REVIEW

Heart failure in sub-Saharan Africa: A clinical approach

S Kraus,[1] FCP (SA); **G Ogunbanjo**,[2] MFamMed, FCFP (SA); **K Sliwa**,[1,3] MD, PhD; **N A B Ntusi**,[1] FCP (SA), DPhil

[1] *Division of Cardiology, Department of Medicine, Faculty of Health Sciences, University of Cape Town and Groote Schuur Hospital, Cape Town, South Africa*
[2] *Department of Family Medicine and Primary Health Care, Sefako Makgatho Health Sciences University, Pretoria, South Africa*
[3] *Hatter Institute for Cardiovascular Research in Africa, Department of Medicine, Faculty of Health Sciences, Groote Schuur Hospital and University of Cape Town, South Africa*

Corresponding author: *N A B Ntusi (ntobeko.ntusi@gmail.com)*

Despite medical advances, heart failure (HF) remains a global health problem and sub-Saharan Africa (SSA) is no exception, with decompensated HF being the most common primary diagnosis for patients admitted to hospital with heart disease. In SSA the in-hospital mortality rate of decompensated HF is up to 8.3%. HF is a clinical syndrome that is caused by a diverse group of aetiologies, each requiring unique management strategies, highlighting the need for diagnostic certainty and a broad understanding of the complex pathophysiology of this condition. While there are a number of advanced medical, device and surgical interventions being tailored for HF internationally, the fundamental basic principles of HF management, such as patient education, effective management of congestion and initiation of disease-modifying medical therapies, remain a challenge on our continent. This review addresses both the epidemiology of HF in SSA and principles of management that focus specifically on symptom relief, prevention of hospitalisation and improving survival in this population.

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Heart failure (HF) is a major public health challenge, accounting for significant morbidity and premature mortality globally, including in sub-Saharan Africa (SSA).[1] Owing to high prevalence and poor clinical outcomes, HF is associated with recurrent hospitalisation and substantial healthcare expenditure.[2] In contrast to Western countries, where HF is considered a disease of older persons, in SSA it affects younger individuals.[1-3] Acute decompensated HF is the most common primary diagnosis for patients admitted to hospital with heart disease in SSA, and it is encountered at all levels of care.[1,3,4]

The goals of the clinical approach to HF include: (*i*) correctly diagnosing the clinical syndrome of HF; (*ii*) identifying the underlying cause; and (*iii*) implementing an effective management strategy for symptom control, prolonging survival and reversing factors that predispose to precipitation of HF exacerbations.

Definitions

HF is a clinical syndrome of effort intolerance characterised by breathlessness and fatigue, due to structural and functional abnormalities of the myocardium, resulting in salt and water retention that is associated with neurohormonal adaptations, mainly in the renin-angiotension-aldosterone system (RAAS).

Ejection fraction is the stroke volume (end-diastolic volume minus the end-systolic volume) divided by the end-diastolic volume. Systolic dysfunction is reduced contraction and emptying of the left ventricle, and diastolic dysfunction is impaired relaxation of the left ventricular myocardium resulting in impaired filling of the left ventricle.

Epidemiology of HF in SSA

Although there have been no population-based epidemiological studies of HF in Africa, there have been a number of hospital-based studies that give important insights into the incidence and prevalence of HF in SSA. In contrast to other parts of the world, non-ischaemic aetiologies are predominant, with hypertension, rheumatic heart disease (RHD) and cardiomyopathy accounting for two-thirds of cases of HF in hospitalised patients in the region.[1,5]

Classification of HF

Patients with HF can be divided into two categories: HF with reduced ejection fraction (HF-REF), and HF with preserved ejection fraction (HF-PEF) (Fig. 1). Although there is poor correlation between symptom severity and left ventricular ejection fraction (LVEF), the LVEF carries independent prognostic significance and is considered abnormal when <50%.[2] The diagnosis of HF-PEF is more difficult, and although LVEF is normal or only mildly reduced in this condition, relevant structural heart disease and/or diastolic dysfunction should be present to make this diagnosis. Importantly, HF-PEF is a diagnosis of exclusion where other non-cardiac causes for patients' symptoms must be considered and discounted.[2] Patients with HF-PEF are older, more often female and obese, and more likely to have hypertension and atrial fibrillation, compared to those with HF-REF, and their prognosis appears to be better overall.[6]

Aetiology of HF in SSA

HF is a final common pathway for a number of conditions affecting the heart, and it is useful to classify the aetiology according to the following diseases: (*i*) hypertension; (*ii*) primary myocardial disease that includes cardiomyopathies and myocarditis; (*iii*) valvular heart disease; (*iv*) ischaemic heart disease; (*v*) congenital heart disease; (*vi*) pericardial disease; and (*vii*) pulmonary hypertension (PH) (Table 1). It is important to consider alternative causes for fluid retention (e.g. renal or liver disease) and pulmonary oedema (e.g. neurogenic) in the context of a structurally normal heart.

