

Managing heart failure

The key role of the GP

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Management of most patients with heart failure occurs at an outpatient level, with the GP playing a central role in initial diagnosis, counselling, regular assessment of fluid status, titration and monitoring of medications and end-of-life care.

KEY POINTS

- Heart failure (HF) should be considered in all older patients presenting with a new wheeze.
- Patients with severe acute decompensated HF (ADHF) are best managed in hospital; those with mild decompensation or chronic compensated HF are often managed as outpatients by their GPs.
- The first priority in patients with ADHF is achievement of a euvolaemic state (via diuretics and fluid intake restriction).
- Medications of use in patients with systolic HF are diuretics, ACE inhibitors/angiotensin receptor antagonists, beta blockers, aldosterone receptor antagonists, ivabradine and digoxin.
- Apart from diuretics, no specific drug therapies have been shown to be consistently of benefit in patients with diastolic HF, the type of HF usually present in elderly patients.
- Renal dysfunction is common in HF. Assessment of volume status, peripheral perfusion and recent changes in drug therapy should guide management.

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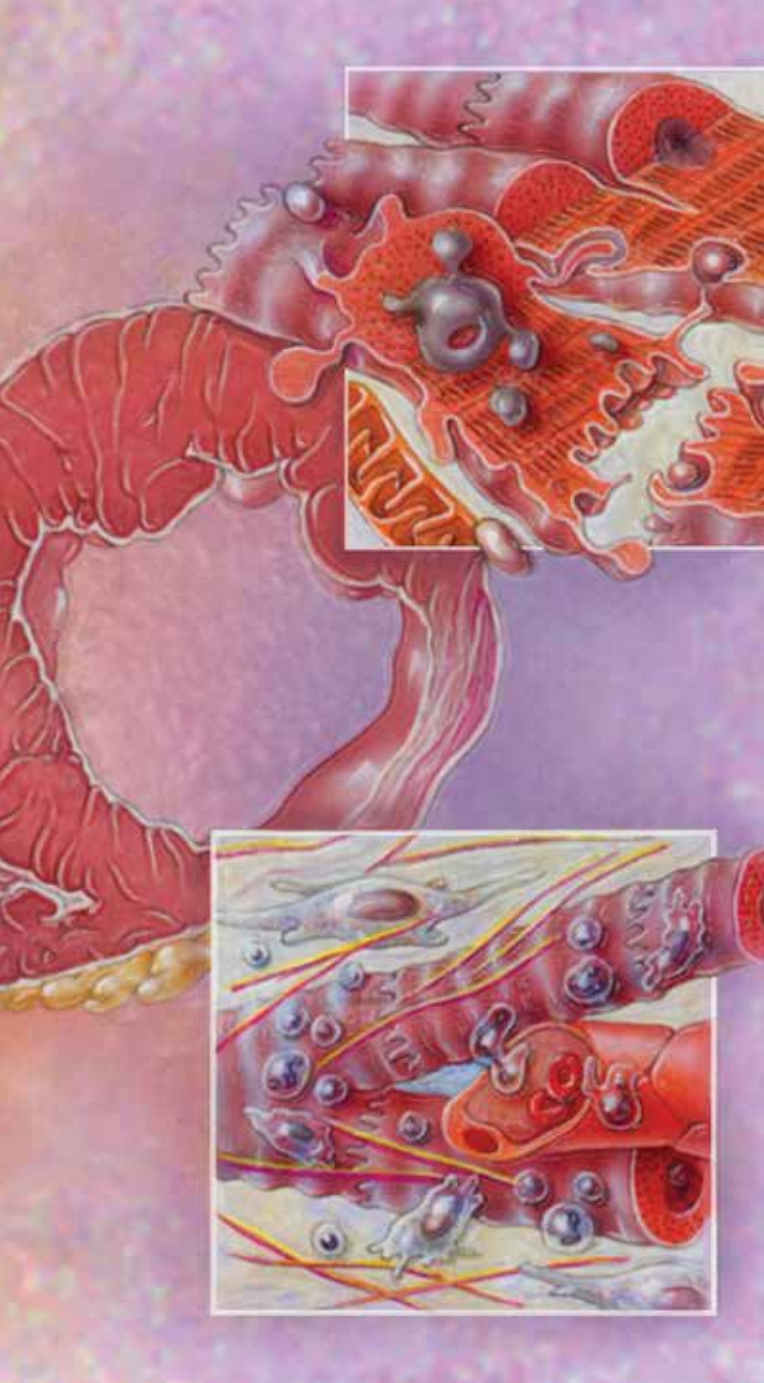


With an ageing population coupled with the growing burden of hypertension, diabetes and coronary disease, heart failure (HF) is an ever-present problem. An estimated 300,000 people in Australia are living with HF, and there are 30,000 diagnoses each year. Tackling this problem requires an integrated, multidisciplinary approach involving GPs, cardiologists and consultant physicians, HF nurses and allied heart professionals. This article reviews the GP's central role in the management of patients with HF. Further information can be obtained from the various published guidelines on this condition (Box 1).

