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# Chronic heart failure

*Improving life with modern therapies*

## Background

Chronic heart failure (CHF) is an increasingly common condition with increasing prevalence in the aging population. It has a significant mortality and is associated with a high incidence of hospitalisation and morbidity.

## Objective

This article describes the aspects of modern therapy that can improve survival, reduce hospitalisation and improve quality of life for CHF patients.

## Discussion

A careful history, physical examination and judicious investigation (including chest X-ray, electrocardiogram, complete blood profile and echocardiogram) can often identify the cause of CHF, the severity of CHF and help guide management. Treatments which have been shown to be of significant benefit include angiotensin converting enzyme inhibitors, beta-blockers, aldosterone antagonists and angiotensin receptor blockers. Loop diuretics, nitrates, digoxin, hydralazine and amiodarone may be used when patients do not respond to initial therapy. Review by a cardiologist is often useful to exclude myocardial ischaemia and to perform echocardiography which is a key investigation in assessment of CHF patients. Ongoing regular review with uptitration of medications to achieve target blood pressure and pulse and exclude exacerbating conditions can lead to improvements in care and facilitate successful outcomes in CHF patients who are often very unwell.

**Keywords:** heart failure; therapy; general practice



Chronic heart failure (CHF) affects over 300 000 Australians with another 30 000 new cases diagnosed each year.<sup>1</sup> Prevalence increases with age, from 2.5% in people aged 55–64 years to 8.2% in those aged over 75 years.<sup>1</sup> Despite improved understanding of the pathophysiology and management, morbidity and mortality remain high, with CHF causing 43 000 hospitalisations and 2200 deaths in 2006. This underestimates the burden of disease as it does not include indirect deaths and hospitalisations due to CHF.<sup>1</sup> A sound understanding of modern therapies is crucial as general practitioners play a central role in the management of CHF.

The National Heart Foundation and Cardiac Society of Australia and New Zealand *Guidelines for the prevention, detection and management of chronic heart failure*<sup>2</sup> is a valuable reference for GPs (available at [www.heartfoundation.org.au](http://www.heartfoundation.org.au)).

## Initial clinical assessment

### History

Early CHF symptoms include exertional dyspnoea and fatigue, with orthopnoea, paroxysmal nocturnal dyspnoea and ankle oedema occurring later. Less obvious symptoms include persistent cough, especially when supine or nocturnal, fatigue, nausea and anorexia. The New York Heart Association functional classification<sup>3</sup> should be documented initially and with each review to monitor progress. Their classification of CHF symptoms is:

- Class I – asymptomatic left ventricular dysfunction
- Class II – symptoms with normal activities
- Class III – symptoms with less than normal activities
- Class IV – symptoms at rest.

Careful history to identify conditions or medications to explain the aetiology of CHF is important. This should include a history of myocardial infarction, risk factors for coronary artery disease, long standing hypertension, alcohol intake, antecedent viral illness, and prior rheumatic fever or murmurs. *Table 1* lists the common causes of CHF.

### Examination

Physical examination should focus on signs of the underlying condition (eg. murmur), ventricular strain (gallop rhythm, tachycardia)



**Table 1. Causes of chronic heart failure<sup>4</sup>**

<b>More common causes</b>
Ischaemic heart disease
Hypertension
Valvular heart disease (stenosis or regurgitation)
Idiopathic dilated cardiomyopathy
<b>Less common causes</b>
Diabetes
Myocarditis (postviral or inflammatory)
Congenital heart disease
Drug induced, eg. alcohol or chemotherapy (anthracyclines)
HIV infection
Peripartum cardiomyopathy
Thyroid disease, eg. hypo- or hyper-thyroidism
Infiltrative conditions, eg. sarcoidosis, amyloidosis
Connective tissue diseases
Iron overload
Arrhythmia or tachycardia, eg. tachycardia induced cardiomyopathy

and signs of severity of heart failure. Mild heart failure patients may present with mildly elevated jugular venous pressure, basal inspiratory crepitations and mild peripheral or sacral oedema. More severe heart failure exhibits signs such as markedly elevated jugular venous pressure, crepitations beyond the mid-zones of the lungs, oedema above the mid tibia, pulsatile hepatomegaly and ascites.

