DIAGNOSIS AND TREATMENT OF HEART FAILURE IN THE COMMUNITY

Heart failure still remains a serious public health problem with significant morbidity and mortality, despite significant therapeutic interventions.

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The complexity of heart failure has led to the development of guidelines seeking to clarify or simplify diagnostic approaches and therapeutic interventions (American College of Cardiology (ACC)/American Heart Association (AHA) guidelines and European Society of Cardiology (ESC) — guidelines revised, as well as South African guidelines).[1-3]

Heart failure is a common disorder that affects 1 - 2% of the adult population and more than 10% of those aged more than 65 years. For the GP, the important factors in the management of patients with heart failure can be summarised as follows:

• Identification of patients at risk and institution of appropriate therapeutic and preventive measures.
• Identification of relevant symptoms and clinical features that lead to a diagnosis of heart failure (and being able to exclude other aetiological causes of congestion/fluid overload) and institution of basic, standard treatment where appropriate.
• Identification of clinical features that require further diagnostic imaging techniques.
• The ability to detect complications in those being followed up.

DEFINITION

There is no simple, objective definition of heart failure. The definition by Milton Packer is detailed and embodies all aspects of this complex syndrome: 'Heart failure represents a clinical syndrome characterized by abnormalities of left ventricular function and neurohormonal regulation which are accompanied by effort intolerance, fluid retention and reduced longevity.'[4]

AETIOLOGY

Numerous aetiological factors lead to the development of heart failure; hence the saying 'Many roads lead to a broken heart' (Fig. 1).

CLASSIFICATION

The current staging of heart failure takes cognisance of it as a progressive disease with identifiable risk factors (conditions) and serves to complement the New York Heart Association (NYHA) classification, which is based on the severity of symptoms (Tables I and II).

Risk factors (conditions)
The risk factors are as follows:

• hypertension
• coronary artery disease   } independently or in co-existence contribute to majority of heart failure cases

• diabetes mellitus
• valvular heart disease
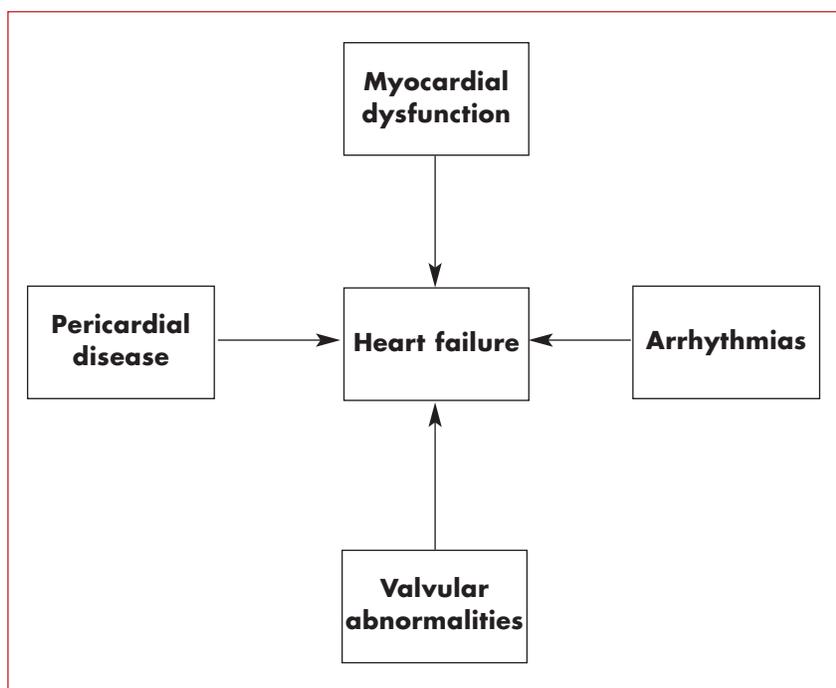• idiopathic dilated cardiomyopathy.

Fig. 1. Heart failure — broad aetiological factors.

GPs can play a significant role in the identification of patients at risk of developing heart failure as they are often the first to see them, particularly those with hypertension and diabetes. Appropriate therapeutic/preventive measures may prevent, retard or delay progression to heart failure.

