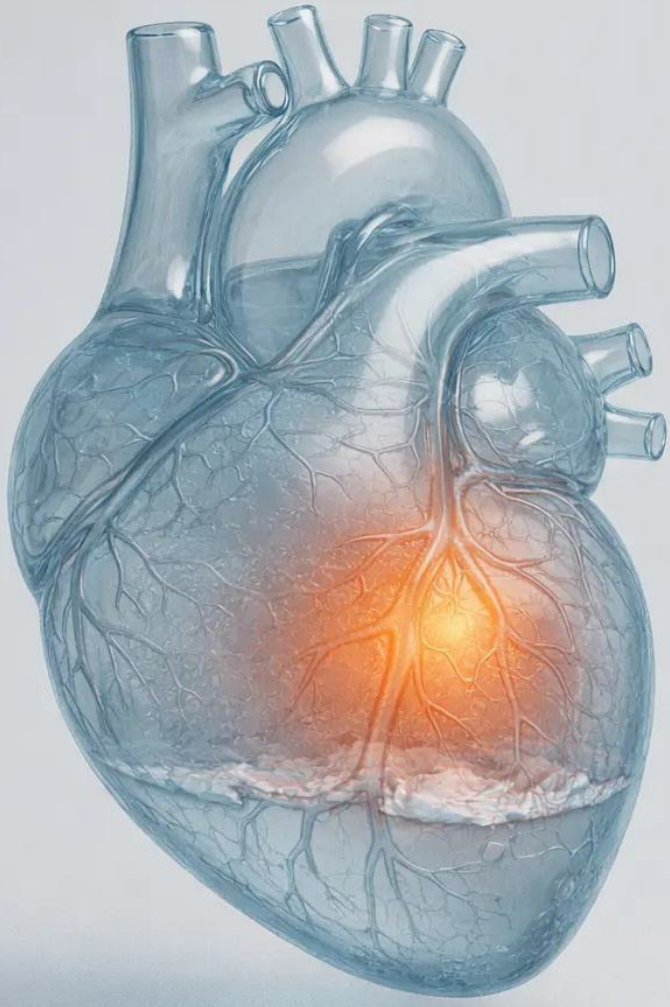
HEART FAILURE

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

ADB

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Japan Fund for Prosperous and Resilient Asia and the Pacific

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World Health Organization
Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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HEART FAILURE

QUICK REFERENCE GUIDELINE

Heart failure (HF) is a major cause of morbidity and hospital admissions, and annual mortality ranges from 5–10% in mild cases to over 50% in advanced stages. Outcomes improve significantly with early diagnosis and guideline-directed therapy, reducing hospitalizations and enhancing survival and quality of life.

Heart failure defined as a clinical syndrome of symptoms/signs due to cardiac structural or functional abnormality causing reduced cardiac output and/or elevated intracardiac pressures.

- By ejection fraction (EF):
  - HFrEF - HF with reduced left ventricular ejection fraction (LVEF) <40%.
  - HFmrEF - HF with mildly reduced LVEF 40-49%.
  - HFpEF - HF with preserved LVEF ≥50% with evidence of structural heart disease and/or diastolic dysfunction.
- By course: acute decompensated HF vs chronic/stable HF.

Causes, risk factors & triggers

- Causes: Coronary artery disease/acute coronary syndrome (ACS), long-standing hypertension, valvular disease, cardiomyopathies (dilated/hypertrophic/restrictive), myocarditis, congenital heart disease, toxins (alcohol, chemo).
- Risk factors: Diabetes, dyslipidemia, ... obesity, chronic kidney disease (CKD), smoking, high-salt diet, sedentary lifestyle, family history.
- Common triggers of decompensation: Arrhythmias (atrial fibrillation [AF] with rapid rate), infection, uncontrolled hypertension, ACS, excess sodium/fluid, missed medicines, non-steroidal anti-inflammatory drugs (NSAIDs).

Evaluation for diagnosis

- Clinical features (history): Exertional dyspnea, fatigue, orthopnea, paroxysmal nocturnal dyspnea, ankle swelling; place in context of risks/known heart disease.
- Physical exam: Tachycardia, elevated jugular venous pressure (JVP), basal crackles, third heart sound (S3), hepatomegaly, peripheral pitting edema.
- Laboratory/initial tests:
  - Electrocardiogram (ECG), chest X-ray (CXR), complete blood count (CBC), renal function/electrolytes.
  - B-type natriuretic peptide (BNP) or N-terminal pro-BNP (NT-proBNP) if available (low levels make HF unlikely).
- Confirmation of diagnosis: Echocardiography (gold standard): LVEF, wall motion, chamber size, structural valve disease; consider cardiac magnetic resonance (CMR) if etiology unclear.

Classification / severity assessment

- New York Heart Association (NYHA) functional class: I (no limitation) → IV (symptoms at rest).
- Stages (A-D): At risk → structural disease → symptomatic → refractory advanced HF.
- Volume status: “Warm/wet/dry/cold” clinical profiles.
- Risk markers: Recurrent admissions, hypotension, rising creatinine, hyponatremia, elevated natriuretic peptides.

Differential diagnosis (key separates)

- Chronic obstructive pulmonary disease (COPD): Obstructive spirometry; normal echo.
- Anemia: Low hemoglobin causing exertional dyspnea/fatigue.
- Nephrotic syndrome: Edema with heavy proteinuria and hypoalbuminemia.
- Constrictive pericarditis: Right HF signs; pericardial thickening on imaging.
- Also consider pulmonary embolism, pneumonia, hypothyroidism, cirrhosis, primary venous/lymphatic disease, obesity/deconditioning.

Management goals & principles

- Goals: Reduce mortality and ... hospitalizations; relieve symptoms; improve quality of life and functional capacity; slow disease progression; prevent decompensation; identify candidates for advanced therapies.
- Principles: Confirm objectively, treat cause, use guideline-directed medical therapy (GDMT), pair drugs with lifestyle measures, titrate to targets, monitor safety, educate patients/ caregivers, and adapt to resource setting.

Approach to management

- Acute/decompensated HF (first hours-days):
  - Airway, breathing, circulation; oxygen if hypoxemic; non-invasive positive pressure ventilation (NIPPV) if needed.
  - Intravenous loop diuretic; vasodilator (e.g., IV nitroglycerin) if hypertensive; treat triggers (AF rate/rhythm control, infection, ACS).
  - Early risk stratification and decongestion; start or optimize GDMT before discharge if stable.
- Chronic/stable HF:
  - Build the four-pillar base for HFrEF: angiotensin receptor-neprilysin inhibitor (ARNI) or angiotensin-converting enzyme inhibitor (ACEi)/ angiotensin receptor blocker (ARB) + evidence-based beta-blocker + mineralocorticoid receptor antagonist (MRA) + sodium-glucose cotransporter-2 inhibitor (SGLT2i).
  - Control blood pressure, heart rate, ...

and congestion; manage comorbidities; vaccinate; enroll in cardiac rehabilitation where feasible.

- Control blood pressure, heart rate, and congestion; manage comorbidities; vaccinate; enroll in cardiac rehabilitation where feasible.

(For management of pediatric heart failure see below)

Non-pharmacological interventions

- Sodium restriction (<2 g/day), fluid guidance (often 1.5-2 L/day if hyponatremic or congested).
- Daily weights; symptom diary; leg elevation; activity as tolerated.
- Smoking and alcohol cessation.
- Vaccinations: influenza, pneumococcal per policy.