## Initial investigations

Initial investigations include chest X-ray, electrocardiogram (ECG), blood tests (including electrolytes, renal function, liver enzymes, full blood count, thyroid function, iron studies, autoimmune screen) and a transthoracic echocardiogram – this is the key investigation in management of heart failure. Chest X-ray will identify cardiomegaly and pulmonary congestion, while the ECG will identify arrhythmia, tachycardia and evidence of previous myocardial infarction (q-waves). Blood results identify causes and aggravating factors such as anaemia or thyroid dysfunction. Measurement of the plasma brain natriuretic peptide (BNP), which is released by ventricular myocardium in response to pressure or volume stress, may help ‘rule out’ CHF.<sup>2</sup> A BNP <100 pg/mL makes the diagnosis of CHF very unlikely. However, BNP levels may be elevated in renal impairment, pulmonary embolism, or in patients with a history of CHF who present with other illnesses. BNP levels may also be mildly elevated in women and in those aged over 60 years with other severe illness.<sup>5</sup>

All patients with heart failure should undergo a transthoracic echocardiogram to assess left ventricular (LV) systolic function. However, up to one-third of CHF patients have normal LV systolic function. In the presence of abnormal diastolic function on

echocardiography, diastolic heart failure may be considered. This typically occurs in the elderly and in females with hypertension, but can also occur in those with diabetes, obesity and coronary artery disease (CAD).<sup>2</sup>

The echocardiogram can assess response of LV function to therapy by serial measurement of ejection fraction and identifying other causes of CHF, eg. valvular heart disease, LV hypertrophy, pulmonary hypertension and regional wall motion abnormalities suggesting CAD.

In all newly diagnosed CHF patients, assessment of CAD should be considered, as this represents a potentially reversible cause of CHF. This makes a critical difference in the management of patients with heart failure because if there is significant coronary artery disease and the patient is revascularised, this will improve ventricular function and prognosis. When there is a high clinical suspicion of CAD (ie. angina, risk factors or prior history of myocardial infarction (MI), echocardiographic or ECG evidence of silent MI) coronary angiography should be considered. If CAD is less likely then a noninvasive imaging test should be undertaken (eg. stress echocardiogram or nuclear perfusion scan). If CAD is identified, then revascularisation needs to be considered in patients with myocardial viability (eg. by dobutamine stress echocardiography, PET scan or cardiac magnetic resonance imaging [MRI]).<sup>2</sup>

## Management

Management aims to improve CHF symptoms, reduce hospitalisation and mortality. This involves identification and treatment of reversible causes and institution of proven medical and device therapies. To achieve this, the clinician must understand the pathophysiology of CHF. In CHF, there is activation of the renin-angiotensin-aldosterone system and the sympathetic nervous system. This is detrimental to ventricular structure and function. Chronic heart failure therapies, including angiotensin converting enzyme inhibitors (ACEIs), angiotensin receptor blockers (ARB) and beta blockers, target these activated pathways (*Figure 1*).

Many CHF treatments promote peripheral vasodilatation and relative bradycardia to enhance ventricular function. It is recommended to achieve a target heart rate of 55–60 bpm and systolic blood pressure of 105–110 mmHg.<sup>6</sup>

*Table 2* outlines important aspects of nonpharmacological therapies in CHF.

