LG SC: ESC

LG BPCO: GOLD

COMORBILITA’

Table 9 Practical guidance on using β-adrenergic blockers in heart failure (modified from Ref. 133)

Who should receive β-blocker therapy
- All patients with chronic, stable heart failure
- Without contraindications (symptomatic hypotension or bradicardia, asthma)

What to promise
Treatment is primarily prophylactic against death and new hospitalisations for cardiovascular reasons. Some patients will experience improvement of symptoms.

When to start
- No physical evidence of fluid retention (use diuretics accordingly)
- Start ACE-I first if not contraindicated
- In stable patients, in the hospital or in outpatient clinics
- NYHA class IV/severe CHF patients should be referred for specialist advice
- Review treatment. Avoid verapamil, diltiazem, antiarrhythmics, non-steroidal anti-inflamatory drugs

Beta-blocker
- Bisoprolol, carvedilol or metoprolol

Dose
- Start with a low dose
- Increase dose slowly. Double dose at not less than 2 weekly intervals
- Aim for target dose (see above) or, if not tolerated, the highest tolerated dose

	Starting dose mg	**Target dose mg**
Bisoprolol	1.25 once daily	10 once daily
Carvedilol	3.125 twice daily	25—50 twice daily
Metoprolol CR/XL	12.5—25 once daily	200 once daily

Monitoring
- Monitor for evidence of heart failure symptoms, fluid retention, hypotension and bradycardia
- Instruct patients to weigh themselves daily and to increase their diuretic dose if weight increases

Problem solving
- Reduce/discontinue β-blocker only if other actions were ineffective to control symptoms/secondary effects
- Always consider the reintroduction and/or uptitration of the β-blocker when the patient becomes stable
- Seek specialist advice if in doubt.

Symptomatic hypotension (dizziness, light headedness and/or confusion)
- Reconsider need for nitrates, calcium channel blockers and other vasodilators
- If no signs/symptoms of congestion consider reducing diuretic dose

Worsening symptoms/signs (increasing dyspnoea, fatigue, oedema, weight gain)
- Double dose of diuretic or/and ACE-I.
- Temporarily reduce the dose of β-blockers if increasing diuretic dose does not work
- Review patient in 1—2 weeks; if not improved seek specialist advice
- If serious deterioration halve dose of β-blocker
- Stop β-blocker (rarely necessary; seek specialist advice)

Bradycardia
- ECG to exclude heart block
- Consider pacemaker support if severe bradycardia or AV block or sick sinus node early after starting β-blockers
- Review need, reduce or discontinue other heart rate slowing drugs, e.g., digoxin, amiodarone, diltiazem
- Reduce dose of β-blocker. Discontinuation rarely necessary

Severe decompensated heart failure, pulmonary oedema, shock
- Admit patient to hospital
- Discontinue β-blocker if inotropic support is needed or symptomatic hypotension/bradycardia is observed
- If inotropic support is needed, levosimendan may be preferred

CHF: Congestive Heart Failure; NYHC: New York Heart Association.

ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2008‡

European Heart Journal (2008) **29**, 2388–2442

ESC GUIDELINES

HF is a syndrome in which the patients should have the following features: symptoms of HF, typically shortness of breath at rest or during exertion, and/or fatigue; signs of fluid retention such as pulmonary congestion or ankle swelling; and objective evidence of an abnormality of the structure or function of the heart at rest (*Table 3*). A clinical response to treatment directed at HF alone is not sufficient for the diagnosis, but is helpful when the diagnosis remains unclear after appropriate diagnostic investigations. Patients with HF would usually be expected to show some improvement in symptoms and signs in response to those treatments from which a relatively fast symptomatic improvement could be anticipated (e.g. diuretic or vasodilator administration). The major and common clinical manifestations of HF are shown in *Table 4*.