DIAGNOSIS

The diagnosis of heart failure relies on clinical judgement based on history, physical examination and appropriate, relevant cost-effective investigations.

The ESC heart failure diagnostic guidelines and the Framingham diagnostic criteria for heart failure are useful tools in the diagnosis (Tables III and IV).

Once a diagnosis of heart failure is made, the underlying aetiology should be identified and appropriate treatment of correctable or precipitating factors instituted.

Symptoms
Dyspnoea
Exertional dyspnoea is the earliest and commonest symptom; however, it is neither specific nor restricted to heart failure. Other forms of dyspnoea, i.e. paroxysmal nocturnal dyspnoea and orthopnoea, usually indicate advanced stages of heart failure (however, these are also not specific for heart failure).

Fatigue
Fatigue is nonspecific and non-diagnostic, but may be useful when interpreted in the context of other symptoms.

Poor effort tolerance
Poor effect tolerance is also nonspecific.

Ankle oedema
This is a common presenting feature, but it is nonspecific and becomes helpful in the diagnosis of heart failure in men rather than in women.

Clinical findings
The clinical signs are caused by congestion and can be specific and nonspecific.[5]

Specific clinical findings
• Elevated jugular venous pressure.
• Gallop rhythm.
• Displaced apical impulse.

The above signs occur in severe heart failure, which reduces their sensitivity in the detection of earlier stages of heart failure.

Nonspecific signs
• Tachycardia.
• Pulmonary crackles.
• Peripheral oedema.

Comment: None of the above symptoms and physical signs (even the specific ones) in isolation can be predictive of heart failure.

Once a diagnosis of heart failure is made the severity must be determined, for which the NYHA functional classification can be used.

Investigations
The clinical suspicion of heart failure should be followed by supportive diagnostic investigations.

Basic investigations
Urine analysis. This is a basic test done on site by the GP and may give clues to underlying renal problems (which may result in fluid overload) and presence of diabetes.

Basic haematology and biochemistry tests. The following tests are recommended as part of a routine diagnostic evaluation of heart failure:

Haematology
• full blood count
  • haemoglobin
  • platelets
  • white cell count.

Biochemistry
• serum glucose
• serum creatinine
• urea and electrolytes
• hepatic function.

Additional tests may be required. These will depend on whether there is a clinical suspicion of the underlying cause, e.g. cardiac markers in the context of myocardial infarction.

Results from the above basic investigations may be extremely useful in determining other aetiological factors with regard to clinical presentation or precipitating factors (Table V).

B-type natriuretic peptide is becoming important as a useful screening tool in

Table I. **Stages of heart failure (HF)**

	Description	Examples
A	Patients at high risk of developing HF because of the presence of conditions that are strongly associated with its development. Such patients have no identified structural or functional abnormalities of the pericardium, myocardium or cardiac valves and have never shown signs or symptoms of HF	Systemic hypertension, coronary artery disease, diabetes mellitus, history of cardiotoxic drug therapy or alcohol abuse, personal history of rheumatic fever, family history of cardiomyopathy
B	Patients who have developed structural heart disease that is strongly associated with the development of HF but who have never shown signs or symptoms of HF	Left ventricular hypertrophy or fibrosis, left ventricular dilatation or hypocontractility, asymptomatic valvular heart disease, previous myocardial infarction
C	Patients who have current or prior symptoms of HF associated with underlying structural heart disease	Dyspnoea or fatigue due to left ventricular systolic dysfunction, asymptomatic patients undergoing treatment for prior symptoms of HF
D	Patients with advanced structural heart disease and marked symptoms of HF at rest, despite maximal medical therapy, and who require specialised interventions	Patients who are frequently hospitalised for HF or cannot safely be discharged from hospital; patients in hospital awaiting heart transplantation; patients at home receiving continuous intravenous support for symptom relief or being supported with a mechanical circulatory assist device; patients in a hospice setting for the management of HF

Adapted from ACC/AHA guidelines.[1]

Table II. **New York Heart Association classification of heart failure**

Class I	No limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations
Class II	Slight limitation of physical activity: comfortable at rest but ordinary activities result in fatigue, palpitations or dyspnoea
Class III	Marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms
Class IV	Unable to carry out any physical activity without discomfort: symptoms of heart failure are present even at rest, with increased discomfort with any physical activity