Pathophysiology

Damage to cardiac myocytes and the extracellular matrix after myocardial injury results in pathological remodelling of the left ventricle with dilatation, impaired contractility, perfusion, fibrosis and electrical instability. If left untreated, these changes worsen over time, exacerbated by additional myocardial injury from neurohormonal imbalance resulting from activation of the RAAS and the sympathetic nervous system, increased cytokine expression, immune and inflammatory changes, altered fibrinolysis and oxidative stress. Reduced cardiac output results in arterial

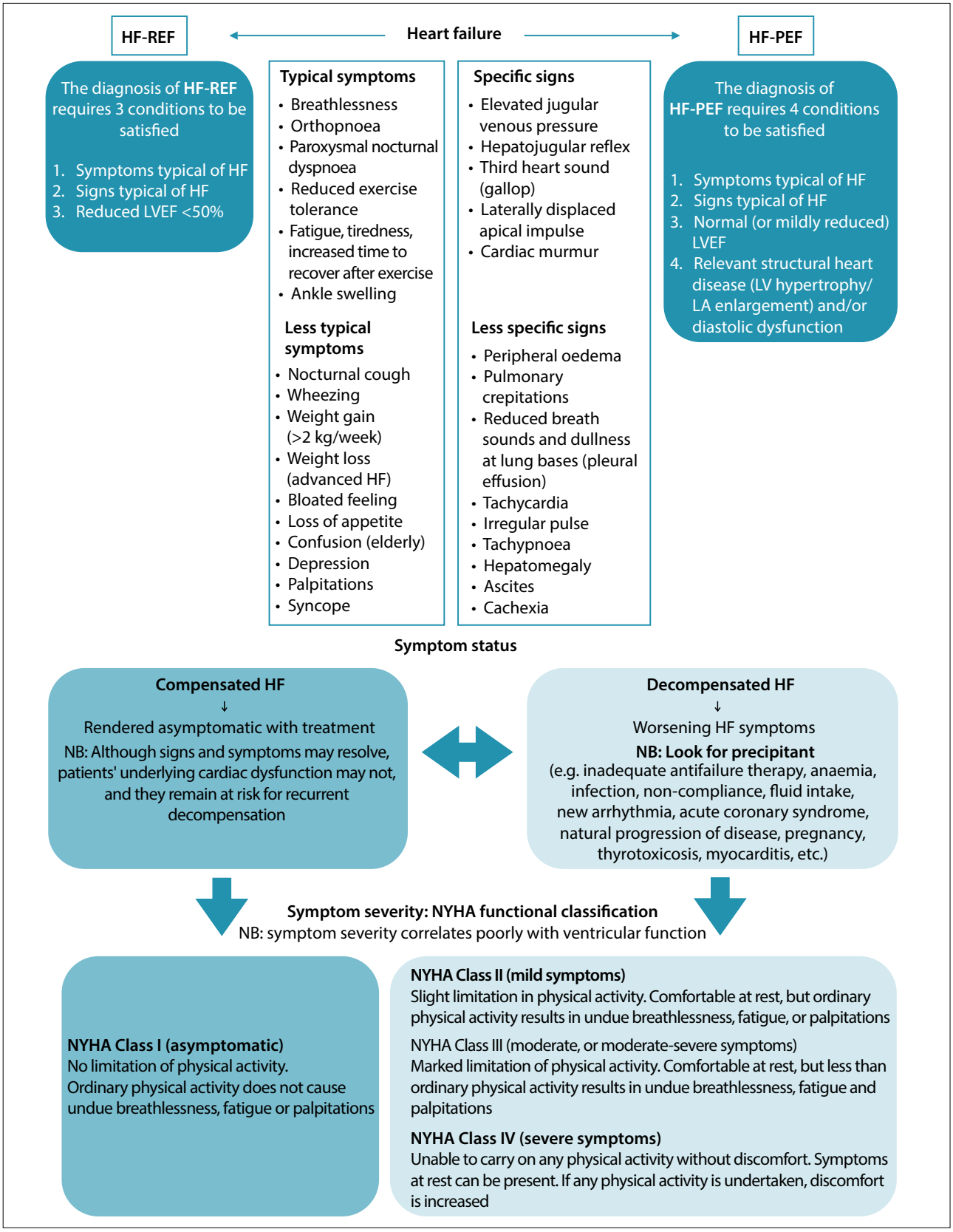

Fig. 1. Classification, clinical profile, grading of severity and natural history of heart failure (NYHA = New York Heart Association; LA = left atrial).

underfilling, leading to renal sodium and water retention via activation of the above-mentioned neuro-endocrine systems, in an attempt to restore arterial circulatory integrity (Fig. 2).[7]

Clinical presentation

The history is key in making the diagnosis of HF, grading symptom severity, and establishing not only the underlying cause but also identifying factors that may have precipitated decompensation (Table 2). The typical symptoms of HF are breathlessness, orthopnoea, paroxysmal

