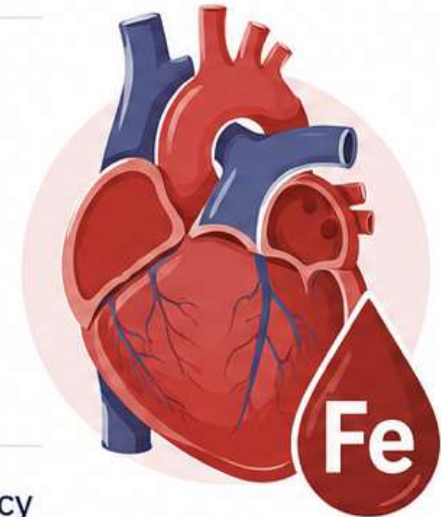
- Patients with heart failure are considered **iron deficient** with:
  - Serum ferritin **<100 mcg/L**, or
  - Serum ferritin **100–300 mcg/L** combined with transferrin saturation **<20%**.



- Intravenous iron replenishment in patients with heart failure and iron deficiency **improves exercise capacity, symptoms and quality of life**. Recent evidence suggests that intravenous iron replenishment likely also **reduces heart failure hospitalisation** and is a **cost-effective** treatment.



- Oral iron supplementation **does not appear to be effective** in patients with heart failure and iron deficiency.





## DIAGNOSTIC CRITERIA FOR HFpEF

All three are required:

- 1 LVEF  $\geq 50\%$
- 2 Elevated natriuretic peptides
  - BNP or NT-proBNP
- 3 Evidence of structural or functional cardiac abnormalities
  - Echocardiographic findings consistent with elevated filling pressures or diastolic dysfunction.



**Important:** Up to 15% of patients with HFpEF may have normal BNP or NT-proBNP levels at rest.



## ECHOCARDIOGRAPHIC FINDINGS SUGGESTIVE OF HFpEF

Consider HFpEF when there is:

-  Preserved LVEF ( $\geq 50\%$ )
-  Left atrial enlargement
-  Elevated E/e' ratio ( $>9$ )
-  Increased left ventricular mass index
-  Evidence of diastolic dysfunction
-  Normal mitral valve function



Left atrial enlargement with preserved ejection fraction should particularly raise suspicion for HFpEF.



## LIMITATIONS OF RESTING INVESTIGATIONS



BNP/NT-proBNP may be normal at rest.



Resting echocardiography has limited sensitivity.



Symptoms frequently occur only during exercise.



**Patients with persistent unexplained dyspnoea may require:**



Exercise stress echocardiography



Diastolic stress testing



Invasive haemodynamic assessment





## INTERPRETATION OF H<sub>2</sub>FPEF SCORE

| Score | Interpretation             |
|-------|----------------------------|
| 0–1   | ✓ HFpEF unlikely           |
| 2–5   | ⚠ Intermediate probability |
| ≥6    | 🎯 HFpEF highly likely      |



### CLINICAL USE

The H<sub>2</sub>FPEF score:

- ✓ Improves diagnostic accuracy.
- ✓ Identifies patients requiring further testing.
- ✓ Helps determine the need for:
  - Exercise stress echocardiography
  - Diastolic stress testing
  - Invasive haemodynamic assessment.



## H<sub>2</sub>FPEF SCORE

| Variable               | Criteria                        | Points |
|------------------------|---------------------------------|--------|
| Heavy                  | BMI >30 kg/m <sup>2</sup>       | 2      |
| Hypertensive           | ≥2 antihypertensive medications | 1      |
| Atrial Fibrillation    | Paroxysmal or persistent AF     | 3      |
| Pulmonary Hypertension | PASP >35 mmHg                   | 1      |
| Elderly                | Age >60 years                   | 1      |
| Filling Pressure       | E/e' >9                         | 1      |



**Maximum score = 9**





## Box – Principles of management in patients with heart failure with preserved ejection fraction



### Avoid tachycardia

For patients with atrial fibrillation, use digoxin or beta blockers



### Blood pressure control

ACE inhibitors, angiotensin receptor antagonists (sartans) or mineralocorticoid receptor antagonists may be of the greatest benefit



### Comorbidities

Optimise cardiac and noncardiac conditions, particularly atrial fibrillation, obesity and diabetes mellitus



### Diuretics

Use loop diuretics to relieve congestion, with close monitoring of renal function