Table III. **European Society of Cardiology guidelines for the diagnosis of heart failure**

Essential features

Symptoms of heart failure, e.g breathlessness, fatigue, ankle oedema
Objective evidence of cardiac dysfunction (at rest)

Non-essential features

In cases where the diagnosis is in doubt, there is a response to treatment directed towards heart failure

Adapted from *Heart* 2000; **84:** 334-338.[5]

Table IV. **Framingham criteria for congestive heart failure**

Major criteria	Paroxysmal nocturnal dyspnoea
	Neck vein distension
	Crackles
	Radiographic cardiomegaly
	Acute pulmonary oedema
	S_3 gallop
	Central venous pressure >16 cm H_2O
	Circulation time 25 s
	Hepatojugular reflux
	Pulmonary oedema, visceral congestion, or cardiomegaly at outopsy
	Weight loss — 4.5 kg in 5 days in response to treatment of congestive heart failure
Minor criteria	Bilateral ankle oedema
	Nocturnal cough
	Dyspnoea on ordinary exertion
	Hepatomegaly
	Pleural effusion
	Decrease in vital capacity by one-third from maximal value recorded
	Tachycardia (120 beats/min)

In this study the diagnosis of congestive heart failure requires that two major or one major and two minor criteria be present concurrently. Minor criteria were acceptable only if they could not be attributed to another medical condition.

Adapted from Braunwald E. *Heart Disease — A Textbook of Cardiovascular Medicine.* 6th ed.[4]

heart failure. However, its role as part of routine, clinical assessment in general practice is not yet supported by studies.

Once such studies are available and positive, the cost-effectiveness of this screening tool will have to be determined.

Electrocardiogram (ECG)

An ECG is a very useful, cheap diagnostic tool that is available on site and should be performed routinely in all patients with a clinical suspicion of heart failure.

Abnormal ECG findings may reflect the following:
- arrhythmias — predominantly atrial, but occasionally ventricular
- signs of previous myocardial infarction and/or ischaemia
- features of right ventricular enlargement or hypertrophy
- features of left ventricular hypertrophy

A normal ECG is unusual in heart failure and should suggest a review of the diagnosis.[2,3]

Chest radiograph

Chest radiography is a relatively cheap, easily accessible, important, supportive diagnostic tool that should be performed in all patients with a clinical suspicion of heart failure. The following three factors need to be evaluated:
- Size and shape of the cardiac shadow (this can provide important

information concerning the exact nature of the underlying heart disease).

- Distribution of the pulmonary vascular markings and fluid in the interstitial space.
- Fluid in the pleural/subpleural space (Table VI).

Transthoracic echocardiography

This is one of the imaging methods used objectively to assess ventricular function. It is also extremely useful in identifying primary valvular abnormalities and pericardial disease as aetiological factors.

Ideally *all* patients with clinical evidence of heart failure should have echocardiographic examination,

specifically to assess the abovementioned structural changes as part of

their initial assessment. However, in view of lack of expertise at a primary care physician level (GP level), coupled with cost implication, routine echocardiography is not feasible. The question that arises is who should then be referred for this important, non-invasive examination in the initial assessment of heart failure?

The South African guidelines[3] suggest:

- obvious clinical diagnosis of a heart murmur.
- clinical evidence of organic valvular heart disease, ventricular hypertrophy, pericardial disease and restrictive cardiomyopathy
- uncertain clinical diagnosis and inconclusive radiological findings.

TREATMENT

The syndrome of heart failure requires multiple strategies — from prevention to advanced mechanical therapeutic interventions.

Prevention

Risk factors (conditions) (outlined above) predisposing to the development of ventricular dysfunction and subsequent heart failure must be treated.