Table 1. Diseases causing heart failure

Hypertension	
Essential hypertension	Unknown
Secondary hypertension	Primary aldosteronism (Conn syndrome), Cushing syndrome, phaeochromocytoma, chronic kidney disease, renal artery stenosis, coarctation of the aorta, obstructive sleep apnoea
Primary myocardial disease; cardiomyopathies and myocarditis	
HCM	Familial Sporadic Obesity, infants of diabetic mothers, amyloid, athletes
DCM	Familial Non-familial Alcohol, pregnancy, tachymyopathy, thyrotoxicosis, myocarditis, nutritional (e.g. thiamine, selenium), drugs (e.g. anthracycline, cocaine), iron overload
ARVC	Familial Non-familial
RCM	Familial Non-familial Endomyocardial fibrosis, radiation, amyloid, carcinoid
Unspecified cardiomyopathy	Familial (left ventricular non-compaction) Non-familial (Takotsubo cardiomyopathy)
Myocarditis	Infective Viral, HIV, bacterial, fungal, helminths, protozoa, rickettsia, spirochetes Toxic/hypersensitivity Anthracycline chemotherapy, alcohol, methamphetamines, other drugs Immune Lupus, rheumatoid arthritis, sarcoidosis
Valvular heart disease	
	Rheumatic heart disease, endocarditis (infective and non-infective), degenerative, myxomatous, congenital
Ischaemic heart disease	
	Atherosclerosis Spasm Atherothrombosis Coronary artery dissection
Congenital heart disease	
	Atrial septal defects, ventricular septal defects, transposition of the great vessels, tetralogy of Fallot, single ventricle, patent ductus arteriosis, etc.

Continued …

Table 1. (continued) Diseases causing heart failure

Pericardial disease	
Pericarditis, pericardial effusion, pericardial constriction	Idiopathic Infectious Viral, tuberculosis, fungal Non-infectious Uraemia, acute myocardial infarction, neoplasm, post-cardiac injury syndrome (trauma, cardiothoracic surgery), systemic auto-immune disease, mediastinal radiation
Pulmonary hypertension	
Pulmonary arterial hypertension	Idiopathic, heritable, drugs/toxins, associated with connective tissue disease, HIV, portal hypertension, CHD, schistosomiasis, chronic haemolytic anaemia, viral hepatitis
Pulmonary veno-occlusive disease	Unknown
PH due to left heart disease	Rheumatic heart disease
PH due to lung disease and/or hypoxia	Post-tuberculous bronchiectasis, chronic obstructive pulmonary disease, occupational lung disease, interstitial lung disease
Chronic thrombo-embolic PH	Thrombophilia, deep venous thrombosis
PH with unclear and/or multifactorial mechanism	

HCM = hypertrophic cardiomyopathy; DCM = dilated cardiomyopathy; ARVC = arrhythmogenic right ventricular cardiomyopathy; PH = pulmonary hypertension; CHD = congenital heart disease; RCM = restrictive cardiomyopathy.

nocturnal dyspnoea, reduced effort tolerance, fatigue, and ankle swelling. The New York Heart Association (NYHA) functional class allows a grading of symptom severity in a standardised manner (Fig. 1).[2] Exploring past medical history, environmental exposures and family history may assist in deciphering a possible aetiology.

The physical examination findings may differ, depending on the underlying aetiology, but pedal oedema, raised jugular venous pressure, a tender hepatomegaly and basal crackles indicate congestive HF. Additional features that can be found on examination are listed in Table 3.

Diagnostic tests in suspected HF

The baseline investigations recommended in clinical assessment of HF are outlined in Table 4. The electrocardiogram (ECG) and echocardiogram are the most useful investigations, as they confirm the presence of underlying structural heart disease. The likelihood of a normal ECG in a patient presenting with HF is low, making it an extremely helpful screening tool.[2] It is recommended that all patients with a new diagnosis of HF undergo echocardiographic evaluation as it confirms the type of structural heart disease present and provides information on cardiac function.[2]

Understanding the aetiology of HF is vital when determining definitive management strategies and prognosis. The pursuit of a correctable cause and identification of reversible factors are central to improving outcomes in these patients. Patients with unexplained HF, particularly those who are not improving on standard therapy, should be referred for specialist review where advanced investigations can be done to establish a diagnosis (Table 4).

Specific aetiologies of HF

Hypertension

Hypertension has been reported as the dominant cause of HF in Africa, responsible for up to 46% of cases of HF in hospitalised patients.[4,5,8] Young hypertensive patients should be investigated for secondary causes of hypertension (Table 1). Standard anti-failure therapy and blood pressure control are the mainstays of therapy.