## Pharmacological therapies

### ACEIs and ARBs

Angiotensin converting enzyme inhibitors are indicated in all classes of CHF, including asymptomatic patients with LV dysfunction and following MI. They improve mortality, reduce hospitalisation and improve symptoms.<sup>2</sup> ACEIs should be started at low doses and titrated upward over 3–4 weeks. Renal function and electrolytes should be checked within 2 weeks of commencement and the drug discontinued if potassium levels exceed 5.5 mmol/L or

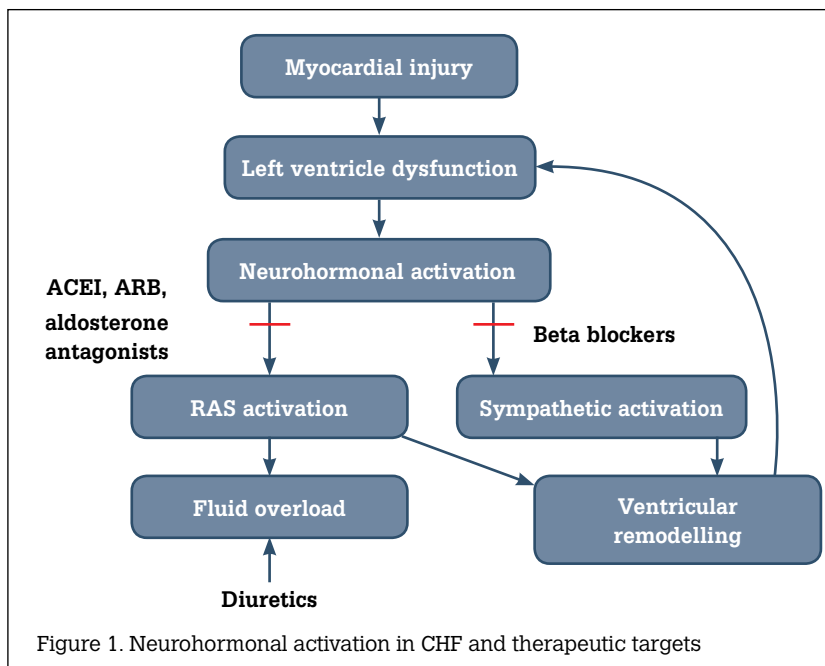


Figure 1. Neurohormonal activation in CHF and therapeutic targets

ACEIs due to cough, or in addition to ACEIs in CHF patients who remain symptomatic or hypertensive. Titration of ARBs and measurement of renal function and electrolytes is similar to that of ACEIs.

### Beta blockers

Beta blockers are indicated in all patients with CHF. They improve both mortality and morbidity.<sup>2</sup> Beta blockers with a proven mortality benefit include carvedilol, bisoprolol, nebivolol and extended release metoprolol.<sup>2</sup> Beta blockers should be commenced at small doses when the patient is euvoalaemic and titrated upward over a 1–2 month period. Patients should be haemodynamically stable with a systolic BP >85 mmHg without symptomatic postural drop, minimal peripheral oedema and no pulmonary crackles (rales) before prescribing beta blockers. Rapid uptitration may lead to adverse effects and inappropriate discontinuation of the drug. Beta blocker dose

should be reduced if the heart rate falls below 55 bpm. Hypotension can be treated by reducing doses of diuretics or other vasodilating drugs first, rather than reducing the dose of the beta blocker.

Side effects include hypotension, fatigue, bronchoconstriction in patients with reversible airways obstruction (>15% improvement in FEV<sub>1</sub> with bronchodilators) and mild worsening of CHF symptoms initially. It is important to warn patients of this last side effect before initiation. Nonvasodilatory beta blockers (bisoprolol or extended release metoprolol) may be helpful in patients with hypotension and cardioselective beta blockers (eg. bisoprolol, nebivolol) may be trialled in COPD patients without reversible airways obstruction. Nebivolol has been shown to be effective in patients over 70 years of age, regardless of ejection fraction (Table 4).