Non-pharmacological measures

These include:
- dietary measures (reduction of salt intake, moderate alcohol intake, fluid restriction when necessary)
- appropriate weight loss programme if overweight

Table V. **Haematology and biochemistry**

Haemoglobin: Estimation, mean cell volume, white cell count

Aetiology: Anaemia that is severe enough (Hct < 25%) can produce signs and symptoms of congestive heart failure (CHF) without underlying cardiac abnormalities

Differential diagnosis: Polycythaemia and a raised haematocrit suggest that breathlessness may be caused by lung disease, cyanotic heart disease or a pulmonary arteriovenous malformation

Precipitating factor: Haematocrit between 25% and 35% may aggravate underlying cardiac disease. A raised white cell count may prompt investigation to exclude infection

Urea and creatinine
Differential diagnosis: Renal insufficiency or failure may cause fluid overload mimicking heart failure, especially if severe hypertension is the cause of the renal dysfunction. Untreated heart failure is rarely associated with the major electrolyte disturbances found in renal failure

Precipitating factor: Worsening renal function either due to the natural progression of the heart disease or to a side-effect of non-cardiac medication (especially NSAIDs); cardiac medication (over-diuresis together with ACE) may precipitate a worsening in the cardiac status

Liver function tests
Aetiology: An increase in aspartate transminase (AST) greater than that in alanine aminotransferase (ALT) increased gamma glutamyl transferase (GT) and alkaline phosphatase (ALP) may be found with alcohol abuse. A congested, ischaemic liver may show a transaminitis (ALT, AST)

Differential diagnosis: Severe primary liver disease with hypoalbuminaemia, and deranged enzymes may present with a congested state not dissimilar to CHF

Precipitating factor: Drug-related liver toxicity (e.g. amiodarone) may precipitate clinical deterioration

Adapted from *S Afr Med J* 1998; **88** (9): part-2.[3]

Table VI. **Chest radiographic findings**

Syndrome: A normal cardiothoracic ratio on the posteroanterior radiograph and a normal left ventricular size on the lateral chest radiograph make the diagnosis of CHF unlikely, urging a careful review of the clinical evidence
Cardiomegaly together with pulmonary venous congestion in symptomatic patients is highly suggestive of CHF, although reliable interpretation of pulmonary congestion varies
The chest radiograph should never be reviewed in isolation, as congestion of renal or neurogenic origin may be indistinguishable from cardiac congestion

Aetiology/differential diagnosis: The chest radiograph is also useful in helping to exclude other diagnoses
Pulmonary disease, pericardial calcification, calcified heart valves, left atrial enlargement associated with mitral stenosis and isolated right ventricular enlargement would all support diagnoses other than pure, chronic left ventricular systolic dysfunction

Precipitating factor(s): The chest radiograph helps to diagnose a correctable precipitating factor/s — a pneumonia, or pulmonary infarct(s) that can precipitate the episode of clinical heart failure may be identified

Adapted from *S Afr Med J* September 1998; **88** (9): part 2.[3]

- regular exercise programme
- appropriate counselling on compliance and other issues (family should be involved in the counselling programme).

GPs can play a significant role in the identification of patients at risk of developing heart failure as they are often the first to see them, particulary those with hypertension and diabetes. Appropriate therapeutic/preventive measures may prevent, retard or delay progression to heart failure.

Once a diagnosis of heart failure is made, the underlying aetiology should be identified and appropriate treatment of correctable or precipitating factors instituted.

Pharmacological strategies

The goal of this treatment strategy is to relieve symptoms and improve life expectancy; hence the use of multi-drug therapy.

Symptomatic treatment

Diuretic treatment (loop diuretics) is useful in patients with fluid retention. Improvement in symptoms is noted within a few hours to days. Therapy should be initiated with low doses and titrated up.
Furosemide is a commonly used diuretic and is extremely cheap. Volume and electrolyte depletion (especially K+) are common complications of chronic diuretic therapy. Diuretics are used in combination with other therapeutic agents as maintenance treatment.
Digoxin improves symptoms and exercise tolerance in patients treated with diuretics, ACE inhibitors and β–blockers. It is also useful in heart failure patients with atrial fibrillation. Lower doses (i.e. 0.125 mg) are suggested for use in heart failure to reduce the toxic side-effects profile, especially in the context of hypokalaemia and hypomagnesaemia. Major side-effects are gastrointestinal, neurological and conduction disturbances.