Table 3 Definition of heart failure

Heart failure is a clinical syndrome in which patients have the following features:

- **Symptoms typical of heart failure**
  (breathlessness at rest or on exercise, fatigue, tiredness, ankle swelling)

  and

- **Signs typical of heart failure**
  (tachycardia, tachypnoea, pulmonary rales, pleural effusion, raised jugular venous pressure, peripheral oedema, hepatomegaly)

  and

- **Objective evidence of a structural or functional abnormality of the heart at rest**
  (cardiomegaly, third heart sound, cardiac murmurs, abnormality on the echocardiogram, raised natriuretic peptide concentration)

Table 4 Common clinical manifestations of heart failure

Dominant clinical feature	Symptoms	Signs
Peripheral oedema/congestion	Breathlessness Tiredness, fatigue Anorexia	Peripheral oedema Raised jugular venous pressure Pulmonary oedema Hepatomegaly, ascites Fluid overload (congestion) Cachexia
Pulmonary oedema	Severe breathlessness at rest	Crackles or rales over lungs, effusion Tachycardia, tachypnoea
Cardiogenic shock (low output syndromes)	Confusion Weakness Cold periphery	Poor peripheral perfusion SBP <90 mmHg Anuria or oliguria
High blood pressure (hypertensive heart failure)	Breathlessness	Usually raised BP, LV hypertrophy, and preserved EF
Right heart failure	Breathlessness Fatigue	Evidence of RV dysfunction Raised JVP, peripheral oedema, hepatomegaly, gut congestion

Descriptive terms in heart failure

Acute and chronic heart failure

use words with a slightly different meaning. The word 'acute' in the context of acute HF has become confusing because some clinicians use the word to indicate severity (the medical emergency of life-threatening pulmonary oedema) and others use the word to indicate decompensated, recent-onset, or even new-onset HF.[4] The word is then an indicator of time rather than severity. The

Table 5 Classification of heart failure

• **New onset**	First presentation Acute or slow onset
• **Transient**	Recurrent or episodic
• **Chronic**	Persistent Stable, worsening, or decompensated

Descriptive terms in heart failure

Systolic vs. diastolic heart failure

A distinction is frequently made between systolic and diastolic HF.[12,13] The distinction is somewhat arbitrary.[14–16] Patients with diastolic HF have symptoms and/or signs of HF and a preserved left ventricular ejection fraction (LVEF) >40–50%.[17] There is no consensus concerning the cut-off for preserved EF. The EF is the stroke volume divided by the end-diastolic volume for the relevant ventricular chamber of the heart and is therefore largely determined by the end-diastolic volume of the ventricular chamber (i.e. a dilated heart). An EF below or above 40%, distinguishes between large or normal left end-diastolic ventricular volumes. The distinction has arisen largely because in the past most patients admitted to hospitals for investigation or entered into clinical trials have had dilated hearts with a reduced EF <35 or 40%. Most patients with HF have evidence of both systolic and diastolic dysfunction at rest or on exercise. Diastolic and systolic HFs should not be considered as separate entities.[18] Other phrases have been used to describe diastolic HF, such as HF with preserved ejection fraction (HFPEF), HF with normal ejection fraction (HFNEF), or HF with preserved systolic function (HFPSF). We have elected to use the abbreviation HFPEF in this document.

Table 6 Classification of heart failure by structural abnormality (ACC/AHA), or by symptoms relating to functional capacity (NYHA)

ACC/AHA stages of heart failure		NYHA functional classification	
Stage of heart failure based on structure and damage to heart muscle		Severity based on symptoms and physical activity	
Stage A	At high risk for developing heart failure. No identified structural or functional abnormality; no signs or symptoms.	**Class I**	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnoea.
Stage B	Developed structural heart disease that is strongly associated with the development of heart failure, but without signs or symptoms.	**Class II**	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnoea.
Stage C	Symptomatic heart failure associated with underlying structural heart disease.	**Class III**	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity results in fatigue, palpitation, or dyspnoea.
Stage D	Advanced structural heart disease and marked symptoms of heart failure at rest despite maximal medical therapy.	**Class IV**	Unable to carry on any physical activity without discomfort. Symptoms at rest. If any physical activity is undertaken, discomfort is increased.

ACC = American College of Cardiology; AHA = American Heart Association. Hunt SA *et al. Circulation* 2005;**112**:1825–1852.
The Criteria Committee of the New York Heart Association. Nomenclature and Criteria for Diagnosis of Diseases of the Heart and Great Vessels. 9th ed. Little Brown & Co; 1994. pp 253–256.