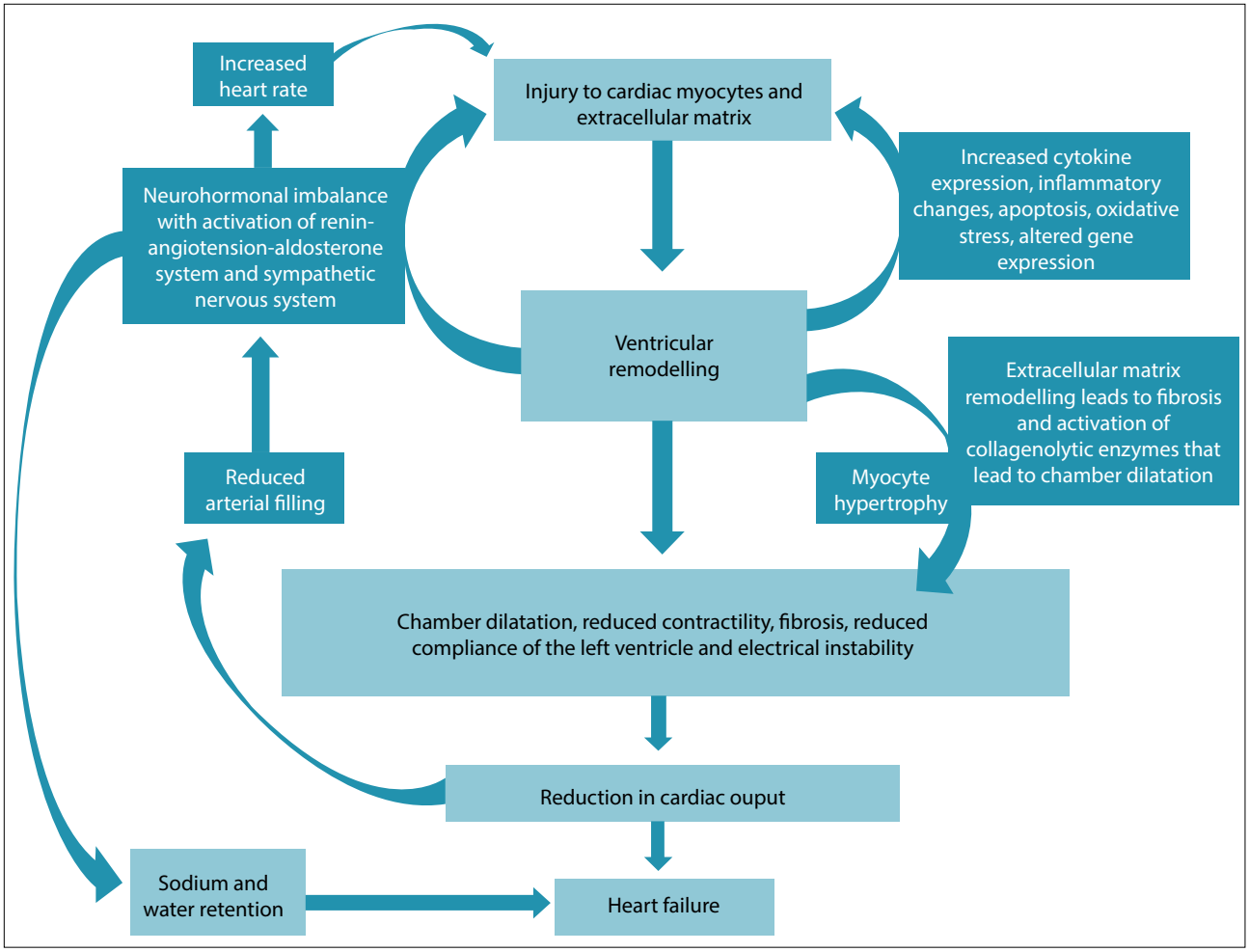

Fig. 2. Pathophysiology of heart failure.

Cardiomyopathy

Cardiomyopathy accounts for 20 - 30% of heart failure in Africans.[1] Most commonly, patients who present with HF have a dilated phenotype, and potentially treatable causes for dilated cardiomyopathy should routinely be excluded (Table 1). Importantly, patients with other forms of cardiomyopathy (hypertrophic cardiomyopathy (HCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), restrictive cardiomyopathy (RCM), and left ventricular non-compaction) presenting with HF should be referred for specialist review, as the management of these conditions is complex and multidisciplinary.[9] Myocarditis should be considered in patients who present with cardiac symptoms and elevated cardiac biomarkers (troponin T or I), ECG abnormalities and/or evidence of functional impairment on echocardiogram, where acute coronary syndrome has been excluded.[10,11] Forty-four percent of HIV-associated cardiomyopathy cases have evidence of myocarditis on endomyocardial biopsy, either as a result of HIV or secondary to opportunistic infections,[12] justifying antiretroviral therapy in these patients.

Valvular heart disease

In contrast to Western populations, where valvular heart disease is mainly degenerative, in SSA valvular heart disease is predominantly caused by rheumatic fever and infective endocarditis. Despite a reduction in RHD as a cause of HF in SSA in recent years, it remains endemic on our continent.[13] The mainstay of treatment for patients with symptomatic valvular heart disease is surgery, complemented by anti-failure therapy and secondary prevention of rheumatic fever with penicillin in RHD. All patients should be referred for evaluation for surgery.

Ischaemic heart disease

Ischaemic heart disease (IHD) is an uncommon cause of HF in SSA, accounting for only 7.7 - 9% of cases.[3,4,8] Although IHD is considered uncommon among black Africans, there has been a notable rise in risk factors for atherosclerotic vascular disease in both urban and rural communities over the last few decades.[14] Patients presenting with ischaemic left ventricular dysfunction require rigorous risk factor management in addition to conventional HF therapy. Patients with suspected coronary artery disease with ongoing symptoms of angina should be referred to a cardiologist for consideration for revascularisation therapy.[2,15]

Table 2. Precipitating factors to consider in acute decompensated heart failure

Anaemia
Onset of a new arrhythmia (e.g. atrial fibrillation/flutter, supraventricular tachycardia, ventricular tachycardia)
Hyperthyroidism
Infection
Pregnancy
Infective endocarditis
Recurrence of rheumatic fever
Renal failure
Malignant hypertension
Myocardial infarction
Non-compliance on maintenance therapy

Congenital heart disease

Although the prevalence of congenital heart disease (CHD) in SSA is considered to be the lowest in the world, it is likely that this

reflects the paucity of readily available estimates and that the true number of individuals affected with CHD is grossly underestimated. While CHD is an important cause of HF in children in SSA, it accounts for only a small percentage of cases of HF in adults.[16] CHD is riddled with complexity and requires management by experienced clinicians.[2]