Prioritize generics (ACEi/ARB, beta-blocker, MRA, loop diuretic); standardized paper titration charts; community health-worker follow-ups; tele/phone check-ins; point-of-care labs; echo when available; use clinical assessment when BNP/NT-proBNP not accessible.

PHARMACOLOGICAL THERAPY (HFREF FOCUS; INDIVIDUALIZE FOR HFPEF/HFMREF)

Class / drug	Indication	Start → Target dose	Route	Key cautions
ARNI sacubitril/ valsartan	HFrEF symptomatic; replace ACEi/ARB if possible	24/26-49/51 mg BID → 97/103 mg BID	PO	Avoid with ACEi within 36 h; hypotension, hyperkalemia, renal dysfunction
ACEi enalapril / lisinopril & tolerability	HFrEF if ARNI not used	Enalapril 2.5 mg BID → 10-20 mg BID; Lisinopril 2.5-5 mg OD → 20-40 mg OD	PO	Cough/angioedema (ACEi), hyperkalemia, renal dysfunction
ARB valsartan	ACEi intolerance	40 mg BID → 160 mg BID	PO	Hyperkalemia, renal dysfunction, hypotension
Beta-blocker carvedilol / metoprolol succinate / bisoprolol	Stable HFrEF	Carvedilol 3.125 mg BID → 25-50 mg BID; Metoprolol-XL 12.5-25 mg OD → 200 mg OD; Bisoprolol 1.25 mg OD → 10 mg OD	PO	Start when euvolemic; watch bradycardia, hypotension
MRA spironolactone / eplerenone	HFrEF NYHA II-IV	Spironolactone 12.5-25 mg OD → 25-50 mg OD; Eplerenone 25 mg OD → 50 mg OD	PO	Hyperkalemia; avoid if K^+>5.0 or eGFR <30
SGLT2i dapagliflozin / empagliflozin	HFrEF (± diabetes)	Dapagliflozin 10 mg OD; Empagliflozin 10 mg OD	PO	Genital infections; adjust with severe CKD per label
Loop diuretic furosemide / torsemide	Congestion relief	Furosemide 20-40 mg OD-BID (titrate); Torsemide 10-20 mg OD	PO/IV	Monitor weight, K^+, creatinine; over-diuresis → hypotension/AKI
Hydralazine + isosorbide dinitrate	ACEi/ARB/ARNI intolerance or add-on	Hydralazine 25-75 mg TID + ISDN 20-40 mg TID	PO	Headache, hypotension
Ivabradine	Sinus rhythm, LVEF ≤35%, HR ≥70 bpm despite beta-blocker	5 mg BID (titrate by HR)	PO	Bradycardia; needs sinus rhythm
Digoxin	Symptom relief, AF rate control	0.125-0.25 mg OD (aim level 0.5-0.9 ng/mL)	PO	Toxicity ↑ with renal dysfunction, drug interactions
Iron (IV ferric carboxymaltose)	Symptomatic HF with iron deficiency	Dose by deficit	IV	Hypersensitivity; monitor ferritin/TSAT
Vericiguat	Recent decompensation HFrEF despite GDMT	2.5 mg OD → 10 mg OD	PO	Hypotension; avoid in pregnancy

Tailor HFpEF/HFmrEF to control BP, diuretics for congestion, SGLT2i benefit across EF ranges; treat comorbidities (AF, obesity, OSA, CKD).

Assessment of response, review, follow-up & treatment adjustment

- Monitor: symptoms (dyspnea, orthopnea, exercise tolerance), daily weight, blood pressure/heart rate, renal function/electrolytes after dose changes and periodically.
- Follow-up frequency: every 2-4 weeks during initiation/titration; once stable every 3-6 months.
- Before changing therapy, assess: NYHA class, adherence, salt/fluid intake, diuretic response, BP/HR, labs, triggers (infection, arrhythmia), and comorbidities.

When to step up (intensify)

- Persistent/worsening symptoms or rising NYHA class; rapid weight gain >1-2 kg in 2-3 days; edema/crackles/ JVP up; uncontrolled BP/HR; recurrent decompensation.
- Actions: up-titrate GDMT, intensify diuresis (consider short-course thiazide add-on), add MRA/SGLT2i/ ARNI, optimize AF control, evaluate for cardiac resynchronization therapy (CRT) or implantable cardioverter-defibrillator (ICD) when criteria met; consider referral.

When to step down (de-escalate/ hold)

- Symptomatic hypotension, bradycardia/ conduction block, acute kidney injury or hyperkalemia, over-diuresis, intercurrent sepsis/procedures.
- Actions: temporarily reduce/hold offending drug(s), correct volume/ electrolytes, restart cautiously once stable.

Referral (tiered approach)

- Primary → secondary care: diagnostic uncertainty; first HF confirmation with echocardiography; poor response to initial therapy; significant CKD/ electrolyte issues.
- Secondary → tertiary/advanced HF center: NYHA III-IV despite optimized GDMT, recurrent hospitalizations, LVEF ≤35% with device therapy consideration (ICD/CRT), suspected advanced cardiomyopathy or amyloidosis, refractory congestion, cardiogenic shock, evaluation for ventricular assist device/transplant.

Complications (recognize and prevent)

- Arrhythmias (AF, ventricular tachycardia/fibrillation): anticoagulation AF per *CHA_2DS_2-VASc, correct electrolytes, consider ICD for indicated HFrEF.
- Cardiorenal syndrome: balance diuresis/RAAS blockade; avoid nephrotoxins.
- Thromboembolism: anticoagulation when indicated (AF/LV thrombus).
- Sudden cardiac death: GDMT, ICD in eligible patients; treat ischemia.
- Other: hyponatremia, anemia/iron deficiency, depression, cachexia.

***CHA_2DS_2-VASc Score**: A risk stratification tool to assess stroke risk in non-valvular AF:

C: Congestive heart failure (1); **H**: Hypertension (1); **A_2**: Age ≥75 (2); **D**: Diabetes mellitus (1); **S_2**: Prior Stroke/TIA/thromboembolism (2); **V**: Vascular disease (1); **A**: Age 65–74 (1); **Sc**: Sex category (female = 1).

Anticoagulation: Indicated if score ≥2 (men) or ≥3 (women); options include DOACs (e.g., apixaban, rivaroxaban) or warfarin (if valvular AF or mechanical valves).

Patient education

- Medicines: take exactly as prescribed; never stop without advice.
- Salt/fluids: <2 g/day salt; fluids 1.5-2 L/day if advised.
- Daily weight: same scale/time; report >2 kg in 3 days.
- Activity: regular, within tolerance; elevate legs when sitting.
- Avoid: NSAIDs, excess alcohol, smoking/tobacco; high-salt processed foods.
- Vaccines: influenza and pneumococcal if recommended.
- Red flags (seek urgent care): worsening breathlessness, chest pain, fainting, rapid swelling, reduced urine, fever/infection.

INTRODUCTION

Heart failure (HF) is a clinical syndrome in which the heart is unable to pump enough blood to meet the body's metabolic demands or can do so only at elevated filling pressures. It occurs due to structural or functional impairment of ventricular filling or ejection of blood. Globally, HF affects over 64 million people and is a leading cause of morbidity and hospital admissions, with a prevalence of 1-2% that rises to over 10% in people aged 70 years and above. In Southeast Asia, prevalence is estimated at 4-6% among adults, with higher rates driven by untreated hypertension, ischemic heart disease, and rheumatic heart disease.