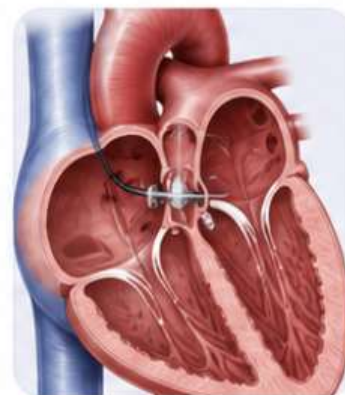
### Exercise training

Improves exercise capacity and quality of life



## Devices

The lack of benefit from drug therapies is likely due to the myriad of pathways activated in HFpEF, with the only definite uniting pathology being elevated left ventricular filling pressures. Consequently, devices targeting this pathway have been tested in trials over the past few years.



### Interatrial septal device

A transcatheter interatrial left to right shunt has been shown to offset the high left atrium pressure that develops in HFpEF.<sup>64–66</sup> One-year observational outcomes have shown the safety of this device, with increased exercise tolerance, quality of life, and a trend toward decreased hospitalisations and heart failure symptoms.<sup>67,68</sup> A trial is under way.<sup>69</sup>



### Implantable pulmonary arterial pressure monitoring

Continuous monitoring of haemodynamics through an implanted device allows for assessment of diastolic left ventricular pressures, and early appropriate administration of diuretics. The CHAMPION trial demonstrated reduced hospitalisations with this device by alerting physicians to high pulmonary pressures and directing subsequent changes to medicines.<sup>70,71</sup> This device is available for clinical use, however it is currently limited by availability and cost.



# SGLT2 Inhibitors in HFpEF



- **Foundational therapy for HFpEF**, regardless of diabetes status.



- **Reduce heart failure hospitalisations** and improve quality of life.



- **Standard dose:** Empagliflozin 10 mg daily or Dapagliflozin 10 mg daily (no dose titration required).



- **Can be safely initiated:**
  - In stable outpatients.
  - During hospitalisation for acute decompensated heart failure once clinically stable.
  - Before discharge to improve long-term adherence.



## **Beneficial across a broad range of patients, including those with:**

- Diabetes mellitus
- Chronic kidney disease
- Obesity
- Atrial fibrillation
- Previous heart failure hospitalisation.

## **Monitor:**

- Blood pressure
- Renal function (eGFR and creatinine)
- Volume status and symptoms.



**Clinical Pearl:** Early initiation of SGLT2 inhibitors has rapid clinical benefits and should be considered a cornerstone of HFpEF management alongside optimisation of comorbidities and congestion control.





## Long-Term Management and Prognosis



### Long-Term Care Goals

- ✓ Optimise and maintain GDMT.
- ✓ Improve quality of life and functional capacity.
- ✓ Reduce hospitalisations and mortality.
- ✓ Promote self-management and treatment adherence.
- ✓ Integrate psychosocial and palliative care support when appropriate.



### Follow-Up and Models of Care

- Once clinically stable and receiving optimal GDMT, review patients every **3–6 months**.
- Consider:
  -  Cardiac rehabilitation programs
  -  Heart failure disease management services
  -  Telehealth and virtual care services, particularly in rural and remote settings.



## Heart Failure Disease Management Programs

### Multidisciplinary programs involving:

- Heart failure nurses
- Allied health professionals
- General practitioners
- Cardiologists

### have been shown to:

-  Reduce hospitalisations
-  Reduce mortality
-  Improve patient outcomes



### Supporting Self-Management

#### Patients should be encouraged to:

- Monitor symptoms and body weight
- Maintain medication adherence
- Engage in regular physical activity when appropriate
- Adopt healthy dietary practices
- Recognise symptoms of deterioration early



### Technology May Assist With

- Activity monitoring
- Weight management
- Medication reminders
- Communication with the heart failure team
- Tracking dietary and lifestyle behaviours.



## Addressing Barriers to Care

### Potential barriers include:

-  Limited access to medications and healthcare services
-  Mental health disorders
-  Cognitive impairment
-  Polypharmacy
-  Homelessness
-  Social disadvantage and geographical remoteness



### Practical Primary Care Strategies

- Simplify medication regimens when possible.
- Develop individualised care plans and treatment goals.
- Provide culturally appropriate patient education.
- Address mental health concerns.
- Arrange pharmacist medication reviews when appropriate.
- Consider home-based nursing support.