Epidemiology

Much is now known about the epidemiology of HF.[23–27] The ESC represents countries with a population of >900 million, and there are at least 15 million patients with HF in those 51 countries. The prevalence of asymptomatic ventricular dysfunction is similar, so that HF or asymptomatic ventricular dysfunction is evident in ~4% of the population. The prevalence of HF is between 2 and 3% and rises sharply at ~75 years of age, so the prevalence in 70- to 80-year-old people is between 10 and 20%. In younger age groups HF is more common in men because the most common cause, coronary heart disease, occurs in earlier decades. In the elderly, the prevalence is equal between the sexes.

modern treatment.[28,30–32] The mean age of patients with HF in the community in developed countries is 75 years. HFPEF is more common in the elderly, women, and those with hypertension or diabetes. HF is the cause of 5% of acute hospital admissions, is present in 10% of patients in hospital beds, and accounts for ~2% of national expenditure on health, mostly due to the cost of hospital admissions.[33] Substantial under-reporting is probably due to clinicians' preference for aetiological diagnoses (e.g. aortic stenosis) or the diagnosis of a major co-morbidity (e.g. diabetes).

The outlook is, in general, gloomy, although some patients can live for many years.[23,29,34,35] Overall 50% of patients are dead at 4 years. Forty per cent of patients admitted to hospital with HF are dead or readmitted within 1 year.

Studies show that the accuracy of diagnosis of HF by clinical means alone is often inadequate, particularly in women, the elderly, and the obese.[36,37] HFPEF (EF >45–50%) is present in half the patients with HF. The prognosis in more recent studies has been shown to be essentially similar to that of systolic HF.[38,39]

Aetiology of heart failure

Table 7 Common causes of heart failure due to disease of heart muscle (myocardial disease)

Coronary heart disease	Many manifestations
Hypertension	Often associated with left ventricular hypertrophy and preserved ejection fraction
Cardiomyopathies*	Familial/genetic or non-familial/non-genetic (including acquired, e.g. myocarditis)
	Hypertrophic (HCM), dilated (DCM), restrictive (RCM), arrhythmogenic right ventricular (ARVC), unclassified
Drugs	β-Blockers, calcium antagonists, antiarrhythmics, cytotoxic agents
Toxins	Alcohol, medication, cocaine, trace elements (mercury, cobalt, arsenic)
Endocrine	Diabetes mellitus, hypo/hyperthyroidism, Cushing syndrome, adrenal insufficiency, excessive growth hormone, phaeochromocytoma
Nutritional	Deficiency of thiamine, selenium, carnitine. Obesity, cachexia
Infiltrative	Sarcoidosis, amyloidosis, haemochromatosis, connective tissue disease
Others	Chagas' disease, HIV infection, peripartum cardiomyopathy, end-stage renal failure

Diagnosis of heart failure

Symptoms and signs of heart failure

The symptoms and signs of HF are the key to early detection because that is what causes patients to seek medical attention. Taking a good history and careful physical examination are skills, which are essential to master (*Table 8*). Breathlessness, tiredness, and fatigue are the characteristic symptoms, but eliciting and assessing these symptoms particularly in the elderly requires experience and skill.[44–46] The

Table 8 Key features of the clinical history in patients with heart failure

Symptoms	Breathlessness	(orthopnoea, paroxysmal nocturnal dyspnoea)
	Fatigue	(tiredness, exhaustion)
	Angina, palpitations, syncope	
Cardiovascular events	Coronary heart disease	
	Myocardial infarction	Thrombolysis
	Intervention	PCI
	Other surgery	CABG
	Stroke or peripheral vascular disease	
	Valvular disease or dysfunction	
Risk profile	Family history, smoking, hyperlipidaemia, hypertension, diabetes	
Response to current and previous therapy		

Table 9 Key features of the clinical examination in patients with heart failure

Appearance	Alertness, nutritional status, weight
Pulse	Rate, rhythm, and character
Blood pressure	Systolic, diastolic, pulse pressure
Fluid overload	Jugular venous pressure Peripheral oedema (ankles and sacrum) hepatomegaly, ascites
Lungs	Respiratory rate Rales Pleural effusion
Heart	Apex displacement Gallop rhythm, third heart sound Murmurs suggesting valvular dysfunction

Symptoms and severity of heart failure

There is a poor relationship between symptoms and the severity of cardiac dysfunction. Symptoms do relate more closely to prognosis if persistent after therapy and can then be used to classify the severity of HF and to monitor the effects of therapy. However, symptoms alone should not guide the optimal titration of neurohormonal inhibitors such as angiotensin-converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARBs), β-blockers, or aldosterone antagonists, because these drugs impact on mortality in a manner that is not closely related to symptoms. Patients should be titrated to the optimal, tolerated dose.