HF is often underdiagnosed in early stages and frequently presents late due to delayed health-seeking behaviour and limited diagnostic facilities in peripheral centres. Mortality ranges from 5-10% annually in mild HF to over 50% in advanced stages. While HF is not curable in most cases, it is controllable, and early diagnosis with proper management can significantly reduce hospitalizations and improve survival. Recognizing HF early and managing it in a standardized manner is crucial, as under-treatment delays recovery and increases mortality, whereas over-treatment, such as excessive diuresis, can lead to renal failure, hypotension, and electrolyte imbalances. Standardised treatment ensures uniform, evidence-based care, prevents harmful practices, and maximizes the use of available resources.

SCOPE OF THE GUIDELINES

These guidelines apply to the evaluation and management of adults aged 18 years and above with suspected or confirmed heart failure (HF), encompassing both newly diagnosed cases and patients with established disease requiring ongoing care. The focus is on medical management, stabilization, and optimization of therapy, including guidance for both acute and chronic presentations.

The document does not provide detailed operative or procedural instructions for surgical or catheter-based interventions (e.g., valve replacement, coronary artery bypass grafting, percutaneous device implantation). However, it outlines when such procedures should be considered and the referral process to appropriate centres.

These recommendations are applicable across all levels of healthcare from primary, secondary, to tertiary while taking into account the constraints of low-resource environments, such as limited access to echocardiography, advanced imaging, or subspecialty cardiology services. Adaptations are provided to ensure effective diagnosis and treatment even in facilities with basic infrastructure, with an emphasis on early recognition, stabilization, and appropriate referral pathways.

The intended users include general practitioners, internists, emergency physicians, nurses, and allied health professionals involved in the care of patients with HF. The guidelines are also relevant for health administrators and policymakers seeking to develop or strengthen HF care pathways. By providing a standardized and simplified approach, these guidelines aim to bridge gaps in care, minimize delays in diagnosis, reduce inappropriate variations in management, and improve patient outcomes until specialized or tertiary care becomes available.

DEFINITIONS

Heart Failure: Complex clinical syndrome resulting from structural or functional impairment of ventricular filling(diastole) or pumping out blood(systole).
Types:

1. **HFrEF** (Heart Failure with Reduced Ejection Fraction): EF <40%.
2. **HFmrEF** (Mildly Reduced Ejection Fraction (EF): EF 41-49%.
3. **HFpEF** (Preserved EF): EF ≥50% with symptoms and signs of HF plus evidence of diastolic dysfunction.

Stages of heart failure

AHA stages of heart failure		Mortality at 1 year
A	AT Risk of HF, No structural disease, No symptoms (Eg: Diabetic, Hypertensive)	2-3 %
B	Structural heart disease, but no signs or symptoms of HF	5-10 %
C	Structural heart disease with prior or current symptoms of HF	15-30 %
D	Refractory HF (requiring specialized intervention)	2-3 %

Type of HF	LVEF Criteria	Diagnostic Requirements	Key Pathophysiology	Management Implications
HFrEF (Heart Failure with Reduced EF)	<40%	Symptoms and/or signs of HF plus LVEF <40%	Impaired systolic function due to reduced myocardial contractility	Strong evidence for mortality reduction with ACEi/ARB, ARNI, β-blockers, MRAs, SGLT2 inhibitors; device therapy in select cases
HFmrEF (Mildly Reduced EF)	41-49%	Symptoms/signs of HF plus LVEF 41-49%	Mixed features of systolic and diastolic dysfunction	Treat as HFrEF; benefits likely but less robust evidence; optimize comorbidity management
HFpEF (Preserved EF)	≥50%	Symptoms/signs of HF plus structural heart disease or diastolic dysfunction (e.g., LA enlargement, LV hypertrophy)	Impaired ventricular relaxation, increased stiffness, elevated filling pressures	No proven mortality-reducing drugs; focus on symptom relief, blood pressure control, diuretic use for congestion, comorbidity management

CAUSES, RISK FACTORS & TRIGGERS

Section	Sub-category	Examples / Details
Causes	Coronary artery disease (CAD)	Chronic ischemia or prior MI → systolic dysfunction
	Hypertension	Long-standing pressure overload → LV hypertrophy → failure
	Valvular heart disease	Stenosis or regurgitation increases workload
	Cardiomyopathies	Dilated, hypertrophic, restrictive; idiopathic or secondary
Risk factors	Myocarditis	Viral, autoimmune, or toxin-induced myocardial injury
	Metabolic	Diabetes, obesity, dyslipidemia
	Renal	Chronic kidney disease → volume overload, neurohormonal activation
	Lifestyle	Smoking, excess alcohol, sedentary habits, high-salt diet
	other	Family history of cardiomyopathy or premature CVD
	Arrhythmias	AF with RVR, ventricular tachyarrhythmias
	Infections	Pneumonia, sepsis → higher metabolic demand and cardiac stress
Triggers for acute decompensation	Uncontrolled hypertension	Sudden afterload rise worsens function
	Acute coronary syndromes	New ischemia or injury reduces contractility
	Medication issues	Missed HF meds; NSAID use
	Dietary indiscretion	Excess sodium or fluid → congestion

EVALUATION FOR DIAGNOSIS

- Clinical History: Focus on hallmark symptoms: exertional dyspnea, fatigue, orthopnea, paroxysmal nocturnal dyspnea, and peripheral edema. Always interpret symptoms in the context of existing cardiovascular risk factors (hypertension, diabetes, CAD) and history of prior cardiac disease.
- Physical Examination: Common findings: tachycardia, elevated jugular venous pressure (JVP), basal lung crackles (rales). Additional signs: presence of a third heart sound (S3 gallop), hepatomegaly, and peripheral pitting edema.
- Basic tests: 12-lead ECG, chest X-ray, complete blood count, renal function, and serum electrolytes.
- Biomarkers: B-type natriuretic peptide (BNP) or NT-proBNP, where available, to distinguish cardiac from non-cardiac dyspnea.

- Definitive Test: Echocardiography is the key diagnostic tool to assess left ventricular ejection fraction (LVEF), wall motion abnormalities, chamber size, and detect structural problems such as valvular disease.

CONFIRMATION OF DIAGNOSIS

The diagnosis of heart failure is confirmed when characteristic clinical features such as dyspnea, fatigue, orthopnea, and weight gain (fluid retention) are supported by objective evidence of cardiac dysfunction. Combining symptom assessment with objective measures ensures diagnostic accuracy, helps classify HF type, and guides appropriate management.