Pericardial disease

Pericardial disease has a broad aetiology, but tuberculous pericarditis is the commonest cause of pericardial effusion, cardiac tamponade, and constrictive pericarditis in SSA, and carries a high mortality rate despite antituberculosis therapy, pericardiocentesis and pericardectomy.[17]

Pulmonary hypertension

PH is a debilitating progressive disease that leads to right HF, and although little is currently known about the epidemiology of PH in Africa, the reported incidence appears to be higher than in developed countries. The Pan African Pulmonary Hypertension Cohort study hopes to address the paucity of our knowledge. Importantly, many risk factors associated with PH are endemic in SSA (Table 1).[18] PH (not associated with left heart pathology) requires investigation in the absence of significant pulmonary disease.

Table 3. Additional examination findings in heart failure

Structural heart disease/ aetiology	Examination findings that could be present
Conditions that result in left ventricular dilatation and systolic dysfunction	Laterally displaced apex, a third heart sound, cardiac murmur
Pulmonary hypertension	Palpable and/or loud pulmonary component of the second heart sound, parasternal heave, pulmonary pathology, cyanosis, clubbing
Pericardial disease	Elevated venous pressure, pedal oedema and ascites, unremarkable precordial examination and clear lung fields Pericardial rub may be present in pericarditis Hypotension, distended neck veins, muffled heart sounds, and pulsus paradoxus are suggestive of pericardial effusion with tamponade Diastolic knock may be present in constrictive pericarditis
Congenital heart disease	Depending on underlying cardiac lesion; cardiac murmur, signs of pulmonary hypertension, parasternal heave (right ventricular hypertrophy), fixed split of the second heart sound (atrial septal defect), cyanosis, clubbing, surgical scars Radial-radial delay, radial-femoral delay may be present in coarctation of the aorta
Hypertensive heart disease	Blood pressure may be elevated, or normal in end-stage disease Pressure-loaded apex beat, loud aortic component of the second heart sound, fourth heart sound Evidence of target organ damage (retinopathy, proteinuria)

Management of HF

Although a significant portion of HF management falls within the realm of the general practitioner and general physician, it is important to be able to recognise which patients require specialist referral, particularly where there is diagnostic uncertainty and/or failure to improve, or deterioration, on anti-failure therapy.

The goals of treatment in patients with established HF are to: (*i*) relieve symptoms with the aim to improve quality of life and functional capacity; (*ii*) prevent recurrent hospitalisations; and (*iii*) improve survival. In SSA these goals are achieved predominantly through patient education and medical therapy. Despite resource restraints, there is a role for advanced medical, device and surgical interventions, including orthotopic heart transplantation, for HF in SSA. It is important for

Table 4. Investigations in heart failure

Baseline investigations	
Pro-BNP/BNP	Elevated in heart failure Not routinely required
Electrocardiogram	Heart rate and rhythm disturbances (atrial fibrillation/flutter) Electrical conduction abnormalities (left or right bundle branch block, heart block) Cardiac wall and/or chamber abnormalities (ventricular hypertrophy, atrial enlargement, Q-waves)
Chest radiograph	Cardiac size and shape (cardiomegaly) Pulmonary congestion Presence or absence of pulmonary pathology
Echocardiogram	Chamber size Systolic and diastolic function Ventricular wall thickness Valve morphology and function
Advanced investigations	
Cardiovascular magnetic resonance imaging	Cardiac structure, size and function Tissue characterisation Perfusion imaging Late gadolinium imaging (scar) Velocity-encoded flow imaging Defining anatomy in complex CHD
Cardiac computed tomography	Coronary artery angiography
Nuclear medicine imaging	Cardiac function (right and left ventricular ejection fraction) Myocardial perfusion studies
Angiography	Haemodynamic assessment Coronary artery angiography
Endomyocardial biopsy	Histological diagnosis

BNP = brain natriuretic peptide; CHD = congenital heart disease.

Table 5. Patient education

Patients should be well informed about:
- What heart failure is and why symptoms occur
- The underlying cause of their heart failure
- Prognosis
- The treatment options available to them

Patients must be educated about:
- Medications, specifically the role of each drug used to treat heart failure
- Fluid retention and how to manage it (i.e. how to restrict fluids, monitor weight and adjust diuretic therapy accordingly)
- Remembering the names, doses and frequency of medication they are on or bringing the drugs to hospital with them

Patients should be encouraged to make realistic decisions regarding:
- Their ability to work. Temporary or permanent disability grant applications should be made if anticipated time away from work is ≥6 months
- Financial implications related to loss of employment or added healthcare costs
- Legal issues in the event of their death
- Obtaining medical aid, where possible

Patients are at increased risk of depression and may require referral for counselling or antidepressant drug therapy

Exercise
- An active lifestyle should be encouraged
- Heart rate monitoring can be helpful in guiding patients with regard to safe levels of exercise
- Aiming for a maximum heart rate of (180 – age – 20) beats/minute during exercise is recommended

Excessive alcohol consumption should be discouraged and excessive use of caffeine/stimulants avoided