Therapies that improve survival

ACE inhibitors. Clinical trials have shown that ACE inhibitor therapy significantly improves survival in addition to symptomatic improvement throughout the different stages of heart failure. Current guidelines recommend their use as first-line therapy in conjunction with diuretics. Therapy should be initiated at low doses and titrated to maximum appropriate dosages (as used in the clinical trials) (Table VII). Availability of generic ACE inhibitors has further reduced the cost of this important therapeutic intervention in heart failure.
Renal function and potassium levels should be assessed within 1 - 2 weeks of starting treatment. Important side-effects are the following:
- angio-oedema
- hyperkalaemia
- renal insufficiency.
ACE inhibitor therapy is contraindicated in renal artery stenosis, pregnancy and documented angio-oedema in cases of previous exposure to ACE inhibitors.

Table VII. **ACE inhibitor dosages — from clinical trials**

Agent	Starting dosage	Target dosage	Trial
Captopril	6.25 mg tds	25 - 50 mg tds	SAVE
Enalapril	5 mg bd	10 mg bd	SOLVD P/T
Lisinopril	2.5 mg daily	40 mg daily	ATLAS
Ramipril	2.5 mg bd	5 mg bd	AIRE

AIRE = Acute Infarction Ramipril Efficacy; ATLAS = Assessment of Treatment with Lisinopril and Survival; SAVE = Survival and Ventricular Enlargement; SOLVD P/T = Studies of Left Ventricular Dysfunction (Prevention/Treatment).

Adapted from *American Journal of Cardiology* May 2003, Supplement on heart failure. A symposium: from prevention to management of chronic heart failure.

Table VIII. **Dosage of β-blockers used in heart failure trials**

Agent	Starting dosage	Target dosage	Trial
Bisoprolol	1.25 mg daily	10 mg daily	CIBIS II
Carvedilol	3.125 mg bd	25/50 mg bd	US carvedilol heart failure studies COPERNICUS
Metoprolol CR/XL	12.5/25 mg daily	220 mg daily	MERIT-HF

CIBIS II = Cardiac Insufficiency Bisoprolol Study II; COPERNICUS = Carvedilol Prospective Randomised Cumulative Survival Trial; MERIT-HF = Metoprolol CR/XL Randomised Intervention Trial in Congestive Heart Failure.

Adapted from: *American Journal of Cardiology* May 2003, Supplement on heart failure. A symposium: from prevention to management of chronic heart failure.

Table IX. **Pharmacological therapy of symptomatic chronic heart failure due to systolic left ventricular dysfunction**

	For symptoms	For survival morbidity	For symptoms if intolerance to ACE inhibitor or beta-blockade
NYHA I	Reduce/stop diuretic	Continue ACE inhibitor if asymptomatic. Add beta-blocker if post-myocardial infarction	
NYHA II	=/- diuretic, depending on fluid retention	ACE inhibitor as first-line treatment	Angiotensin II receptor antagonist (ARB) if ACE inhibitor intolerant
		Add beta-blocker if still symptomatic	or ACE inhibitor + ARB if beta-blocker intolerant
NYHA III	+ diuretics + digitalis if still symptomatic + nitrates/hydralazine if tolerated	ACE inhibitor and beta-blockade, add spironolactone	ARB if ACE inhibitor intolerant or ACE inhibitor + ARB if beta-blocker intolerant
NYHA IV	Diuretics + digitalis + nitrates/hydralazine if tolerated + temporary inotropic support	ACE inhibitor beta-blockade spironolactone	ARB if ACE inhibitor intolerant or ACE inhibitor + ARB if beta-blocker intolerant

Adapted from the Task force report: ESC: Guidelines for the diagnosis and treatment of heart failure. *Eur Heart J* 2001; **22:** 1527-1560.[2]

Table X. **Factors affecting survival in patients with heart failure**

Clinical

Male gender
Coronary artery disease aetiology
NYHA class
Exercise capacity
Heart rate at rest
Systolic arterial pressure
Pulse pressure
S_3 gallop
Cheyne-Stokes respiration
Cardiac cachexia

Haemodynamic

Left ventricular ejection fraction
Right ventricular ejection fraction
Left ventricular stroke work index
Left ventricular filling pressure
Right arterial pressure
Left ventricular systolic pressure
Mean arterial pressure
Cardiac index
Exercise cardiac output or stroke work index
Systemic vascular resistance