Table 10 Two classifications of the severity of heart failure in the context of acute myocardial infarction

Killip classification		Forrester classification
Designed to provide a clinical estimate of the severity of circulatory derangement in the treatment of acute myocardial infarction.		Designed to describe clinical and haemodynamic status in acute myocardial infarction.
Stage I	No heart failure. No clinical signs of cardiac decompensation	1. Normal perfusion and pulmonary wedge pressure (PCWP—estimate of left atrial pressure)
Stage II	Heart failure. Diagnostic criteria include rales, S3 gallop, and pulmonary venous hypertension. Pulmonary congestion with wet rales in the lower half of the lung fields.	2. Poor perfusion and low PCWP (hypovolaemic) 3. Near normal perfusion and high PCWP (pulmonary oedema)
Stage III	Severe heart failure. Frank pulmonary oedema with rales throughout the lung fields	4. Poor perfusion and high PCWP (cardiogenic shock)
Stage IV	Cardiogenic shock. Signs include hypotension (SBP <90 mmHg), and evidence of peripheral vasoconstriction such as oliguria, cyanosis and sweating	

Killip T, 3rd, Kimball JT. Treatment of myocardial infarction in a coronary care unit. A two year experience with 250 patients. *Am J Cardiol* 1967;**20**:457–464.
Forrester JS, Diamond GA, Swan HJ. Correlative classification of clinical and hemodynamic function after acute myocardial infarction. *Am J Cardiol* 1977;**39**:137–145.

Algorithm for the diagnosis of heart failure

An algorithm for the diagnosis of HF or LV dysfunction is shown in *Figure 1*. The diagnosis of HF is not sufficient alone. Appropriate investigations are required to establish the cause of the HF,

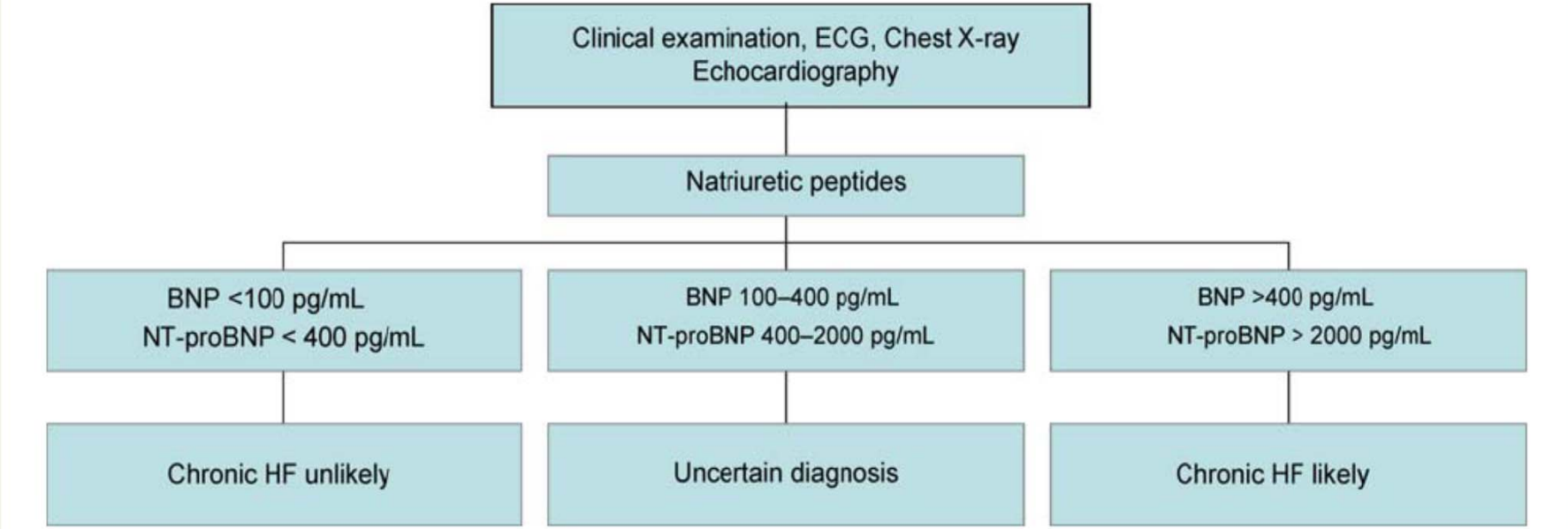

Figure 1 Flow chart for the diagnosis of HF with natriuretic peptides in untreated patients with symptoms suggestive of HF.