Clinical	Investigations	Confirmation
Exertional dyspnea, fatigue, orthopnea, PND, leg edema	12-lead ECG (ischemia/ arrhythmia, LVH)	Echo: objective cardiac dysfunction (LVEF ↓ for HFrEF; structural/diastolic dysfunction for HFpEF/ HFmrEF)
Signs: tachycardia, elevated JVP, basal crackles, S3, hepatomegaly, pitting edema	Chest X-ray (cardiomegaly, pulmonary congestion)	BNP/NT-proBNP: elevated supports HF; low makes HF unlikely
Risk context: HTN, CAD/MI, diabetes, CKD, valvular disease	CBC, renal function, electrolytes (to assess contributors/impact)	Clinical features + objective echo/biomarker evidence aligned; alternate causes excluded
	Optional: troponin (if ACS), thyroid tests (if indicated)	Consider CMR for etiology (fibrosis/infiltrative), angiography if ischemic cause suspected

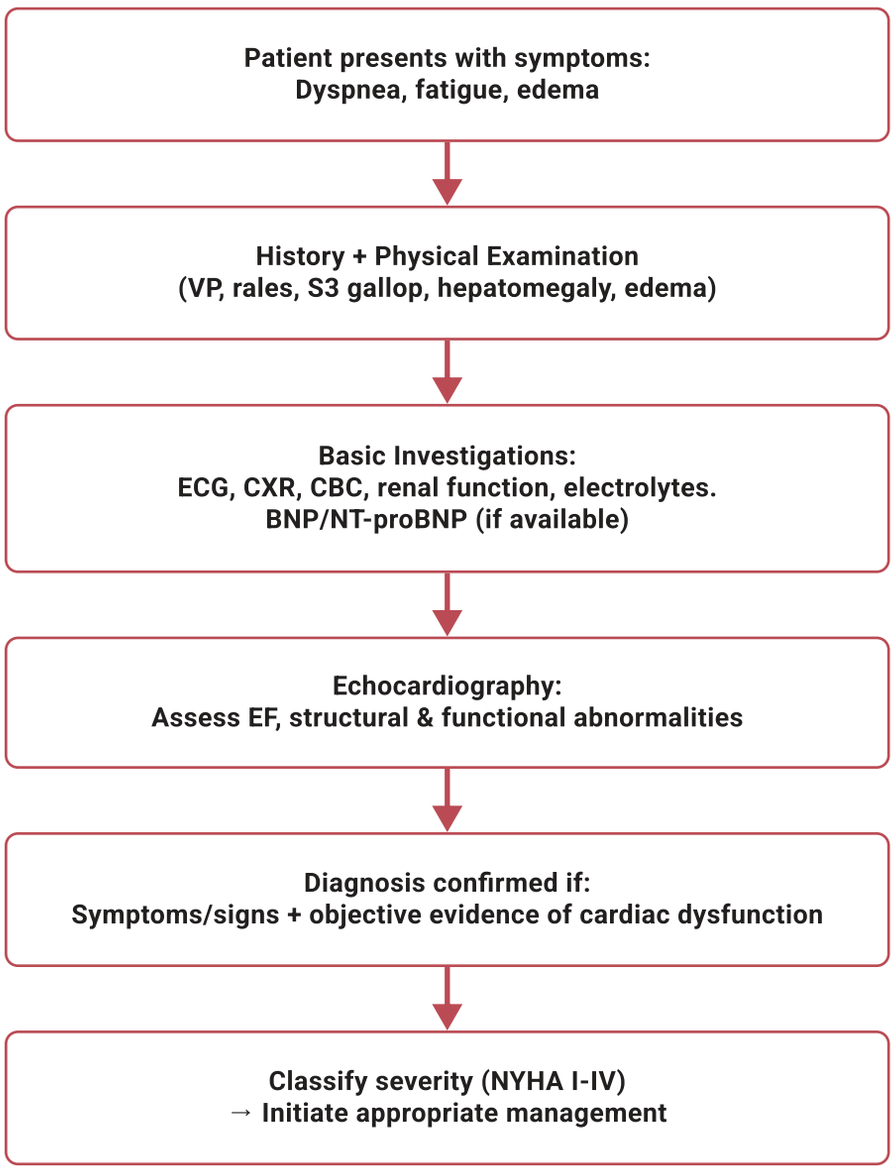
CLASSIFICATION / SEVERITY ASSESSMENT

Heart failure severity is most commonly graded using the New York Heart Association (NYHA) Functional Classification, which correlates symptoms with physical activity tolerance: Patient must have underlying heart disease.

NYHA Class	Functional limitation	Symptoms triggered by	Remarks
I	None	Ordinary activity: no undue angina, dyspnea, fatigue, or palpitations	Requires underlying heart disease
II	Slight	Ordinary activity causes angina, dyspnea, fatigue, or palpitations	Comfortable at rest
III	Marked	Less than ordinary activity causes symptoms	Comfortable at rest
IV	Severe	Any activity causes discomfort; symptoms at rest	Unable to perform physical activity without symptoms

Diagnostic Flowchart

Diagnostic Flowchart - Heart Failure



DIFFERENTIAL DIAGNOSIS

Several conditions can mimic the symptoms and signs of heart failure and should be excluded through careful history, examination, and targeted investigations:

Condition	Usual features	Primary test(s)	Distinguishes from HF
COPD	Chronic dyspnea, exercise intolerance, cough with sputum (often smoker)	Spirometry: obstructive pattern (↓FEV_1/FVC); Echo: no cardiac dysfunction	Lung obstruction without ventricular dysfunction
Anemia	Fatigue, pallor, exertional dyspnea	CBC: low hemoglobin/hematocrit	Dyspnea from low oxygen-carrying capacity, not pump failure
Nephrotic syndrome	Generalized edema, foamy urine	Urine protein (heavy proteinuria), serum albumin (low)	Edema from hypoalbuminemia, not fluid overload from HF
Constrictive pericarditis	Right-sided HF signs, Kussmaul sign, ascites	Echo; CT/MRI: pericardial thickening/calcification; hemodynamics if needed	Pericardial constraint with preserved myocardium; equalized diastolic pressures, pericardial knock

MANAGEMENT GOALS

The primary aim of heart failure management is to improve patient survival, relieve symptoms, prevent hospitalisations, and enhance quality of life.

Specific goals include:

- Reduce mortality and HF hospitalizations.
- Relieve symptoms (dyspnea, fatigue, edema) and improve quality of life and functional capacity.
- Stabilize acute/decompensated episodes by restoring perfusion and decongesting.
- Slow disease progression by treating causes (hypertension, ischemia, valvular disease) and controlling risk factors.
- Prevent future decompensation via education, adherence, diet/fluid guidance, and regular follow-up; each episode worsens prognosis.
- Identify candidates for advanced therapies (cardiac resynchronization therapy, implantable cardioverter-defibrillator, transplant) when indicated.