## Palliative Care and Goals of Care

Patients who are unsuitable for advanced heart failure therapies should have:

- ✓ Early goals-of-care discussions
- ✓ Advance care planning
- ✓ Appropriate palliative care referral when indicated

### Palliative care may:

-  Improve symptom control
-  Enhance quality of life
-  Provide psychological and spiritual support
-  Reduce distressing symptoms in advanced disease



**Clinical Pearl:** Guideline-directed medical therapy should generally be continued in advanced heart failure unless it contributes to patient discomfort or is no longer consistent with the individual's goals of care.



**Key Message:** Long-term management of HFrEF extends beyond pharmacotherapy. Regular follow-up, multidisciplinary care, patient empowerment, and timely integration of psychosocial and palliative care are essential to improving outcomes and quality of life.



# 1. MULTIDISCIPLINARY HEART FAILURE CARE

Heart failure management is best delivered through a **multidisciplinary team**, which may include:



Heart failure nurses



Cardiologists or specialist physicians



General practitioners



Pharmacists



Physiotherapists



Occupational therapists



Exercise physiologists



Dietitians



Psychologists



Palliative care physicians



## BENEFITS:



Improved survival



Reduced hospital readmissions



Greatest benefit in **high-risk patients**, particularly following recent hospitalisation



If face-to-face services are unavailable, **telemonitoring** or **telephone support programs** are effective alternatives.

## 2. NURSE-LED MEDICATION TITRATION

Nurse-led medication titration helps:



Increase achievement of **target doses** of guideline-directed therapy



Reduce heart failure **rehospitalisation**



Improve **survival**



## 3. EXERCISE TRAINING

Regular **low- to moderate-intensity continuous exercise** is recommended, particularly in patients with **HFrEF**, to:



Improve **quality of life**



Reduce heart failure **hospitalisations**



These multidisciplinary and supportive care strategies improve outcomes and help people with heart failure **live longer and better**.



## Device Therapy in HFrEF

### Implantable Cardioverter-Defibrillator (ICD)



#### Primary Prevention

Consider ICD therapy in patients with:

- Symptomatic ischaemic cardiomyopathy (NYHA Class II–III)
- LVEF  $\leq 35\%$
- Persistent LV dysfunction despite  $\geq 3$  months of optimal GDMT
- Expected survival  $> 12$  months



#### Secondary Prevention

ICD therapy is indicated in patients with:

- Previous ventricular arrhythmias associated with haemodynamic instability
- Survivors of sudden cardiac arrest when appropriate



**Clinical Pearl:** Evidence for ICD therapy is strongest in ischaemic cardiomyopathy, although selected patients with non-ischaemic cardiomyopathy may also benefit.



## Cardiac Resynchronisation Therapy (CRT)

CRT improves symptoms and reduces morbidity in selected patients with HFrEF and electrical dyssynchrony.



#### Consider CRT in Patients With:

- Symptomatic HFrEF
- LVEF  $\leq 35\%$
- Sinus rhythm
- Left bundle branch block (LBBB)
- QRS duration 130–149 ms
- Persistent symptoms despite optimal GDMT



#### Strongest Benefit

Patients with:

- LVEF  $\leq 35\%$
- Sinus rhythm
- QRS duration  $\geq 150$  ms
- Left bundle branch block morphology



#### CRT Upgrades

Consider upgrading to CRT in patients who:

- Have a permanent pacemaker or ICD
- Develop worsening heart failure symptoms despite GDMT
- Have a significant burden of right ventricular pacing



#### Patients Requiring Ventricular Pacing

CRT is preferred over right ventricular pacing in patients with:

- HFrEF
- High-degree atrioventricular block requiring ventricular pacing



**Clinical Pearl:** Right ventricular pacing may worsen cardiac dyssynchrony and heart failure symptoms. CRT should be considered when long-term ventricular pacing is anticipated.



**Key Message:** Device therapy should be considered after optimisation of GDMT. Repeat echocardiography at 3–6 months is essential to identify patients who may benefit from ICD or CRT and improve long-term outcomes in HFrEF.





## Management of Valvular Heart Disease

- ✓ Significant valvular heart disease is associated with poor outcomes in patients with HFrEF.
- ✓ Early identification and specialist referral are essential.