The dangers of illicit drug use should be addressed

In women, pregnancy and contraception should be discussed:
- Contraception is recommended in all patients with cardiac disease
- Women need to be well informed of the dangers of pregnancy, particularly in the setting of LVEF <45%, pulmonary hypertension or mitral stenosis
- Patients should be informed that medication used to treat heart failure, such as ACE inhibitors, are teratogenic
- Patients who strongly desire a pregnancy, or who have fallen pregnant inadvertently, should be referred to a specialist centre for assessment
- Pregnant patients with underlying cardiac disease are at extremely high risk and require a multidisciplinary team (cardiologist, obstetrician, anaesthetist) to manage them throughout their pregnancy, during delivery and post partum

clinicians to familiarise themselves with the indications and the availability of services.[2]

Patient education

It is the attending clinicians' responsibility to inform patients about their condition. Important aspects to consider are listed in Table 5.

Optimising medical therapy

Medical therapy consists of two components: (*i*) disease-modifying drugs consisting of three neurohormonal antagonists (angiotensin-converting enzyme (ACE) inhibitors (or angiotensin receptor blockers), beta-blockers, and mineralocorticoid receptor antagonists) that are fundamental in modifying the course of disease and improving survival; and (*ii*) symptomatic therapies, such as diuretics and digoxin, that relieve congestion, reduce hospitalisation and improve quality of life.

Fig. 3 illustrates an approach for the medical management of HF. The first step is to manage and alleviate congestion. Disease-modifying drugs should be introduced at recommended starting doses and titrated up to maximum tolerated doses over a number of weeks (Table 6).[2] Digoxin has been shown to relieve symptoms and reduce hospitalisations,[19] but is associated with an increase in mortality in HF patients.[20] Current guidelines recommend low-dose digoxin in selected patients who remain symptomatic despite optimal ACE inhibitor and beta-blocker therapy. Hypokalaemia and renal failure predispose patients to digoxin toxicity, and its use is contraindicated in these circumstances.[21]

The prognosis of acute decompensated HF remains poor, with greater severity of congestion being associated with worse outcomes. Acute decompensated HF is associated with an in-hospital mortality rate of up to 8.3% in Africa.[8] Fluid retention and congestion are responsible for 90% of HF hospital admissions, and even with diuretic therapy approximately 40% of patients are discharged with unresolved congestion.[22]

The mainstay therapy for the treatment of congestion is loop diuretics. Diuretic resistance is the failure to adequately control salt and water retention despite appropriate dose escalation of loop diuretics. The dose-response curve for loop diuretics shifts in HF, resulting in the need for increased doses of the drug to achieve a therapeutic effect; thus inadequate dosing must be differentiated from diuretic resistance. Infrequent dosing can result in rebound salt and water retention, which can be addressed by increasing the frequency of dosing or changing to a continuous intravenous infusion.

Strategies for overcoming diuretic resistance include the addition of thiazide diuretics and/or spironolactone. Importantly, diuretic combinations can result in severe volume depletion and electrolyte disturbances, and should only be used in circumstances where volume status and electrolytes can be monitored. RAAS activation plays an important role in sodium and water retention by increasing distal sodium reabsorption in the kidney. Introducing ACE inhibitors is crucial in managing congestion. The challenge is maintaining adequate arterial blood pressure, as low blood pressure drives plasma renin activity and further activation of RAAS (Fig. 4).[22]

Advanced medical, device and surgical interventions

Heart rate reduction improves clinical outcomes in HF. Beta-blocker dosage should be titrated to maintain a resting heart rate <75 beats/minute. In instances where patients are unable to tolerate increased doses of beta-blockers, ivabradine can be considered. Ivabradine inhibits the I_f channel in the sinus node and can be used to slow the heart rate in patients in sinus rhythm.[2]

Indications for device and surgical interventions are listed in Table 7.[2,23]

Conclusion

HF is a common condition that is caused by a diverse group of aetiologies, representing unique disease entities that require different management strategies. It is for this reason