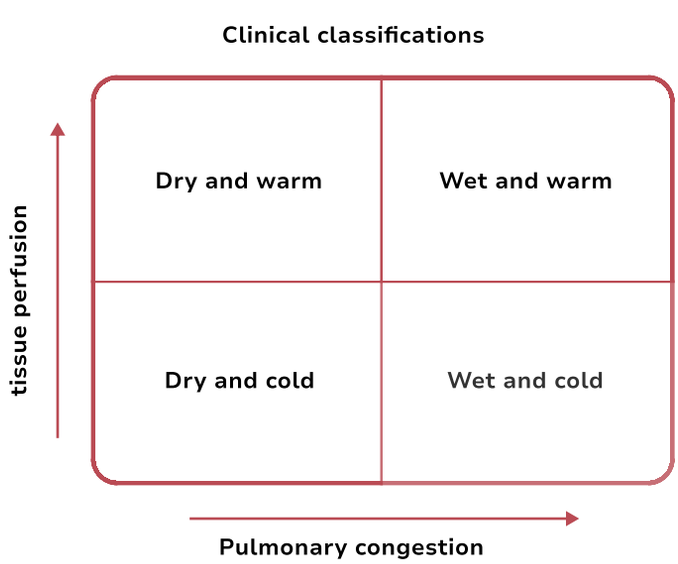
MANAGEMENT PRINCIPLES

- Recognize early and confirm objectively (echocardiography; B-type natriuretic peptide [BNP]/NT-proBNP).
- In acute HF: secure airway/breathing/circulation, correct hypoxia, relieve congestion, and treat triggers (arrhythmia, infection, ischemia, hypertension).
- Treat the underlying etiology (revascularization for ischemia; valve intervention for severe lesions).
- Start and up-titrate guideline-directed medical therapy for HFrEF: ACE inhibitor or angiotensin receptor-neprilysin inhibitor (ARNI), beta-blocker, mineralocorticoid receptor antagonist, and SGLT2 inhibitor; individualize for HFpEF/HFmrEF.
- Pair drugs with non-pharmacological measures: sodium restriction, fluid management, exercise-based cardiac rehab, weight and vaccine optimization, smoking/alcohol cessation.
- Monitor closely: symptoms/NYHA class, weight, renal function, electrolytes, blood pressure, adherence; adjust therapy.
- Prevent complications (arrhythmias, thromboembolism, recurrent congestion); anticoagulate atrial fibrillation when appropriate.
- Refer to higher centers for persistent symptoms despite optimal therapy or for device/surgical options.
- Adapt to resource settings: prioritize affordable, widely available treatments and standardized care pathways.

APPROACH TO MANAGEMENT

The approach should be stepwise, integrating acute stabilization, cause-specific therapy, and long-term disease modification.

1. **Initial Assessment**
   - Determine if the patient is in acute decompensated or chronic stable heart failure.
   - Assess severity using NYHA class and hemodynamic status (e.g., warm/dry vs. cold/wet profile)



Profile	Findings	First-line treatment	Avoid / cautions
Warm & Dry (compensated)	No congestion; good perfusion	Optimize guideline-directed medical therapy (GDMT): angiotensin receptor-neprilysin inhibitor (ARNI) or angiotensin-converting enzyme inhibitor (ACEi)/angiotensin receptor blocker (ARB), evidence-based beta-blocker, mineralocorticoid receptor antagonist (MRA), sodium-glucose cotransporter-2 inhibitor (SGLT2i); lifestyle and follow-up	Over-diuresis; unnecessary IV meds
Warm & Wet (congested, perfused)	Dyspnea, elevated JVP, edema; blood pressure (BP) often normal/high	Intravenous (IV) loop diuretic (dose ≥ home total daily); add IV vasodilator (e.g., nitroglycerin) if hypertensive; salt/fluid restriction; treat triggers	Routine inotrope use (no hypoperfusion); watch renal function and electrolytes
Cold & Dry (hypoperfused, not congested)	Cool extremities, low output; flat JVP	If truly volume-depleted, cautious fluid trial (250-500 mL crystalloid) with close reassessment; if no response and BP low, consider inotrope (e.g., dobutamine)	Aggressive diuresis; vasodilators if hypotensive; hold/reduce beta-blocker during instability
Cold & Wet (hypoperfused and congested)	Pulmonary edema, edema + signs of shock; often low BP	ICU care. IV loop diuretic, vasodilator if BP allows; if hypotensive, inotrope ± vasopressor (norepinephrine). Urgent workup for ischemia; consider mechanical circulatory support if refractory	Delays in decongestion; continuing full-dose vasodilators in hypotension; unmonitored inotrope use

2. **Stabilization (if acutely unwell)**
   - Secure airway and oxygenation; provide non-invasive or invasive ventilation if needed.
   - Initiate diuretics for volume overload; adjust dose based on urine output and renal function.
   - Treat precipitating factors such as infections, arrhythmias, or acute coronary syndromes immediately.
3. **Etiology Identification**
   - Evaluate for ischemic heart disease, hypertension, valvular lesions, myocarditis, or other underlying causes.
   - Address reversible causes (e.g., thyroid dysfunction, anemia, alcohol excess).
4. **Initiation of Guideline-Directed Medical Therapy (GDMT)**
   - HFrEF: Start ACE inhibitor (or ARNI), beta-blocker, MRA, and SGLT2 inhibitor as tolerated.
   - HFmrEF and HFpEF: Focus on control of blood pressure, comorbidities, and symptom relief; consider SGLT2 inhibitors.
   - Titrate drugs gradually to target or maximally tolerated doses.

5. **Non-Pharmacological Measures:** Sodium restriction (2-3 g/day), fluid restriction in selected patients (1.5-2 L/day); Daily weight monitoring; educate on recognizing signs of fluid retention; Encourage gradual physical activity when stable; consider cardiac rehabilitation.
6. **Ongoing Monitoring:** Regular review for symptom changes, adverse drug effects, and biochemical parameters. Adjust medications based on renal function, potassium, and blood pressure.
7. **Advanced Therapies & Referral:** Refer for device therapy (ICD/CRT) if indicated. Consider referral to a tertiary center for advanced HF therapies or transplant evaluation in refractory cases.
8. **End-of-Life and Palliative Care:** For advanced, non-transplant candidates, focus on symptom control, quality of life, and advance care planning.

MANAGEMENT OF ACUTE HEART FAILURE (AHF)

Goals: Rapid stabilization, relief of congestion, restoration of adequate perfusion, prevention of organ damage, and identification/treatment of precipitating cause.

Initial Approach (Emergency/Acute Setting):

1. **Airway & Breathing** - Oxygen if hypoxemic (SpO_2 <90%). Consider non-invasive ventilation in pulmonary edema.
2. **Circulation** - Continuous ECG, BP, and SpO_2 monitoring; establish IV access.
3. **Symptom Relief & Hemodynamic Optimization:**
   - **Congested (wet) & warm:** IV loop diuretics (furosemide 20-40 mg IV bolus; higher if already on oral diuretics).
   - **Congested & cold (hypoperfused):** IV diuretics + cautious inotropes (dobutamine, milrinone) if persistent hypoperfusion.
   - **Hypotensive shock:** Vasopressors (norepinephrine) + inotropes, urgent escalation.
4. **Vasodilators:** In selected hypertensive patients with preserved BP, IV nitrates for rapid symptom relief. (See below)
5. **Identify and treat triggers:** ACS, arrhythmias, infection, uncontrolled hypertension, medication non-adherence.

6. **Monitoring:** Urine output, weight, electrolytes, renal function, BNP/NT-proBNP trend if available.

Discharge Planning from Acute Episode:

- Ensure clinical euvolemia.
- Optimize guideline-directed medical therapy (GDMT) before discharge.
- Arrange early post-discharge review (within 7 days).