What is heart failure?

HF is a complex syndrome whereby systemic perfusion is insufficient to meet the body's metabolic demands. Although its clinical features are quite varied, it is frequently characterised

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1. GUIDELINES ON HEART FAILURE

Australian guidelines

- National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand. Guidelines for the prevention, detection and management of chronic heart failure in Australia. Canberra: National Heart Foundation of Australia; 2011. Available online at: http://www.csanz.edu.au/documents/guidelines/clinical_practice/2011_HF_CSANZ_Chronic_Heart_Failure.pdf

US guidelines

- Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation* 2013; 128: e240-e327. Available online at: <http://circ.ahajournals.org/content/128/16/e240.full.pdf>

European guidelines

- McMurray JJ, Adamopoulos S, Anker SD, et al; European Society of Cardiology (ESC) Committee for Practice Guidelines. ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure 2012. *Eur Heart J* 2012; 33: 1787-1847. Available online at: <http://www.escardio.org/guidelines-surveys/esc-guidelines/Pages/acute-chronic-heart-failure.aspx>

distinction is relevant as many therapies have been shown to be beneficial only in systolic HF. HF can further be classified according to its chronicity (acute versus chronic), whether it predominantly involves the LV or right ventricle (RV) and whether it predominately leads to congestion (backward failure) or effort intolerance (forward failure).

Causes of heart failure

Identifying and treating the underlying aetiology of HF is critical and doing so may result in significant improvement and/or recovery of cardiac function (Table 1). Cardiomyopathy, defined as a disease of the heart muscle, is 'idiopathic' or familial in some patients but it is crucial to look for its underlying aetiologies in anyone presenting with either a new diagnosis of cardiomyopathy or a deterioration in systolic function. In particular, ischaemic heart disease accounts for over 50% of patients presenting with HF, and is often occult. Thus, exclusion of coronary disease is paramount in all patients presenting with HF of unclear aetiology.

Clinical presentation of heart failure

The syndrome of HF exists on a spectrum, ranging from asymptomatic LV dysfunction to chronic symptomatic compensated HF to acute decompensated HF (ADHF). Patients with chronic compensated HF may be 'euvolaemic', and may either be asymptomatic or have mild to moderate dyspnoea on exertion – the

by elevated left ventricular (LV) filling pressures, inadequate peripheral oxygen delivery and neurohormonal activation. In particular, activation of both the renin–angiotensin–aldosterone and sympathetic nervous systems leads to fluid retention and adverse pathological remodelling of the heart.

There are numerous underlying aetiologies of HF, and classification can be confusing. HF is referred to as 'systolic' (or 'HF with a reduced ejection fraction' [HF_rEF]) if LV contraction is impaired, as defined by an LV ejection fraction (LVEF) less than 50%. 'Diastolic' HF, or 'heart failure with preserved ejection fraction' (HF_pEF), is characterised by a normal EF, indicating a problem with myocardial relaxation and LV filling. Diastolic HF is particularly common in the elderly, and is thought to affect, at varying degrees, up to 50% of individuals over the age of 70 years.

Although systolic and diastolic HF frequently coexist, the

TABLE 1. IDENTIFYING THE UNDERLYING CAUSE OF CARDIOMYOPATHY

Aetiology	Specific investigations	Treatment(s)
Ischaemic heart disease	ECG, stress test (nuclear or echocardiography), cardiac CT, coronary angiography, cardiac MRI	Risk factor modification, antianginal therapy, revascularisation (stent or bypass surgery)
Hypertension	ECG, echocardiography, consider secondary causes	Antihypertensive medication
Valvular heart disease	Echocardiography	Valve repair or replacement
Alcoholic cardiomyopathy	Liver function tests, MCV	Abstinence, counselling, thiamine, anticraving medications
Tachycardia-mediated cardiomyopathy	ECG, Holter monitor	Antiarrhythmic medications, cardioversion, radiofrequency ablation
Thyroid disease (hyper- or hypothyroidism)	Thyroid function tests	Medications to restore euthyroid state
Drugs (anthracyclines, trastuzumab, clozapine, amphetamines, cocaine)	Drug history, urine toxicology screen	Dose adjustment or withdrawal
Infiltrative disease (haemochromatosis, amyloidosis, sarcoidosis)	Iron studies, calcium, serum ACE inhibitor level, protein electrophoresis, cardiac MRI, endomyocardial biopsy	Treat specific cause (e.g. venesection, iron chelation for haemochromatosis)
Viral cardiomyopathy	Consider HIV serology, routine 'viral antibody panel' often unhelpful	Antiretroviral therapy for HIV infection (where applicable), corticosteroids for fulminant myocarditis