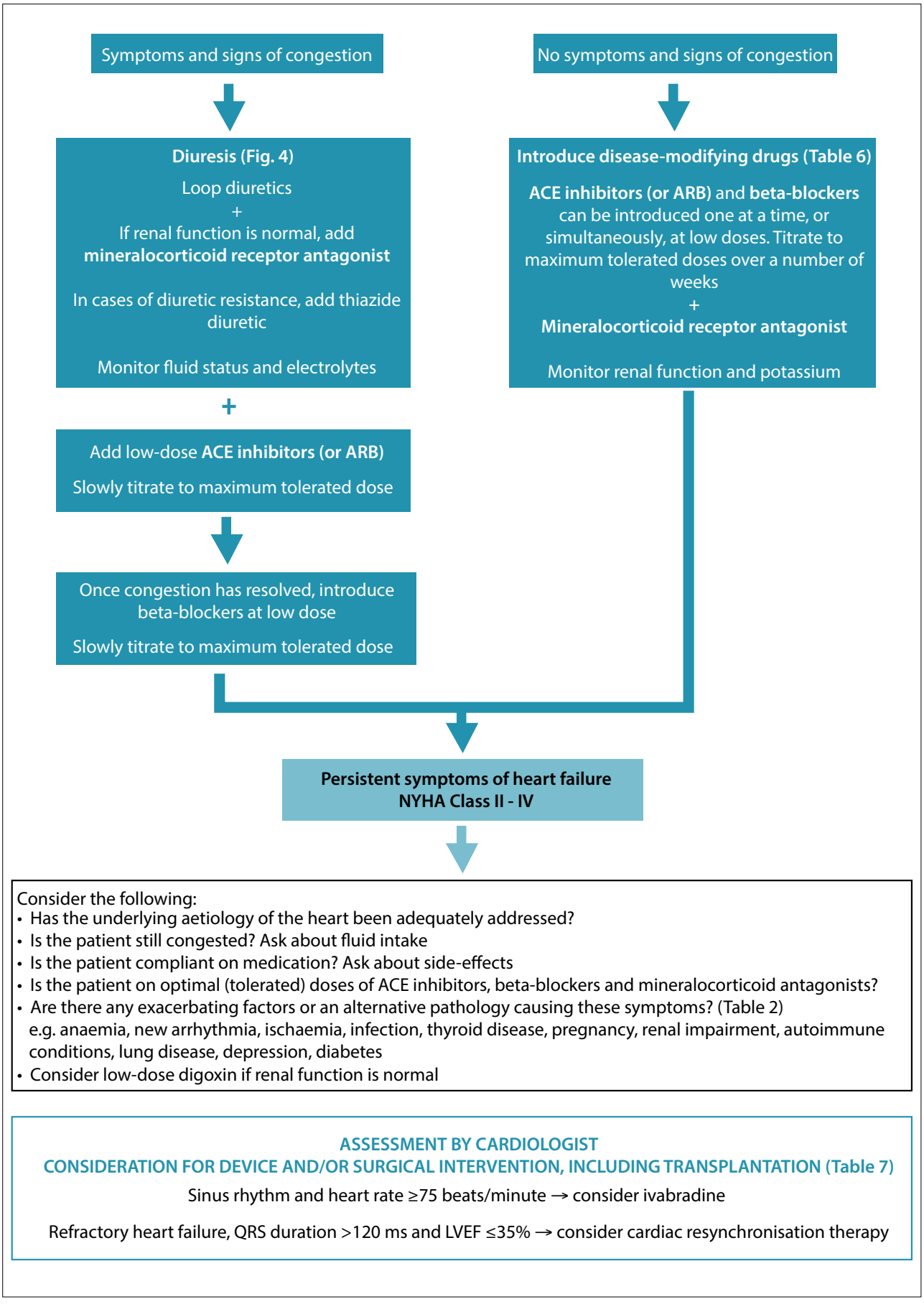

Fig. 3. Approach to management of heart failure (ARB = angiotensin receptor blocker).