Vasodilators in Acute Heart Failure (Hypertensive, Preserved BP)

Drug	Dose	Route	Cautions
Nitroglycerin	Start at 5-10 mcg/min, increase by 5-10 mcg/min every 3-5 min as needed; usual effective dose 20-100 mcg/min	IV infusion	Avoid if systolic BP <90 mmHg; use with caution in right ventricular infarction or severe aortic stenosis; monitor for headache, tachycardia, hypotension
Isosorbide dinitrate	1-2 mg IV bolus every 10 min or continuous infusion at 2-10 mg/hr	IV bolus or infusion	Same as above; avoid with concurrent PDE-5 inhibitors (e.g., sildenafil, tadalafil) within past 24-48 h due to risk of severe hypotension
Sodium nitroprusside	Start at 0.3 mcg/kg/min, titrate up to max 10 mcg/kg/min (usually ≤2 mcg/kg/min in most cases)	IV infusion	Monitor for cyanide/thiocyanate toxicity (especially in renal/hepatic impairment or prolonged use); avoid in hypotension; requires continuous BP monitoring

PHARMACOLOGICAL THERAPY

- **HFrEF (EF <40%):** Initiate and titrate guideline-directed quadruple therapy:
  1. **ACE inhibitor** (or **ARNI** if feasible) - reduces mortality and hospitalization.
  2. **Beta-blocker** - improves survival, reverses remodeling.
  3. **Mineralocorticoid receptor antagonist (MRA)** - reduces morbidity/mortality.
  4. **SGLT2 inhibitor** - reduces HF admissions and CV death regardless of diabetes status.
- **Diuretics** (loop ± thiazide) - for symptom relief in congestion; adjust to maintain euvolemia.
- **HFmrEF / HFpEF:** Focus on comorbidity control (BP, ischemia, AF, CKD, COPD) and symptom relief; SGLT2 inhibitors (dapagliflozin, empagliflozin) may provide benefit.
- **Acute decompensation:** IV loop diuretics, vasodilators if BP stable; inotropes for low-output states with hypoperfusion.

Cautions: Monitor renal function, potassium, and blood pressure; avoid abrupt withdrawal of beta-blockers in decompensation unless severe shock is present.

Table - Guideline-Directed Medical Therapy (GDMT) for HFrEF - Adults (≥18 years)

Class	Drug	Indication	Starting Dose	Target Dose	Route	Key Cautions
***ACE Inhibitors (ACEIs)**	Enalapril	All patients with HFrEF unless contraindicated	2.5 mg BID	10-20 mg BID	PO	Avoid in pregnancy, angioedema, bilateral renal artery stenosis; monitor creatinine & K^+
	Lisinopril	Same as above	2.5-5 mg daily	20-35 mg daily	PO	Same as above
ARNI (Angiotensin Receptor-Neprilysin Inhibitor)	Sacubitril/ Valsartan	Preferred over ACEI in symptomatic HFrEF NYHA II-III	24/26 mg BID (low) or 49/51 mg BID (std)	97/103 mg BID	PO	Stop ACEI ≥36 hrs before start; avoid in pregnancy, angioedema; monitor BP, renal function, K^+
ARBs (if ACEI/ ARNI not tolerated)	Valsartan	HFrEF with ACEI/ ARNI intolerance	40 mg BID	160 mg BID	PO	Avoid in pregnancy, bilateral renal artery stenosis; monitor creatinine & K^+
	Losartan	Same as above	25-50 mg daily	150 mg daily	PO	Same as above
Beta-blockers (evidence-based only)	Carvedilol	Stable HFrEF NYHA II-IV	3.125 mg BID	25 mg BID (<85 kg) or 50 mg BID (≥85 kg)	PO	Avoid in severe asthma, bradycardia, high-degree AV block without pacemaker
	Metoprolol succinate (ER)	Same as above	12.5-25 mg daily	200 mg daily	PO	Same as above
	Bisoprolol	Same as above	1.25 mg daily	10 mg daily	PO	Same as above
Mineralocorticoid Receptor Antagonists (MRAs)	Spironolactone	NYHA II-IV with EF ≤35%, post-MI with EF ≤40%	12.5-25 mg daily	25-50 mg daily	PO	Avoid if K^+ >5 mmol/L or eGFR <30; gynecomastia risk
	Eplerenone	Same as above; alternative if spironolactone not tolerated	25 mg daily	50 mg daily	PO	Less endocrine side effects; same renal/K^+ cautions
SGLT2 Inhibitors	Dapagliflozin	HFrEF, NYHA II-IV, with/without diabetes	10 mg daily	10 mg daily	PO	Use if eGFR ≥30; monitor for genital infections, dehydration

	Empagliflozin	Same as above	10 mg daily	10 mg daily	PO	eGFR ≥20 Rest same as above
HR Control in Sinus Rhythm	Ivabradine	Symptomatic HFrEF with EF ≤35%, sinus rhythm, HR ≥70 bpm on max beta-blocker	5 mg BID	7.5 mg BID	PO	Avoid in AF, bradycardia, severe liver disease; monitor HR & rhythm
Diuretics (for fluid overload)	Furosemide	Relief of congestion in HF (any EF)	20-40 mg daily or BID	Adjust to maintain euvolemia	PO/IV	Monitor electrolytes, renal function; risk of hypovolemia, hypokalemia
	Bumetanide	Same as above, alternative if poor oral absorption	0.5-1 mg daily or BID	Adjust as needed	PO/IV	Same as above
	Torsemide	Same as above, longer duration	10-20 mg daily	Adjust as needed	PO	Same as above

*Key cautions for all ACEIs in HF:

- Avoid if history of angioedema, pregnancy, bilateral renal artery stenosis.
- Monitor serum creatinine and potassium within 1-2 weeks of initiation or dose change.
- Reduce dose or hold if potassium >5.5 mmol/L or creatinine rises >30% from baseline.
- Avoid combination with ARB + MRA (triple RAAS blockade) due to hyperkalemia risk.

NON-PHARMACOLOGICAL INTERVENTIONS

- **Sodium restriction:** Limit dietary sodium to less than 2 g/day to help control fluid retention.
- **Fluid restriction:** Restrict total daily intake to 1.5-2 L/day in patients with hyponatremia or persistent fluid overload.
- **Daily weight monitoring:** Instruct patients to weigh themselves at the same time each morning; a gain of >2 kg in 3 days may indicate fluid retention requiring prompt medical review.
- **Exercise:** Encourage regular, moderate-intensity physical activity (e.g. walking,cycling) as tolerated to improve functional capacity and quality of life; avoid overexertion.
- **Lifestyle:** Stop smoking, limit alcohol, maintain a healthy BMI
- **Ensure vaccinations** (influenza, pneumococcal).

Conditional Recommendations for Low-Resource Settings

- **Diagnosis without echocardiography**: Base diagnosis on a combination of history (dyspnea, orthopnea, fatigue), physical examination (elevated JVP, rales, S3 gallop, edema), and response to initial therapy.
- **BNP/NT-pro-BNP testing**: Use only when the diagnosis remains uncertain after clinical evaluation and the test is available at a reasonable cost.
- **Medication regimen**: Prioritize essential drugs with proven mortality benefit (ACE inhibitors/ARBs, beta-blockers, diuretics as needed) in simplified dosing schedules to improve adherence.
- **Task shifting**: Train primary care providers and nurses in recognition, initial stabilization, and follow-up care when specialists are not available.