Abbreviations: ACE = angiotensin-converting enzyme; CT = computed tomography; ECG = electrocardiography; HIV = human immunodeficiency virus; MCV = mean corpuscular volume; MRI = magnetic resonance imaging.

New York Heart Association (NYHA) HF symptom classification system is used to assess the severity of functional limitations (Table 2). An acute deterioration in symptoms is referred to as acute decompensated heart failure (ADHF). Patients with ADHF usually have elevated ventricular filling pressures, which may lead to fluid accumulation in the interstitial or alveolar spaces of the lungs and/or the peripheries, with or without a low cardiac output state.

HF is common, and its symptoms and signs are often non-specific, requiring the clinician to have a high index of suspicion. The significances of these clinical features are discussed in Table 2.

Precipitants of an acute decompensation

In addition to identifying the underlying cause of HF, it is critical to identify precipitants of an acute decompensation (Box 2). These precipitants should be addressed.

Investigations and their interpretation

Patients with evidence of severe ADHF are best managed in an inpatient setting, whereas those with mild decompensation or chronic compensated HF are often managed as outpatients by

their GPs. Numerous investigations are useful in the diagnosis, management and monitoring of HF, as discussed below and listed in Table 3.

Blood tests

Basic blood investigations in patients with HF include the following tests.

- **Full blood count.** Anaemia and sepsis (leukocytosis) are frequent precipitants of ADHF. Iron deficiency is also common in chronic HF, and many patients derive symptomatic benefit from periodic iron infusions.
- **Urea, electrolytes and creatinine.** Renal impairment is common in patients with HF, and it is critical to know the patient's baseline creatinine level and assess their volume state and peripheral perfusion. Causes of renal impairment may include significant right heart failure or 'renal venous congestion' (corrected by diuretic escalation), dehydration or low cardiac output state (which may necessitate hospital admission and inotrope therapy). Drugs (especially ACE inhibitors, angiotensin receptor blockers and aldosterone antagonists) may cause hyperkalaemia and/or renal impairment. Hyponatraemia is seen in patients with

TABLE 2. SYMPTOMS AND SIGNS OF HEART FAILURE

Clinical feature	Comment
Symptom	
Dyspnoea (with exertion and/or at rest)	Severity often classified according to NYHA class: NYHA I (no symptoms with ordinary activity) NYHA II (symptoms with ordinary activity) NYHA III* (symptoms with less than ordinary activity) NYHA IV* (symptoms at rest)
Orthopnoea*	Highly specific symptom for decompensated HF and elevated LV filling pressures
Paroxysmal nocturnal dyspnoea*	Specific symptom for decompensated HF and elevated LV filling pressures
Fatigue	Nonspecific, but consider low cardiac output state
Oedema (peripheral, sacral, ascites)	Suggestive of elevated right atrial pressure
Nausea,* vomiting,* anorexia	Prominent symptom of severe right HF or low cardiac output state
Examination finding (sign)	
Tachycardia	Sinus tachycardia concerning for severe decompensation Atrial tachyarrhythmias also common (e.g. atrial fibrillation)
Hypotension	Concerning for cardiogenic shock if associated with symptoms or signs of poor tissue perfusion (light-headedness, confusion, cool peripheries, poor capillary refill, oliguria) However, many patients with compensated HF (especially on treatment) have asymptomatic hypotension (systolic BP approx 80 to 90 mmHg) without evidence of poor tissue perfusion and do not require alteration in treatment
Coarse bibasal crackles	Indicative of elevated LV filling pressures ± pulmonary oedema Patients may have ADHF without presence of bibasal crackles
Wheeze ('cardiac asthma')	Common finding in ADHF Consider HF, rather than asthma, in an elderly patient presenting with a new wheeze
Elevated jugular venous pressure	Very useful sign; however, one-third of patients with elevated LV filling pressures will have a normal jugular venous pressure May be difficult to see in obese patients
Laterally displaced apex	Suggestive of LV dilatation (chronic HF with a reduced LVEF)
S3 (third heart sound)	Suggestive of acute decompensation
Fluid retention (peripheral oedema, sacral oedema, ascites)	Not all patients with peripheral oedema have right HF In the absence of other evidence of HF, consider venous insufficiency, hypoalbuminaemia, drugs (e.g. calcium channel blockers), renal and liver disease
Cardiac cachexia (wasting)	Seen in end-stage HF; defined as a reduction of >6% in dry weight over a 12-month period