Biochemical

Plasma norepinephrine
Plasma renin
Plasma arginine vasopressin
Plasma atrial and brain natriuretic peptides
Plasma endothelin-1
Plasma interleukin-6
Serum sodium
Serum potassium and total potassium stores
Serum magnesium

Electrophysiological

Frequent ventricular extrasystoles
Non-sustained ventricular tachycardia
Ventricular tachycardia
Atrial fibrillation

Adapted from Braunwald *E. Heart Disease — A Textbook of Cardiovascular Medicine.* 6th ed.[4]

Beta-blockers. Clinical trials have shown β-blockers to reduce mortality and hospitalisation in heart failure. β-blocker treatment should be initiated in patients whose heart failure has been clinically stable for 2 - 4 weeks, with no evidence of acute decompensation or fluid overload with standard care.[6]

Initiation of β-blocker therapy may worsen myocardial performance at the start of treatment; hence the recommendation that the starting dose should be very low and then titrated up. For the suggested dosage see Table VIII.

It is advisable to use the β-blockers used in clinical trials.

Although β-blocker therapy is still relatively expensive compared with other standard drugs used in heart failure, its benefit outweighs the cost, which makes this therapy important for use in heart failure.

Before initiating therapy the patient should be screened for the following contraindications:

- symptomatic bradycardia or hypotension
- 2nd and 3rd degree AV block
- bronchial asthma.

Other therapies
Spironolactone. There is a strong suggestion that this reduces mortality and is useful with other therapeutic agents in severe to advanced heart failure.

Nitrates and hydralazine combination. There is no specific recommendation for the standard use of this combination in clinical practice, except in patients intolerant of ACE inhibitors.

Angiotensin II receptor antagonists. More data are required for their use in heart failure. The current cost of these drugs makes them prohibitive for widespread use in heart failure at a community level.

Mechanical and electrical therapeutic strategies will not be discussed here, as they are beyond the scope of this article.

It is important to realise that patients being treated for heart failure are likely to be on other drug regimens for treatment of concomitant medical conditions, and compliance may become a problem. Appropriate counselling on adherence to therapy is extremely important if therapeutic benefits are to be seen.

Table IX shows suggested therapies according to the severity of symptoms.

PROGNOSIS

Heart failure accounts for a substantial portion of all cardiovascular deaths. Fifty per cent of patients diagnosed with heart failure die within 5 years. Numerous factors have been found to correlate with mortality (Table X).

WHEN TO REFER

Patients must be referred in the following cases:
- clinical suspicion of underlying valvular heart disease
- severe heart failure
- clinical evidence of left ventricular hypertrophy
- clinical suspicion of coronary artery disease
- stability followed by clinical deterioration not explained by non-compliance.

References available on request.

IN A NUTSHELL

The mortality and morbidity of heart failure remain high despite major therapeutic advances in the last two decades, partly because of inadequate health care service programmes and structure as well as the under-use of proven effective treatment (β-blockers and ACE inhibitors).

The new classification which uses the staging system is extremely useful in making it possible to identify the early stages (stages A and B) of heart failure, where appropriate therapeutic intervention can prevent or delay the progression to symptomatic heart failure with irreversible left ventricular dysfunction.

A systematic approach should be followed in the diagnosis of heart failure, taking into account the symptomatology, clinical findings and appropriate cost-effective diagnostic procedures.

The GP can play a significant role by early identification of patients at risk, modification of the predisposing risk factors as well as early, appropriate referral of those who may have correctable aetological factors.

SINGLE SUTURE

EVOLVED TO EAT MEAT

Contrary to what my beloved (now deceased) godmother, a convinced vegetarian, always used to tell me, humans have evolved to eat meat. According to a study of our ancestors' teeth, we evolved into meat-eaters around 2.5 million years ago. Analysis by an anthropologist at the University of Arkansas, showed that the first member of *Homo* had much sharper teeth than their most likely ancestor, *Australopithecus afarensis* — Lucy's species. Meat eating needs teeth which can cut rather than grind, which is determined by the slope of the cusps — they must be sharply sloped. Early *Homo* teeth have these sharply sloped cusps, steeper than those of gorillas that don't eat meat.

(Holtzman D. *New Scientist* 2003; 6 September, p.19.)