Diagnostic techniques

Diagnostic tests in heart failure

Several diagnostic tests are employed routinely to confirm or rule out the diagnosis of HF (*Table 11*). Diagnostic tests are usually most sensitive for the detection of patients with HF and reduced EF. Diagnostic findings are often less pronounced in patients with HFPEF. Echocardiography is the most useful method for evaluating systolic and diastolic dysfunction.

Table 11 Diagnostic assessments supporting the presence of heart failure

Assessment	Diagnosis of heart failure	
	Supports if present	Opposes if normal or absent
Compatible symptoms	++	++
Compatible signs	++	+
Cardiac dysfunction on echocardiography	+++	+++
Response of symptoms or signs to therapy	+++	++
ECG		
Normal		++
Abnormal	++	+
Dysrhythmia	+++	+
Laboratory		
Elevated BNP/NT-proBNP	+++	+
Low/normal BNP/NT-proBNP	+	+++
Hyponatraemia	+	+
Renal dysfunction	+	+
Mild elevations of troponin	+	+
Chest X-ray		
Pulmonary congestion	+++	+
Reduced exercise capacity	+++	++
Abnormal pulmonary function tests	+	+
Abnormal haemodynamics at rest	+++	++

+ = some importance; ++ = intermediate importance; +++ = great importance.

Chest X-ray

The chest X-ray (in two planes) is useful to detect cardiomegaly, pulmonary congestion, and pleural fluid accumulation, and can demonstrate the presence of pulmonary disease or infection causing or contributing to dyspnoea (*Table 13*). Apart from congestion, findings are predictive of HF only in the context of typical signs and symptoms. Cardiomegaly can be absent not only in acute but also in chronic HF.

Table 13 Common chest X-ray abnormalities in heart failure

Abnormality	Causes	Clinical Implications
Cardiomegaly	Dilated LV, RV, atria Pericardial effusion	Echo/Doppler
Ventricular hypertrophy	Hypertension, aortic stenosis, hypertrophic cardiomyopathy	Echo/Doppler
Normal pulmonary findings	Pulmonary congestion unlikely	Reconsider diagnosis (if untreated) Serious lung disease unlikely
Pulmonary venous congestion	Elevated LV filling pressure	Left heart failure confirmed
Interstitial oedema	Elevated LV filling pressure	Left heart failure confirmed
Pleural effusions	Elevated filling pressures HF likely if bilateral Pulmonary infection, surgery, or malignant effusion	Consider non-cardiac aetiology if abundant If abundant, consider diagnostic or therapeutic centres
Kerley B lines	Increased lymphatic pressures	Mitral stenosis or chronic HF
Hyperlucent lung fields	Emphysema or pulmonary embolism	Spiral CT, spirometry, Echo
Pulmonary infection	Pneumonia may be secondary to pulmonary congestion	Treat both infection and HF
Pulmonary infiltration	Systemic disease	Diagnostic work-up

Electrocardiogram

An electrocardiogram (ECG) should be performed in every patient with suspected heart failure.

Electrocardiographic changes are common in patients suspected of having HF (*Table 12*). An abnormal ECG has little predictive value for the presence of HF. If the ECG is completely normal, HF, especially with systolic dysfunction, is unlikely (<10%).

Table 12 Common ECG abnormalities in heart failure

Abnormality	Causes	Clinical implications
Sinus tachycardia	Decompensated HF, anaemia, fever, hyperthyroidism	Clinical assessment Laboratory investigation
Sinus bradycardia	β-Blockade, digoxin Anti-arrhythmics Hypothyroidism Sick sinus syndrome	Evaluate drug therapy Laboratory investigation
Atrial tachycardia/flutter/ fibrillation	Hyperthyroidism, infection, mitral valve diseases Decompensated HF, infarction	Slow AV conduction, medical conversion, electroversion, catheter ablation, anticoagulation
Ventricular arrhythmias	Ischemia, infarction, cardiomyopathy, myocarditis hypokalaemia, hypomagnesaemia Digitalis overdose	Laboratory investigation Exercise test, perfusion studies, coronary angiography, electrophysiology testing, ICD
Ischaemia/Infarction	Coronary artery disease	Echo, troponins, coronary angiography, revascularization
Q waves	Infarction, hypertrophic cardiomyopathy LBBB, pre-excitation	Echo, coronary angiography
LV hypertrophy	Hypertension, aortic valve disease, hypertrophic cardiomyopathy	Echo/Doppler
AV block	Infarction, drug toxicity, myocarditis, sarcoidosis, Lyme disease	Evaluate drug therapy, pacemaker, systemic disease
Microvoltage	Obesity, emphysema, pericardial effusion, amyloidosis	Echo, chest X-ray
QRS length >120 ms of LBBB morphology	Electrical and mechanical dysynchrony	Echo CRT-P, CRT-D