Abbreviations: ADHF = acute decompensated heart failure; BP = blood pressure; HF = heart failure; LV = left ventricle; LVEF = left ventricular ejection fraction; NYHA = New York Heart Association.

* Indicates severity (and consideration for inpatient management).

2. PRECIPITANTS OF ACUTE DECOMPENSATION

- Acute myocardial ischaemia
- Anaemia
- Arrhythmias (tachy- and brady-)
- Sepsis
- Noncompliance with medications (especially diuretics)
- Noncompliance with fluid restriction
- Thyroid dysfunction (hyper- or hypo-)
- Drugs (e.g. alcohol, recreational drugs, NSAIDs, calcium channel blockers, moxonidine, corticosteroids, tyrosine kinase inhibitors)
- Severe hypertension (especially in patients with HFpEF)
- Renal failure (e.g. acute glomerulonephritis)

Abbreviations: HFpEF = heart failure with preserved ejection fraction; NSAID = nonsteroidal anti-inflammatory drug.

advanced HF, and is a poor prognostic sign.

- **Liver function tests.** A cholestatic picture may be seen in patients with right heart failure and hepatic congestion, whereas elevated transaminase levels are suggestive of cardiogenic shock.
- **B-type natriuretic peptide (BNP).** Measurement of the BNP level is a very useful diagnostic test for differentiating ADHF from other causes of breathlessness. If the BNP level is less than 100 pg/mL, ADHF is unlikely; however, a BNP level above 400 pg/mL is highly suggestive of ADHF.
- **Cardiac enzymes/markers (troponin, creatine kinase).** Determining the levels of cardiac enzymes or other cardiac markers is rarely useful in the outpatient setting. They are likely to be trivially elevated in all patients with a cardiomyopathy (even if compensated), and may be

TABLE 3. USEFUL INVESTIGATIONS IN HEART FAILURE

Investigations	Main use(s)
Basic blood tests	
Full blood count	Anaemia and sepsis may precipitate HF
Urea, electrolytes and creatinine	Renal impairment (HF, drugs), hyperkalaemia (drugs)
Liver function tests	Hepatic congestion from RHF (elevated GGT, ALP)
B-type natriuretic peptide	Elevated in HF associated with congestion (useful for diagnosis)
Cardiac enzymes/markers	Elevated in myocarditis, myocardial infarction
Baseline imaging	
12-lead ECG	Identifies cause and precipitants of HF (ischaemia, arrhythmias)
Chest x-ray	Confirms diagnosis of HF, assesses degree of congestion, identifies alternative cause of dyspnoea
Echocardiography	Identifies cause, type (systolic vs HFpEF) and severity of HF
Specialised tests	
Right heart catheterisation	Measures intracardiac and pulmonary pressures invasively
Cardiac MRI	Excellent tissue characterisation (myocarditis, scar)
Gated cardiac blood pool scan	Accurately determines EF (nuclear scan)
Coronary angiography	Accurately determines coronary anatomy and anatomical suitability for revascularisation
Abbreviations: ALP = alkaline phosphatase; ECG = electrocardiography; EF = ejection fraction; GGT = gamma-glutamyl transferase; HF = heart failure; HFpEF = heart failure with preserved ejection fraction; MRI = magnetic resonance imaging; RHF = right heart failure.	

significantly elevated in those with acute ischaemia or myocarditis.