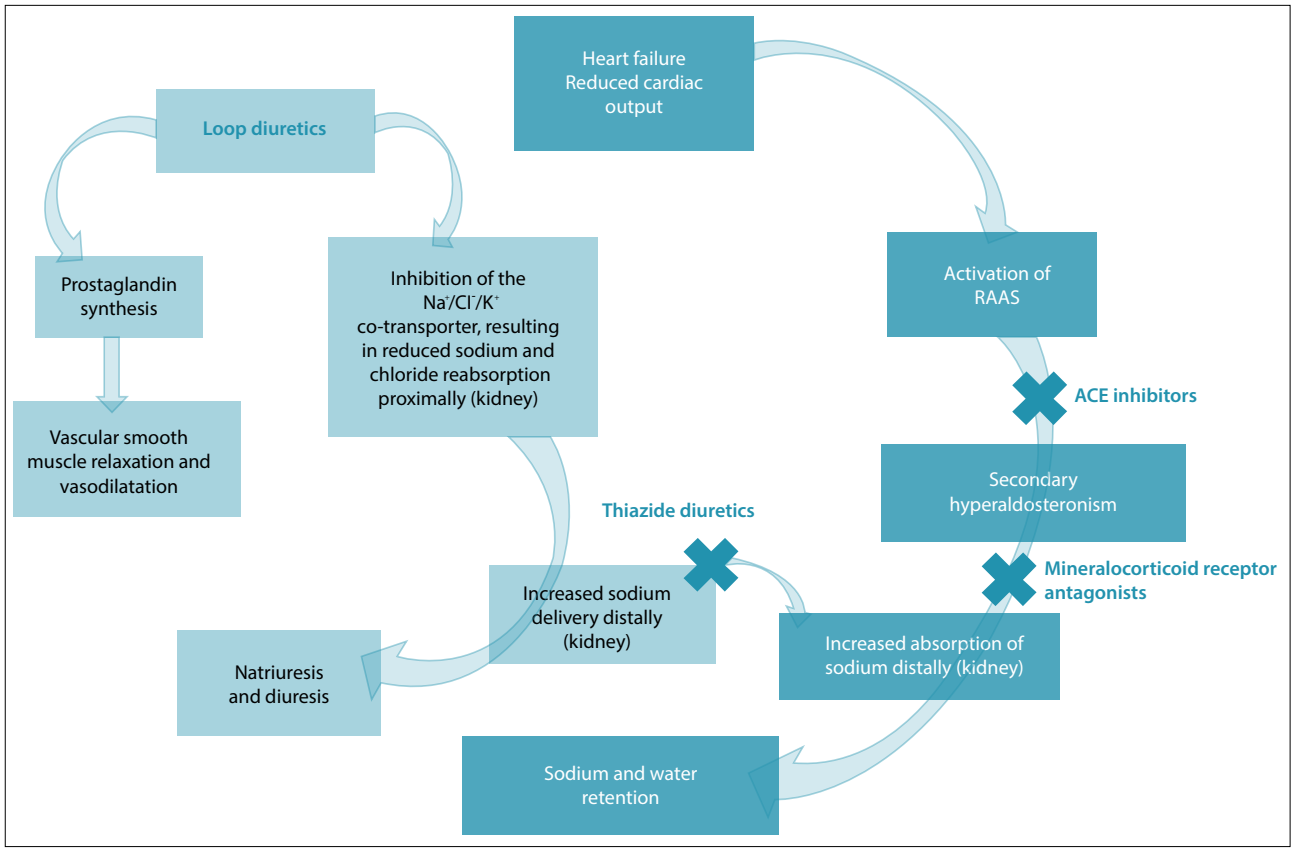

Fig. 4. Mechanisms of action of pharmacotherapy used in heart failure management.

Table 6. Disease-modifying drugs used in heart failure[2]

Drugs	Starting dose	Target dose	Recommendation	Level of evidence
ACE inhibitors				
Captopril	6.25 mg 3 × /day	50 mg 3 × /day	Recommended, in addition to beta-blockers, for all patients with LVEF ≤40% to reduce risk of HF hospitalisation and premature death	IA
Enalapril	2.5 mg 2 × /day	10 - 20 mg 2 × /day		
Lisinopril	2.5 - 5.0 mg daily	20 - 35 mg daily		
Perindopril	2.0 mg daily	4.0 mg daily		
Ramipril	2.5 mg daily	5.0 mg 2 × /day		
Trandolapril	0.5 mg daily	4.0 mg daily		
Beta-blockers				
Bisoprolol	1.25 mg daily	10 mg daily	Recommended, in addition to ACE inhibitors, for all patients with LVEF ≤40% to reduce risk of HF hospitalisation and premature death	IA
Carvedilol	3.125 mg 2 × /day	25 - 50 mg 2 × /day		
Metoprolol succinate	12.5 - 25 mg daily	200 mg daily		
Angiotensin II receptor blockers				
Candesartan	4 mg or 8 mg daily	32 mg daily	Recommended as an alternative to ACE inhibitors, in patients with LVEF ≤40% to reduce risk of HF, hospitalisation and premature death	IA
Valsartan	40 mg 2 × /day	160 mg 2 × /day		
Losartan	50 mg daily	150 mg daily		
Telmisartan	20 mg daily	80 mg daily		
Mineralocorticoid receptor antagonists				
Spirono-lactone	25 mg daily	25 - 50 mg daily	Recommended for all patients with persisting symptoms and an LVEF ≤35% despite treatment with an ACE inhibitor and a beta-blocker to reduce risk of HF hospitalisation and premature death	IA
Eplerenone	25 mg daily	50 mg daily		

that diagnostic certainty is as important as treating the clinical syndrome of HF. Effective diuresis and complete resolution of congestion is key in improving symptoms and functional capacity, reducing the need for recurrent hospitalisation, increasing the probability of establishing patients on good doses of disease-modifying drugs, and ultimately improving outcomes.

Table 7. Device and surgical interventions in heart failure[2,23]

Intervention	Indication
ICD	Secondary prevention
	Survivors of cardiac arrest or patients with sustained symptomatic ventricular tachycardia, irrespective of LVEF
	Good functional status
	Expected survival >1 year
	Primary prevention (limited availability in SSA)
	LVEF ≤35%
	Symptomatic (NYHA Class II - III) despite ≥3 months on optimal medical therapy
	Increased risk of SCD in conditions such as ARVC and HCM
CRT	Cardiac resynchronisation therapy should be considered in patients who fulfil the following criteria
	Typical LBBB (QRS ≥120 ms)
	Persistent symptoms despite ≥3 months of optimal medical therapy
	NYHA Class III/IV (ambulatory)
	LVEF ≤35% and an expected survival >1 year
	Right bundle branch block and prolonged PR interval are predictors of non-favourable outcomes
Mechanical circulatory support	Limited availability in SSA
	Drug-refractory acute circulatory collapse and at immediate risk of death
	To sustain life, as a bridge to decision/candidacy for transplantation or as a bridge to transplantation, recovery or destination therapy
Heart transplantation	End-stage heart failure with severe symptoms, a poor prognosis, and no remaining alternative treatment option
	Motivated, well-informed, and emotionally stable
	Capable of complying with the intense treatment required postoperatively
	Contraindications to transplantation
	Active infection, significant comorbidities (e.g. severe peripheral vascular disease, cerebrovascular disease, renal failure, liver disease, systemic multiorgan disease), recurrent thromboembolism, unhealed peptic ulcer, current alcohol or drug abuse, emotional/psychiatric instability, cancer within the previous 5 years
	High, fixed pulmonary vascular resistance (>4 - 5 Wood units and mean transpulmonary gradient >5 mmHg)

ICD = implantable cardioverter-defibrillator; CRT = cardiac resynchronisation therapy; SCD = sudden cardiac death; HCM = hypertrophic cardiomyopathy.

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