Table 14 Common laboratory test abnormalities in heart failure

Abnormality	Cause	Clinical implications
Increased serum creatinine (>150 μmol/L)	Renal disease ACEI/ARB, aldosterone blockade	Calculate GFR, Consider reducing ACEI/ARB, or aldosterone blocker dose Check potassium and BUN
Anaemia (<13 g/dL in men, <12 in women)	Chronic HF, haemodilution, iron loss or poor utilization, renal failure, chronic disease	Diagnostic work-up Consider treatment
Hyponatraemia (<135 mmol/L)	Chronic HF, haemodilution. AVP release, diuretics	Consider water restriction, reducing diuretic dosage Ultrafiltration, vasopressin antagonist
Hypernatraemia (>150 mmol/L)	Hyperglycaemia Dehydratation	Assess water intake Diagnostic work-up
Hypokalaemia (<3.5 mmol/L)	Diuretics, secondary hyperaldosteronism	Risk of arrhythmia Consider potassium supplements, ACEIs/ARB, aldosterone blockers
Hyperkalaemia (>5.5 mmol/L)	Renal failure, potassium supplement, renin–angiotensin–aldosterone system blockers	Stop potassium-sparing treatment (ACEIs/ARB, aldosterone blockers) Assess renal function and pH Risk of bradycardia
Hyperglycaemia (>6.5 mmol/L)	Diabetes, insulin resistance	Evaluate hydration, treat glucose intolerance
Hyperuricaemia (>500 μmol/L)	Diuretic treatment, gout, malignancy	Allopurinol Reduce diuretic dose
BNP >400 pg/mL, NT-proBNP >2000 pg/mL	Increased ventricular wall stress	HF likely Indication for echo Consider treatment
BNP <100 pg/mL, NT-proBNP <400 pg/mL	Normal wall stress	Re-evaluate diagnosis HF unlikely if untreated
Albumin high (>45 g/L)	Dehydratation, myeloma	Rehydrate
Albumin low (<30 g/L)	Poor nutrition, renal loss	Diagnostic work-up
Transaminase increase	Liver dysfunction Right heart failure Drug toxicity	Diagnostic work-up Liver congestion Reconsider therapy
Elevated troponins	Myocyte necrosis Prolonged ischaemia, severe HF, myocarditis, sepsis, renal failure, pulmonary embolism	Evaluate pattern of increase (mild increases common in severe HF) Coronary angiography Evaluation for revascularization
Abnormal thyroid tests	Hyper/hypothyroidism Amiodarone	Treat thyroid abnormality
Urinalysis	Proteinuria, glycosuria, bacteria	Diagnostic work-up Rule out infection
INR >2.5	Anticogulant overdose Liver congestion	Evaluate anticoagulant dosage Assess liver function Assess anticoagulant dose
CRP >10 mg/L, neutrophilic leukocytosis	Infection, inflammation	Diagnostic work-up

Echocardiography

The term echocardiography is used to refer to all cardiac ultrasound imaging techniques, including pulsed and continuous wave Doppler, colour Doppler and tissue Doppler imaging (TDI).