12-lead ECG

The ECG is rarely normal in patients with HF. Although no findings are specific for HF, the ECG can be important to guide diagnosis and management decisions. Common findings include

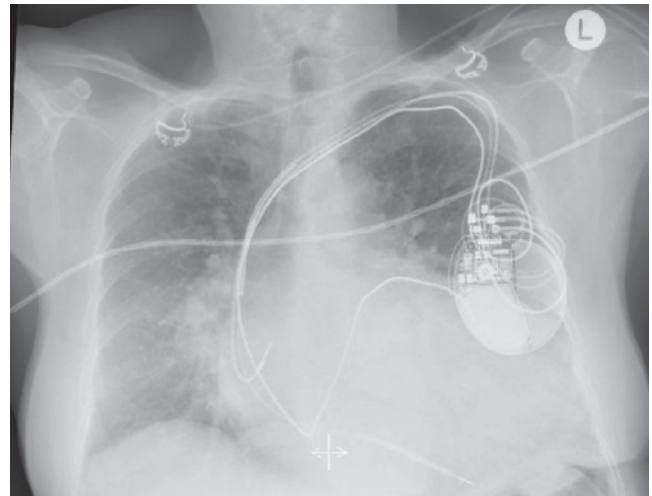


Figure 1. Chest x-ray of a 54-year-old woman with long-standing ischaemic cardiomyopathy admitted with acute decompensated heart failure. The x-ray shows increased cardiothoracic ratio and evidence of pulmonary congestion. There is a biventricular pacemaker in situ (leads in right atrium, right ventricle and coronary sinus/vein overlying the lateral wall of the left ventricle).

the following:

- evidence of underlying cause of HF, such as coronary artery disease (Q waves, ST/T wave changes), LV hypertrophy (hypertension), low voltage (amyloidosis), conduction disturbance (sarcoidosis)
- evidence of atrial enlargement ('p mitrale', 'p pulmonale') indicates chronicity
- arrhythmias (atrial and ventricular) – may lead to or worsen HF; atrial fibrillation should be treated with rate or rhythm control and anticoagulation is usually warranted according to the patient's CHA₂DS₂-VASc score
- left bundle branch block (LBBB) – patients with LBBB may derive benefit from biventricular pacing (discussed later).

Chest x-ray

The chest x-ray is useful for differentiating HF from primary pulmonary disease. Suggestive findings include increased cardiac-to-thoracic (cardiothoracic) width ratio of more than 50% (this is a nonspecific finding), bilateral perihilar airspace opacity, upper lobe diversion, Kerley B-lines and pleural effusions. Many patients may have devices, including defibrillators or pacemakers (Figure 1).

Echocardiography

Echocardiography is a key test for any patient presenting with symptoms or signs of HF. It accurately assesses both systolic function (EF) and diastolic function. Dilated atria and ventricles may suggest a degree of chronicity. Mitral regurgitation may be

3. WHEN TO REFER A PATIENT WITH HEART FAILURE TO A CARDIOLOGIST

Patients with any of the following features should be referred for investigation of aetiology and guidance of therapy:

- newly diagnosed HF, particularly in younger patients
- inability to uptitrate medications due to hypotension, bradycardia or renal dysfunction
- severe systolic dysfunction (LVEF <35%)
- presence of significant arrhythmias
- symptoms suggestive of myocardial ischaemia
- worsening symptoms despite medical therapy

Referral should, however, be considered for all patients with HF.

Abbreviations: HF = heart failure; LVEF = left ventricular ejection fraction.

the primary cause of HF, or may be due to long-standing LV dilatation (i.e. functional mitral regurgitation).

There may be clues regarding the aetiology of the HF, such as regional wall motion abnormalities suggestive of coronary artery disease or LV hypertrophy suggestive of hypertension. Thickened LV walls may also occur in hypertrophic cardiomyopathy or infiltrative disease (e.g. amyloidosis). Echocardiography can also provide a useful estimate of pulmonary artery pressure and pulmonary capillary wedge pressure (by E/E' ratio, using tissue Doppler).

Principles of HF management

In any patient presenting to their GP with HF, it is critical to establish the aetiology, precipitating factors and severity of the clinical syndrome and then to treat the underlying cause of the HF and any precipitants. However, the first priority in patients with ADHF should be achievement of a euvolaemic state by the use of diuretics and fluid intake restriction (as discussed below in the section on medications). Such patients usually have elevated ventricular filling pressures that may lead to pulmonary and systemic oedema.

Hospitalisation and referral

Indications for hospitalisation of patients with HF include:

- tachypnoea (respiratory rate over 40 breaths per minute)
- hypoxia (blood oxygenation saturation [SpO_2] below 90%)
- significant oedema
- persistent tachycardia
- symptomatic or significant hypotension (systolic blood pressure [BP] below 80 mmHg)
- new onset HF
- evidence of ischaemia
- failure of outpatient management.

All patients with HF should be strongly considered for referral to a cardiologist for investigation of aetiology and guidance of therapy. Specific features indicating referral are listed in Box 3.