### Aortic Stenosis

#### Consider:

- Surgical aortic valve replacement (SAVR), or
- Transcatheter aortic valve implantation (TAVI)

in patients with:

- HFrEF
- Severe high-gradient aortic stenosis



#### Benefits include:

- Improved symptoms
- Reduced mortality



### Mitral Regurgitation

#### Primary (Degenerative) Mitral Regurgitation

- Surgical repair or replacement is first-line treatment for severe primary MR resulting in HFrEF.

#### Functional Mitral Regurgitation

- Consider transcatheter edge-to-edge repair (TEER) in selected patients who:
  - Remain symptomatic despite optimal GDMT
  - Are unsuitable for surgery
  - Do not require coronary revascularisation



**Clinical Pearl:** Decisions regarding valve intervention should be made collaboratively with a multidisciplinary structural heart team.



## Referral for Advanced Heart Failure Therapies

Consider referral in patients with:

- Advanced heart failure refractory to optimal GDMT
- Persistent symptoms despite device therapy when indicated
- Progressive clinical deterioration



### Advanced Therapies Include

- Left ventricular assist devices (LVADs)
- Heart transplantation
- Mechanical circulatory support when appropriate



### Candidates for Mechanical Support Should Have

- Good treatment adherence
- Appropriate capacity for device management
- Adequate psychological and social support



### Heart Transplantation

Heart transplantation should be considered in patients with:

- Advanced heart failure
- Refractory symptoms despite optimal medical and device therapy
- No absolute contraindications



**Key Message:** Early referral for specialist assessment is essential in patients with severe valvular disease or advanced heart failure. Primary care remains central to coordinating care, supporting treatment adherence, and providing ongoing psychosocial support throughout the patient's heart failure journey.



## 2. SUGGESTED QUALITY OF CARE MEASURES FOR PATIENTS WITH HEART FAILURE



### Newly diagnosed HF

- What proportion have had an ECG?
- What proportion have had an echocardiogram?



### All patients with HF

- What proportion have had an ECG within 12 months?
- What proportion have had an echocardiogram within two years?
- What proportion have an advanced healthcare directive?
- What proportion have been screened for depression?
- What proportion have had a care plan and care plan review?
- What proportion have had a home medication review?



### HF with a reduced LVEF (HFrEF)

- What proportion receive a prescription for an ACE inhibitor, ARB or ARNI?
- What proportion receive a prescription for a beta blocker?
- What proportion receive a prescription for an MRA?
- What proportion have achieved the target or maximum tolerated dose of an ACE inhibitor, ARB or ARNI by 6 months following commencement?
- What proportion have achieved the target or maximum tolerated dose of a beta blocker by 6 months following commencement?
- What proportion with an LVEF of 35% or less despite medical therapy have been referred for consideration of cardiac resynchronisation or implantable cardioverter defibrillator therapy?



### Atrial fibrillation

- What proportion receive a prescription for an anticoagulant?



### Following HF hospitalisation

- What proportion have been reviewed within 2 weeks?
- What proportion have a written discharge summary and HF action plan?
- What proportion have been referred to a multidisciplinary HF disease management or multidisciplinary telemonitoring/telephone support program?
- What proportion have been referred to an exercise training program?
- What is the 30-day and 6-month mortality rate?
- What is the 30-day and 6-month rehospitalisation rate?



**Abbreviations:** ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; ARNI = angiotensin receptor neprilysin inhibitor; ECG = electrocardiogram; HF = heart failure; LVEF = left ventricular ejection fraction; MRA = mineralocorticoid receptor antagonist.

# References

---

1. Bozkurt B, et al. *Second Universal Definition of Heart Failure*. Circulation. 2026.
2. Chiswell K, Hayward CS. *Heart Failure with Reduced Ejection Fraction: Diagnosis and Management in Primary Care*. Medicine Today. 2026;27(4);20–31.
3. RACGP NewsGP. *Expanded PBS Eligibility for Heart Failure and Migraine Medications*. 2026.
4. *Management of Heart Failure with Preserved Ejection Fraction*. Australian Prescriber. 2025.
5. Atherton JJ, et al. *Heart Failure in Adults: Diagnosis and Management*. Medicine Today. 2019;20(6):15–27. Heart Foundation Australia.





# Thank You Questions



Mohammed Hamed



2026