PEDIATRIC HEART FAILURE

Symptoms by age group

Neonate	Infant	Older children
• Lethargy • Fast breathing • Poor suck • Reduced urine output • Cold extremities	• Rapid, labored breathing • Excessive sweating • Feeding difficulty (suck-rest-suck cycles) • Poor growth • Frequent chest infections	• Breathlessness • Effort intolerance • Growth retardation • Puffiness of face/extremities • Abdominal distension

Red flags (Urgent escalation)	Common heart failure mimics	Key signs on examination
• Poor peripheral perfusion • Oliguria/reduced urine output • Elevated lactate • Altered sensorium	• Sepsis • Respiratory distress syndrome • Inborn errors of metabolism • Bronchiolitis (infants)	• Tachypnea with intercostal/subcostal retractions (RR >60/min in <1 year; >50/min in 1-2 years) • Tachycardia (HR >160/min in <1 year; >140/min in 1-2 years) • Hepatomegaly • Auscultation: basal crackles (limited sensitivity/specificity) • S3 gallop, murmurs • Raised JVP (not useful in infants) • Peripheral edema

Essential investigations

Test	Findings	Examples
Chest X-ray	Cardiac silhouette, pulmonary vasculature, PA dilation, associated skeletal abnormalities	Cardiomegaly with pulmonary plethora suggests overcirculation; oligemia suggests reduced pulmonary flow
ECG	Arrhythmia, chamber hypertrophy, ischemia; identifies some treatable causes	Deep Q waves in I, aVL, V5-V6 can suggest ALCAPA; hypocalcemia → QTc prolongation
Basic labs	Acid-base, perfusion, metabolic status	ABG, lactate, electrolytes, renal/liver function baselines and for drug monitoring

	Optional	Thyroid function test, Natriuretic Peptide (NP) Cardiac enzymes (troponin I, T, CKM) and In suspected cases of myocarditis Viral Panel
Echocardiography	Defect anatomy, ventricular function, valve regurgitation, pulmonary pressures	Obtain urgently when HF suspected; guides definitive management

Modified Ross classification for Heart failure in children	
Class I:	No symptoms/limitations
Class II:	Mild tachypnea/sweating during feeds in infants/ dyspnoea on exertion in older children but no growth failure
Class III:	Significant tachypnea or sweating during feeds/marked dyspnoea on exertion/prolonged feeding time with growth failure
Class IV:	Symptoms (tachypnoea, retractions, grunting and sweating) even at rest with growth failure

Age-specific normal vitals

Age group	Heart rate (awake, bpm)	Respiratory rate(/min)	Systolic BP (mmHg)	SpO_2 (room air)
Neonate (0-28 d)	100-205	40-60	~60-76	≥95%
Infant (1-12 mo)	100-180	30-53	72-104	≥95%
Toddler (1-3 y)	98-140	22-37	86-106	≥95%
Preschool (4-5 y)	80-120	20-28	89-112	≥95%
School-age (6-12 y)	75-118	18-25	97-115	≥95%
Adolescent (13-18 y)	60-100	12-20	110-131	≥95%

Use the vitals table to interpret symptom severity (tachycardia, tachypnea, hypotension) for the child's age when applying the severity criteria above.

Management goal: Correct the underlying cause; Reduce associated morbidity and mortality; Improve functional status and quality of life

Drug dose reference			
Drug	**Usual starting dose**	**Titration / max**	**Key monitoring**
Furosemide (PO/IV)	1 mg/kg/dose (neonate 0.5-1 mg/kg)	q6-12 h; max 2 mg/kg/dose; refractory: 0.05-0.2 mg/kg/h infusion	Urine output, weight, $Na^+/K^+/Mg^{2+}$, creatinine; ototoxicity
Spironolactone (PO)	1-3 mg/kg/day	Split q12-24 h	K^+, creatinine

Chlorothiazide (PO/IV)	5-10 mg/kg/dose q12 h	-	Na^+/K^+, volume status
Hydrochlorothiazide (PO)	1-2 mg/kg/day q12-24 h	-	Na^+/K^+, volume status
Enalapril (PO) — for LV volume load/AV valve regurgitation	0.05 mg/kg/dose once daily (or 0.1 mg/kg/day in 1-2 doses)	Titrate every 1-2 weeks to 0.1-0.4 mg/kg/day in 1-2 doses; max ~0.5-0.6 mg/kg/day (not >40 mg/day)	BP (first-dose hypotension), creatinine/eGFR, K^+; cough/angioedema. Avoid or use specialist oversight in neonates (<1 mo).
Lisinopril (PO) - for LV volume load/AV valve regurgitation (school-age/ adolescents)	0.07 mg/kg/day once daily (max initial 5 mg)	Titrate to 0.2-0.6 mg/kg/day once daily; max 40 mg/day	BP, creatinine/eGFR, K^+; counsel on teratogenicity (adolescents)

General Measures

- **Fluid restrictions**- in acute HF with lung congestion, peripheral edema despite diuretics and in presence of hyponatremia
- **Rest:** Restriction of activity ◦Activity as tolerated for older children with chronic compensated HF
- **Correction of Anemia** - Hematinics; Blood transfusion only for severe anemia (Hb < 7gm/dl)
- **Nutrition** - NG feeds for infants in acute severe HF. ◦In infants calorie intake of 120- 150kcal/kg/with a fluid intake of 100 ml/kg/day. (thickening of feeds or by adding coconut oil/medium chain triglyceride). In older children increase protein content of diet while optimizing the fat and carbohydrate intake. Supplement Calcium and it D3;
- Dietary restriction of sodium is generally not recommended in children unless there is severe edema unresponsive to diuretic therapy
- **Supplementary Oxygen**: May be necessary when there is respiratory distress but must be used with caution in L-R shunts and avoided in neonates with duct dependent lesions

Note: Always individualize by lesion, renal function, blood pressure, and local protocols.

ASSESSMENT OF RESPONSE

Monitor improvement or deterioration by tracking key parameters:

Monitoring domain	Parameter	Frequency	Why it matters / interpret
Symptoms	Dyspnea, fatigue, exercise tolerance, orthopnea, PND	At each contact; patient self-monitoring daily	Worsening → congestion/low output; improvement → response
Weight	Same-time daily weight	Daily	Early signal of fluid retention or over-diuresis
Blood pressure & heart rate	Sitting BP, resting HR	Each visit; home logs if available	Hemodynamic stability and med tolerance
Renal function & electrolytes	Creatinine/eGFR, sodium, potassium	After med/diuretic changes; then periodic	Detects diuretic or RAAS-blocker complications

Review & follow-up

Review & follow-up	Frequency	Purpose	Actions
Clinic/tele follow-up	Every 2-4 weeks during initiation/adjustment; every 3-6 months once stable	Safety and response check	Titrate toward evidence-based targets; watch side effects
Reassessment each visit	Each encounter	Track NYHA class, adherence, comorbidities, triggers	Optimize therapy, reinforce education, address triggers

Step-up treatment

When to step up (intensify treatment)	What to look for	Actions
Persistent or worsening symptoms	↑ NYHA class, reduced exercise tolerance, new/worse orthopnea/ PND	Up-titrate GDMT as tolerated; add/increase loop diuretic; consider adding MRA, SGLT2i, switch to ARNI; evaluate for CRT/ICD if criteria met
Congestion/rapid weight gain	Edema, rising JVP, crackles; weight gain (e.g., >1-2 kg in 2-3 days)	Intensify diuresis; add thiazide-type diuretic short-term for diuretic resistance; reinforce sodium/fluid limits
Uncontrolled BP or tachyarrhythmia	BP above target; AF with RVR, frequent ectopy	Optimize beta-blocker/RAAS blockade; rate/rhythm control; anticoagulate AF as indicated
Recurrent decompensations	Multiple ER visits/admissions, rising BNP/NT-proBNP	Multidisciplinary review; check adherence/causes; consider advanced therapies; refer to HF clinic/higher center

When to step down (de-escalate/hold)	What to look for	Actions
Hypotension or intolerance	Symptomatic low BP, dizziness, syncope	Temporarily reduce/hold vasodilators (e.g., ACEi/ARNI), down-titrate beta-blocker if symptomatic; reassess volumes and restart cautiously
Bradycardia or conduction issues	HR <50 bpm with symptoms, new AV block	Reduce/hold beta-blocker or other rate-slowing drugs; ECG review
Worsening renal function or hyperkalemia	Rising creatinine/eGFR drop; K^+ >5.5 mmol/L	Adjust/hold RAAS blockers or MRA; review NSAIDs and dehydration; correct potassium; reintroduce once safe
Over-diuresis/volume depletion	Rapid weight loss, orthostasis, prerenal azotemia	Reduce diuretic dose; rehydrate judiciously
Intercurrent illness or procedures	Sepsis, AKI, peri-operative period	Temporarily hold nephrotoxic/RAAS-affecting meds per clinical judgment; restart after stabilization

PROGNOSIS

Outcomes in heart failure vary widely and are influenced by multiple factors. Reduced ejection fraction (HFrEF) generally carries a higher mortality risk compared to preserved EF (HFpEF). Advanced NYHA class (III-IV) is associated with poorer survival and higher hospitalization rates.