Lifestyle measures

Once euvolaemia has been achieved in patients with HF, it is important to discuss lifestyle measures. These include daily weight monitoring, adherence to restrictions of intakes of fluid (1.5 to 2 L per day) and sodium (less than 2 g/day), alcohol abstinence, pneumococcal and influenza vaccination and smoking cessation.

Obstructive sleep apnoea should be treated with continuous positive airway pressure (CPAP) therapy if appropriate. Referral to cardiac rehabilitation (or a HF nursing service) and moderate aerobic exercise (intensity up to 70% of heart rate reserve performed three days per week) are also useful in patients with chronic stable HF.

Medications for systolic HF

Several classes of medications have been shown to be effective in patients with systolic HF, and these should be introduced where applicable and carefully uptitrated to the maximum tolerated dose.

Diuretics

Reduction of venous pressure and blood volume in patients with ADHF is achieved in the outpatient setting by commencement or escalation of diuretic therapy (frusemide) and enforcement of a 1.5 L fluid intake restriction. In patients who are 'frusemide-naïve', effective diuresis can sometimes be achieved with low doses (e.g. a frusemide dose of 20 mg daily). Patients with chronic renal impairment or those on long-term therapy may develop frusemide resistance, and the dose required may be significantly higher (up to 250 mg twice daily).

Other diuretics, such as a thiazide (especially hydrochlorothiazide) or a potassium-sparing diuretic (especially spironolactone or eplerenone), may be added for synergy. Occasionally other loop diuretics, such as bumetanide, may be used where frusemide is failing, especially for those patients with refractory oedema.

ACE inhibitors (or angiotensin-receptor blockers)

ACE inhibitors should be commenced in all patients with systolic HF (NYHA class I – i.e. no symptoms – to stable class IV – i.e. symptoms at rest, and especially in those whose LVEF is below 40%) to improve symptoms, prevent hospitalisation and reduce the risk of premature death. They may be initiated during or after diuretic therapy, and attempts should be made to slowly uptitrate therapy to target dose (Table 4). Renal function, potassium and blood pressure should be checked within a week of any dose escalation. As many patients with systolic HF have a low blood

pressure, doses can still be increased in the absence of hypoperfusion.

Patients intolerant of ACE inhibitors (especially development of a dry cough, which is seen in 10% of patients taking these medications) should be switched to an angiotensin-receptor blocker (ARB). Candesartan has the largest evidence-base in HF.

Beta blockers

Beta blockers should be prescribed together with ACE inhibitors in all patients with systolic HF (NYHA I to stable class IV, and especially if LVEF is below 40%) to improve symptoms, prevent hospitalisation and reduce the risk of premature death. Therapy should be commenced when the patient is euvoelaemic, at very low doses, and titrated slowly (every two to four weeks) to maximum tolerated dose. Only some beta blockers have been shown to be beneficial in HF (and indicated by the TGA for use in HF), as listed in Table 4.

Although active bronchospasm is a contraindication to the use of beta blockers, they can usually still be used in patients with stable chronic obstructive pulmonary disease. Nebivolol is highly beta₁-selective, and is often the best choice in these patients. Dosing may be limited by symptomatic bradycardia and hypotension.

Aldosterone receptor antagonists

Mortality benefits for aldosterone receptor antagonists have been well established in numerous trials involving patients with mild to severe symptoms (NYHA II to IV) and severe systolic HF (LVEF below 35%).

Either spironolactone or eplerenone can be used, but endocrine side effects (gynaecomastia, breast pain, menstrual disturbance, impotence) are more common with spironolactone, although it is less expensive. These medications may cause life-threatening hyperkalaemia, and should not be started if the patient's serum creatinine level is greater than 220 µmol/L in men or 180 µmol/L in women, or if the serum potassium level is greater than 5 mmol/L. Potassium and creatinine levels

TABLE 4. COMMONLY USED MEDICATIONS IN HEART FAILURE AND A GUIDE TO THEIR STARTING AND TARGET DOSES

Drug class	Starting dose	Target dose
ACE inhibitors		
Captopril	6.25 mg three times daily	50 mg three times daily
Enalapril	2.5 mg twice daily	10 mg twice daily
Lisinopril	2.5 mg daily	20 mg daily
Perindopril	1.25 mg daily	10 mg daily
Ramipril	1.25 mg daily	5 mg twice daily
Quinapril	5 mg twice daily	20 mg twice daily
Angiotensin receptor blockers		
Candesartan	4 mg daily	32 mg daily
Beta blockers		
Bisoprolol	1.25 mg daily	10 mg daily
Carvedilol	3.125 mg twice daily	50 mg twice daily
Metoprolol extended release	23.75 mg daily	190 mg daily
Nebivolol	1.25 mg daily	10 mg daily
Sinus node inhibitor		
Ivabradine	2.5 mg twice daily	7.5 mg twice daily
Aldosterone receptor antagonists		
Eplerenone	12.5 mg daily	50 mg daily
Spironolactone	12.5 mg daily	50 mg daily

should be checked within a few days, then monthly for six months (upon commencement or after a dose increase).