Table 15 Common echocardiographic abnormalities in heart failure

Measurement	Abnormality	Clinical implications
LV ejection fraction	Reduced (<45–50%)	Systolic dysfunction
LV function, global and focal	Akinesis, hypokinesis, dyskinesis	Myocardial infarction/ischaemia Cardiomyopathy, myocarditis
End-diastolic diameter	Increased (>55–60 mm)	Volume overload HF likely
End-systolic diameter	Increased (>45 mm)	Volume overload HF likely
Fractional shortening	Reduced (<25%)	Systolic dysfunction
Left atrial size	Increased (>40 mm)	Increased filling pressures Mitral valve dysfunction Atrial fibrillation
Left ventricular thickness	Hypertrophy (>11–12 mm)	Hypertension, aortic stenosis, hypertrophic cardiomyopathy
Valvular structure and function	Valvular stenosis or regurgitation (especially aortic stenosis and mitral insufficiency)	May be primary cause of HF or complicating factor Assess gradients and regurgitant fraction Assess haemodynamic consequences Consider surgery
Mitral diastolic flow profile	Abnormalities of the early and late diastolic filling patterns	Indicates diastolic dysfunction and suggests mechanism
Tricuspid regurgitation peak velocity	Increased (>3 m/s)	Increased right ventricular systolic pressure Suspect pulmonary hypertension
Pericardium	Effusion, haemopericardium, thickening	Consider tamponade, uraemia, malignancy, systemic disease, acute or chronic pericarditis, constrictive pericarditis
Aortic outflow velocity time integral	Reduced (<15 cm)	Reduced low stroke volume
Inferior vena cava	Dilated Retrograde flow	Increased right atrial pressures Right ventricular dysfunction Hepatic congestion

Table 16 Doppler-echocardiographic indices and ventricular filling

Doppler indices	Pattern	Consequence
E/A waves ratio	Restrictive (>2, short deceleration time <115 to 150 ms)	High filling pressures Volume overload
	Slowed relaxation (<1)	Normal filling pressures Poor compliance
	Normal (>1)	Inconclusive as may be pseudo-normal
E/Ea	Increased (>15)	High filling pressures
	Reduced (<8)	Low filling pressures
	Intermediate (8–15)	Inconclusive
(A mitral–A pulm) duration	>30 ms	Normal filling pressures
	<30 ms	High filling pressures
Pulmonary S wave	>D wave	Low filling pressures
Vp	<45 cm/s	Slow relaxation
E/Vp	>2.5	High filling pressures
	<2	Low filling pressures
Valsalva manoeuvre	Change of the pseudonormal to abnormal filling pattern	Unmasks high filling pressure in the setting of systolic and diastolic dysfunction

There are three types of abnormal filling patterns recognized conventionally in patients in sinus rhythm.

1. A pattern of 'impaired' myocardial relaxation with a decrease in peak transmitral E-velocity, a compensatory increase in the atrial-induced (A) velocity, and therefore a decrease in the E/A ratio may be seen at an early stage of diastolic dysfunction; it is frequently seen in hypertension and in the normal elderly subject, and is generally associated with normal or low LV filling pressures.
2. In patients with elevated left atrial pressure, (decreased LV compliance, volume overload, mitral insufficiency), there may be a pattern of 'restrictive filling', with an elevated peak E-velocity, a short E-deceleration time, and a markedly increased E/A ratio.
3. In patients with an intermediate pattern between impaired relaxation and restrictive filling, the E/A ratio and the deceleration time may be normal, and a so-called 'pseudo-normalized filling pattern' may be seen. This pattern may be distinguished from normal filling by analysis of other Doppler variables such as pulmonary venous flow or TDI of the mitral plane motion.

Assessment of heart failure with preserved ejection fraction (HFPEF)

Echocardiography plays a major role in confirming the diagnosis of HFPEF. The diagnosis of HFPEF requires three conditions to be satisfied:

1. Presence of signs and/or symptoms of chronic HF.
2. Presence of normal or only mildly abnormal LV systolic function (LVEF $\geq$45–50%).
3. Evidence of diastolic dysfunction (abnormal LV relaxation or diastolic stiffness).

Pulmonary function tests

Measurements of pulmonary function are of limited value in the diagnosis of HF. However, these tests are useful in demonstrating or excluding respiratory causes of breathlessness and assessing the potential contribution of lung disease to the patient's dyspnoea. Routine spirometry evaluates the extent of obstructive airways disease. The presence of pulmonary congestion may influence the test results. Blood gases are normal in well-compensated chronic HF. A reduction of arterial oxygen saturation should lead to a search for other diagnoses.

Exercise testing

Exercise testing is useful for the objective evaluation of exercise capacity and exertional symptoms, such as dyspnoea and fatigue. The 6-min walk test is a simple, reproducible, readily available tool frequently employed to assess submaximal functional capacity and evaluate the response to intervention. A normal peak exercise test in a patient not receiving treatment excludes the diagnosis of symptomatic HF. Either a cycle ergometer or treadmill may be used with a modified HF protocol employing a slow increase in workload. Gas exchange analysis during exercise is preferable as

Ambulatory ECG monitoring (Holter)

Ambulatory ECG monitoring is valuable in the assessment of patients with symptoms suggestive of an arrhythmia (e.g. palpitations or syncope) and in monitoring ventricular rate control in patients with AF. It may detect and quantify the nature, frequency, and duration of atrial and ventricular arrhythmias and silent episodes of ischaemia which could be causing or exacerbating symptoms of HF. Episodes of symptomatic, non-sustained ventricular tachycardia (VT) are frequent in HF and are associated with a poor prognosis.