Prognosis worsens with coexisting conditions such as chronic kidney disease, diabetes, and ischemic heart disease. Good adherence to evidence-based therapies, timely adjustment of treatment, and control of comorbidities can significantly improve quality of life, reduce hospitalizations, and extend survival.

REFERRAL LINKAGES

In resource-limited contexts, timely referral is essential to optimize outcomes while avoiding unnecessary delays in care. Patients with severe or refractory symptoms despite optimal therapy, recurrent hospitalizations, or signs of rapid clinical deterioration should be referred from primary or secondary care to facilities with advanced diagnostic and treatment capabilities.

- **Primary to Secondary Care:** For confirmation of diagnosis (echocardiography), optimization of pharmacotherapy, and management of moderate decompensation not controlled in primary care.
- **Secondary to Tertiary Care:** For patients requiring advanced interventions such as intravenous inotropes, mechanical circulatory support, or evaluation for heart transplantation where available.
- **Specialist Consultation:** Engage cardiology input early when diagnosis is uncertain, comorbidities complicate management, or advanced therapies are being considered.

Clear communication and transfer protocols between levels of care are critical, especially in geographically dispersed settings such as island nations, to ensure patients receive timely specialist input and appropriate escalation of treatment.

COMPLICATIONS

Heart failure can lead to multiple systemic complications that worsen prognosis and increase mortality risk. Early recognition and prevention of these complications are essential to improving survival and quality of life.

Common complications include:

Complication	Mechanism	Key risks	Prevention / first-line management
Arrhythmias (AF, VT, VF)	Atrial fibrillation, ventricular tachycardia/fibrillation causing loss of atrial kick or malignant ventricular rhythms leading to hemodynamic instability	Stroke (AF), syncope, cardiogenic shock, death	Rate/rhythm control, anticoagulate AF by risk stratification tool *CHA_2DS_2-VASc, correct electrolytes, optimize HF meds; ICD for secondary prevention/ high-risk primary prevention
Renal dysfunction (cardiorenal syndrome)	Reduced renal perfusion, venous congestion; diuretic/RAAS blocker effects; nephrotoxins	Rising creatinine, fluid overload, electrolyte disorders, worse outcomes	Optimize volume status, avoid nephrotoxins, adjust/hold RAAS blockers if needed, monitor creatinine/ K^+, consider diuretic strategy (loop ± thiazide)
Thromboembolism	Stasis in dilated chambers (esp. with AF) → clot formation	Ischemic stroke, systemic embolism	Anticoagulate when indicated (AF/low EF with LV thrombus), maintain sinus/ rate control, manage HF to reduce chamber dilation
Sudden cardiac death	Ventricular arrhythmias or conduction block, common in ischemic/advanced HF	Immediate death	Guideline-directed therapy (BB, ACEi/ ARNI, MRA, SGLT2i), ICD in eligible HFrEF, treat ischemia, correct electrolytes, consider CRT if criteria met

***CHA_2DS_2-VASc Score**: A risk stratification tool to assess stroke risk in non-valvular AF:

C: Congestive heart failure (1); **H**: Hypertension (1); **A_2**: Age ≥75 (2); **D**: Diabetes mellitus (1); **S_2**: Prior Stroke/TIA/thromboembolism (2); **V**: Vascular disease (1); A: Age 65–74 (1); **Sc**: Sex category (female = 1).

Anticoagulation: Indicated if score ≥2 (men) or ≥3 (women); options include DOACs (e.g., apixaban, rivaroxaban) or warfarin (if valvular AF or mechanical valves).

PREVENTION AND HEALTH PROMOTION

Prevention of heart failure begins with controlling modifiable cardiovascular risk factors such as hypertension, diabetes, dyslipidemia, obesity, and smoking.

- Community-based screening programs can help detect these conditions early, especially in high-risk groups. Public health campaigns should promote regular...

physical activity, a balanced diet low in salt and trans fats, and avoidance of excessive alcohol.

- For those with established cardiovascular disease, adherence to prescribed medications, regular follow-up, and timely treatment of acute events like myocardial infarction are critical to preventing progression to heart failure. In low-resource settings, integrating heart failure prevention into existing primary healthcare services, non-communicable disease programs, and maternal health initiatives can extend reach.
- Health education should empower individuals to recognize early symptoms such as breathlessness, swelling, or unexplained fatigue, encouraging prompt medical attention to prevent advanced disease and complications.

PATIENT EDUCATION

Patient education in heart failure aims to equip individuals and their caregivers with the knowledge and skills needed to manage the condition effectively, prevent complications, and improve quality of life.

- Education should focus on understanding the disease process, recognizing early warning signs of worsening symptoms, and knowing when to seek medical help.
- Patients should be taught the importance of adhering to medication regimens, maintaining dietary restrictions such as limiting salt and fluid intake, and monitoring daily weight to detect fluid retention early.
- Encouraging regular physical activity within safe limits, avoiding harmful habits like smoking or excessive alcohol consumption, and attending scheduled follow-ups are essential components.
- Involving caregivers ensures consistent support in implementing lifestyle changes and recognizing emergencies. Ultimately, education should foster self-management, reduce hospitalizations, and enhance long-term outcomes.

Instructions to Patient/Caregiver

Do's	Don'ts
Take all prescribed medicines at the correct dose and time.	Do not skip or stop medicines without consulting your doctor.
Monitor and record body weight daily; report a gain of >2 kg in 3 days to your healthcare provider.	Avoid over-the-counter painkillers like NSAIDs (e.g., ibuprofen, diclofenac) unless approved, as they can worsen HF.
Limit salt intake to less than 2 g/day (avoid adding salt at the table, check food labels).	Don't drink excessive fluids or alcohol.

Restrict fluid intake to 1.5-2 L/day if advised, especially if hyponatremic.	Avoid high-salt foods like pickles, chips, instant noodles, and processed meats.
Stay physically active within your tolerance; follow your doctor's recommended exercise plan.	Don't ignore early signs of worsening HF (breathlessness, swelling, fatigue).
Keep all scheduled medical appointments and laboratory checks.	Avoid strenuous activity or lifting heavy weights if symptomatic.
Elevate legs when sitting to reduce swelling.	Avoid smoking, chewing tobacco or exposing yourself to second hand smoke.
Get vaccinated against influenza and pneumococcal disease if recommended.	
Seek immediate care if you experience worsening breathlessness, chest pain, fainting, or rapid swelling.	

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