Other medications: ivabradine, digoxin, nitrates plus hydralazine

In patients unable to tolerate beta blocker dose escalation, the sinus node inhibitor ivabradine can be used if the heart rate remains above 77 beats per minute in sinus rhythm and the LVEF is below 35%. The most common side effect is development of luminous phenomena, described as sensations of enhanced brightness.

Digoxin is used to improve symptoms and prevent hospitalisation in patients with concomitant atrial fibrillation or persistent

HF despite first-line therapies. It has not been shown to improve survival.

The combination of a nitrate vasodilator (especially isosorbide dinitrate) and the peripheral vasodilator hydralazine may be beneficial in patients with hyperkalaemia or renal dysfunction who are unable to tolerate ACE inhibitors or ARBs or in addition to these agents if still symptomatic.

Medications in diastolic HF

No specific drug therapies have been shown in large trials to be consistently beneficial in patients with HFpEF (i.e. diastolic HF). Principles of treatment in these patients include treatment of

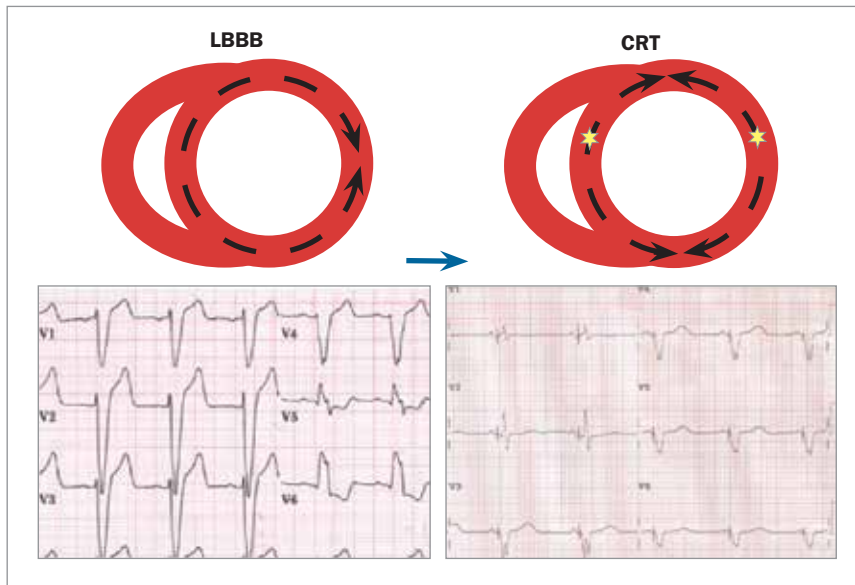


Figure 2. Pacing the septal and lateral walls with cardiac resynchronisation therapy (CRT) resynchronises the heart, negating the adverse effects of left bundle branch block (LBBB). (Leads denoted by yellow stars.)

hypertension, diuretic therapy to relieve symptoms of fluid overload (as for systolic HF) and maintenance of sinus rhythm or control of ventricular rate (as these patients are poorly tolerant of atrial fibrillation).

Device therapy

The past decade has been considered by many as the 'device era' in HF therapy. Implantable cardioverter defibrillators (ICDs) prevent sudden death through antitachycardia pacing and defibrillation for malignant ventricular arrhythmias, as well as antibradycardia pacing. Cardiac resynchronisation therapy (CRT) and ventricular assist devices (for advanced HF) improve the quality of life and prognosis in selected patients.

The presence of an LBBB on ECG is common in patients with HF. An LBBB causes delayed activation of the lateral wall relative to the RV and septum, leading to reduced mechanoenergetic efficiency of the heart. Biventricular pacing (i.e. CRT) aims to reverse the deleterious effects of LBBB, with implantation of RV and LV pacing leads generating two ventricular activation wavefronts that move towards

each other. The benefit of CRT lies in effective fusion of these two depolarisation wavefronts, synchronising the walls of the LV (Figure 2). CRT improves both symptoms and survival in patients with symptomatic HF (NYHA II to stable class IV) who are in sinus rhythm, have an LVEF below 35% despite three to six months of HF therapy, and have an LBBB.