Cardiac catheterization

Cardiac catherization is unnecessary for the routine diagnosis and management of patients with HF. Invasive investigation is frequently indicated to elucidate aetiology, to obtain important prognostic information, and if revascularization is being considered.

Prognosis

Determining prognosis in HF is complex. Diverse aetiologies, age, frequent co-morbidities, variation in individual progression and outcomes (sudden vs. progressive HF death) must be considered. The impact on prognosis of specific treatments in individual patients with HF is often difficult to predict. The variables most

Table 17 Conditions associated with a poor prognosis in heart failure

Demographics	Clinical	Electrophysiological	Functional/ exertional	Laboratory	Imaging
Advanced age*	**Hypotension***	**Tachycardia Q waves**	**Reduced work, low peak VO_2***	**Marked elevation of BNP/NT pro-BNP***	**Low LVEF***
Ischaemic aetiology*	**NYHA functional class III–IV***	**Wide QRS***		**Hyponatraemia***	
Resuscitated sudden death*	**Prior HF hospitalization***	**LV hypertrophy Complex ventricular arrhythmias***		**Elevated troponin* Elevated biomarkers, neurohumoral activation***	
Poor compliance	Tachycardia	Low heart rate variability Atrial fibrillation	Poor 6 min walk distance	Elevated creatinine/BUN	Increased LV volumes
Renal dysfunction	Pulmonary rales	T-wave alternans	High VE/VCO_2 slope	Elevated bilirubin Anaemia	Low cardiac index
Diabetes	Aortic stenosis		Periodic breathing	Elevated uric acid	High LV filling pressure
Anaemia	Low body mass index				Restrictive mitral filling pattern, pulmonary hypertension
COPD	Sleep-related breathing disorders				Impaired right ventricular function
Depression					

* = powerful predictors.

Non-pharmacological management

Table 18 Essential topics in patient education with associated skills and appropriate self-care behaviours

Educational topics	Skills and self-care behaviours
Definition and aetiology of heart failure	Understand the cause of heart failure and why symptoms occur
Symptoms and signs of heart failure	Monitor and recognize signs and symptoms Record daily weight and recognize rapid weight gain Know how and when to notify healthcare provider Use flexible diuretic therapy if appropriate and recommended
Pharmacological treatment	Understand indications, dosing, and effects of drugs Recognize the common side-effects of each drug prescribed
Risk factor modification	Understand the importance of smoking cessation Monitor blood pressure if hypertensive Maintain good glucose control if diabetic Avoid obesity
Diet recommendation	Sodium restriction if prescribed Avoid excessive fluid intake Modest intake of alcohol Monitor and prevent malnutrition
Exercise recommendations	Be reassured and comfortable about physical activity Understand the benefits of exercise Perform exercise training regularly
Sexual activity	Be reassured about engaging in sex and discuss problems with healthcare professionals Understand specific sexual problems and various coping strategies
Immunization	Receive immunization against infections such as influenza and pneumococcal disease
Sleep and breathing disorders	Recognize preventive behaviour such as reducing weight of obese, smoking cession, and abstinence from alcohol Learn about treatment options if appropriate
Adherence	Understand the importance of following treatment recommendations and maintaining motivation to follow treatment plan
Psychosocial aspects	Understand that depressive symptoms and cognitive dysfunction are common in patients with heart failure and the importance of social support Learn about treatment options if appropriate
Prognosis	Understand important prognostic factors and make realistic decisions Seek psychosocial support if appropriate

Self-care management

- Self-care management is a part of successful HF treatment and can significantly impact on symptoms, functional capacity, well-being, morbidity, and prognosis. Self-care can be defined as actions aimed at maintaining physical stability, avoidance of behaviour that can worsen the condition, and detection of the early symptoms of deterioration.[68]
- Important self-care behaviours in heart failure are presented in *Table 18*.
- It is recommended that healthcare professionals provide comprehensive heart failure education and counselling.