Standard pacemakers implanted for bradycardia may also cause dyssynchrony (similar to an LBBB) due to RV pacing. Consideration should be given to upgrading to a CRT device (i.e. insertion of an LV lead) in patients with severe systolic dysfunction who are frequently paced, and in those with a deterioration in HF following pacemaker implantation.

Patient with systolic HF are at risk of sudden death due to ventricular arrhythmias. Those with a prior episode of resuscitated ventricular tachycardia (VT) or fibrillation should receive secondary prevention implantable cardioverter defibrillator (ICD) therapy. Primary prevention ICDs have also been shown to improve survival in those at high risk of ventricular arrhythmias, with the standard indication

4. MANAGING HEART FAILURE: KEY PRACTICE POINTS

- HF should be considered in all older patients presenting with a new wheeze.
- Beta blockers should be started at the lowest dose and uptitrated slowly.
- GPs play a vital role in ongoing management of patients with HF following hospital discharge, including further uptitration of HF therapy (especially ACE inhibitors and beta blockers) to maximum tolerated doses (for greatest benefit).
- Renal dysfunction is common in HF. Assessment of volume status, peripheral perfusion and recent changes in drug therapy should guide management.
- Many patients with severe systolic dysfunction have low blood pressure at baseline (systolic blood pressure may be as low as 85 mmHg). If they are otherwise stable and peripheral perfusion is maintained, this does not necessitate specific therapy.
- Diastolic HF (HFpEF) is a common type of HF, particularly in elderly patients. Echocardiographic evidence of diastolic dysfunction, left atrial enlargement or left ventricular hypertrophy are supportive of this diagnosis.
- Serum BNP is useful in differentiating HF from other causes of dyspnoea.
- In patients with newly diagnosed systolic HF who are treated effectively, EF will improve (or even normalise) in about one-third of cases and remain the same in another one-third.
- Aldosterone antagonists (eplerenone, spironolactone) can cause life-threatening hyperkalaemia and renal impairment. UEC should be checked regularly.

Abbreviations:

ACE = angiotensin-converting enzyme;
 BNP = B-type natriuretic peptide;
 EF = ejection fraction;
 GP = general practitioner;
 HF = heart failure;
 HFpEF = heart failure with preserved ejection fraction;
 UEC = urea, electrolytes and creatinine.

being persistent severe systolic dysfunction (EF below 35%) despite three to six months of HF therapy in patients with NYHA II to III symptoms.

In patients with advanced HF refractory to medical therapy or cardiogenic shock, ventricular assist devices are often considered as a bridge to heart transplantation. These patients should be referred to tertiary hospitals with expertise in HF and transplantation.

Multidisciplinary clinics

With the burden of hospital readmission rates for HF rapidly increasing, new models of care are being developed to deliver holistic, patient-centred care. This often involves GPs with a special interest in HF providing integrated care with the assistance of nurse practitioners, physiotherapists, social workers, dietitians, psychologists and specialists co-located in multidisciplinary community-based practices. Central to this model are patient and family education, recognition of early signs of decompensation (e.g. progressive weight gain), provision of psychological support and advanced care planning.

Remote monitoring

Remote monitoring is also being pioneered to enable patients to have daily 'contact' with healthcare experts, often with the aid of telemedicine. This incorporates stand-alone systems that transmit clinical information (vital signs, weight and symptoms), new devices that monitor haemodynamics, and existing pacemakers or defibrillators that can transmit data such as heart rate variability and arrhythmia burden.

Conclusion

Although HF continues to be a growing problem in our community, there are numerous therapies available to improve symptoms, prevent hospitalisation and improve life expectancy. Because most management occurs at an outpatient level, the GP plays a central role in all aspects of HF – from initial diagnosis, to counselling, to regular assessment of fluid status, to titration and monitoring of medications, and finally end-of-life care. Practice points for GPs are provided in Box 4. **MT**

Further reading

McMurray JJ, Adamopoulos S, Anker SD, et al; European Society of Cardiology (ESC) Committee for Practice Guidelines. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2012. *Eur Heart J* 2012; 33: 1787-1847. Available online at: <http://www.escardio.org/guidelines-surveys/esc-guidelines/Pages/acute-chronic-heart-failure.aspx> (accessed April 2015).

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COMPETING INTERESTS: None.

ONLINE CPD JOURNAL PROGRAM

Is an elderly patient presenting with a new wheeze more likely to have asthma or heart failure?

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