### Aldosterone antagonists

Spirolactone has been shown to have a mortality benefit in patients with Class III/IV CHF.<sup>2</sup> Eplerenone, a selective aldosterone antagonist, has been shown to reduce mortality in patients with post-MI LV dysfunction<sup>2</sup> and has a much less risk of gynecomastia than spironolactone. Careful monitoring of electrolytes and renal function is important as aldosterone antagonism may cause hyperkalaemia. Both drugs are contraindicated in significant renal impairment (GFR <30 mL/min).

### Diuretics

Diuretics are used to treat congestive symptoms due to salt and fluid overload and do not have a long term mortality benefit.<sup>2</sup> Once patients are euvoalaemic, diuretics may be reduced or weaned, especially if this will improve blood pressure and allow initiation of drugs with a proven mortality benefit (eg. beta blockers and ACEIs).

<b>Table 2. Nonpharmacological measures in CHF<sup>2</sup></b>
Support by GP/specialised heart failure nurse home visits
Sodium restriction (<2 g/day)
Fluid restriction 1.5 L/day (1L in more severe CHF)
Daily weighing at home with an action plan*
Alcohol cessation in alcohol related cardiomyopathy (<20 g/day in other patients, with at least 2 alcohol free days per week)
Smoking cessation
Identification and treatment of commonly associated conditions (eg. sleep apnoea, depression)
Regular physical activity but bed rest during acute exacerbations
Vaccination – influenza and pneumococcal
* Action plans are helpful in identifying and treating exacerbations early in order to avoid hospitalisation. They are most suited to patients who understand their condition and treatment and can recognise worsening of their congestive symptoms. Clear instructions on what to do in response to a change in weight or worsening CHF symptoms are needed

creatinine increases by more than 20% from baseline. Renal function and electrolytes should be re-checked after 1 month and then 3–6 monthly. The most common reasons for discontinuation include cough, symptomatic hypotension, and renal or electrolyte disturbance. Day time hypotension may be avoided by giving the ACEI at night (Table 3).

Angiotensin receptor blockers have been shown to have similar improvements in mortality, hospitalisations and symptoms compared to ACEIs.<sup>2</sup> They should be considered in patients intolerant of

**Table 3. Target doses of ACEIs in heart failure<sup>7</sup>**

Ramipril	Perindopril	Enalapril	Fosinopril	Lisinopril	Trandolopril	Captopril	Fosinopril
10 mg/day	10 mg/day	20 mg bd	20 mg/day	30 mg/day	4 mg/day	25 mg tds	20 mg/day

**Table 4. Titration of beta blockers in heart failure<sup>7</sup>**

Week	Carvedilol	Bisoprolol	Nebivolol	Extended release metoprolol
0–2	3.125 mg bd	1.25 mg/day	1.25 mg/day	23.75 mg/day
2–4	6.25 mg bd	2.5 mg/day	2.5 mg/day	47.5 mg/day
4–6	12.5 mg bd	5 mg/day	5 mg/day	95 mg/day
6 onward	25 mg bd*	10 mg/day	10 mg/day	190 mg/day

\* In patients >85 kg, the carvedilol dose can be increased to 50 mg bd

abnormal remodelling and secondary mitral regurgitation. Pacing the left and right ventricles simultaneously, in order to resynchronise LV contraction, cardiac resynchronisation therapy (CRT) has been shown to improve symptoms, functional capacity, CHF hospitalisations and mortality. The criteria for recommending CRT are LV ejection fraction <35%, evidence of cardiac dyssynchrony (QRS duration >120 ms) and Class III/IV symptoms despite optimal medical therapy.<sup>2</sup>

### Digoxin

In patients with persistent symptoms, despite the above therapies, digoxin has been shown to improve symptoms and reduce hospitalisations, but has no effect on mortality.<sup>2</sup> Digoxin is particularly valuable when the patient also has atrial fibrillation. Small doses are recommended, eg. 62.5 µg/day or every 2–3 days in patients with renal impairment.

### Other drug therapies

Nitrates and hydralazine offer alternative vasodilatation in patients intolerant of ACEIs and ARBs. Nitrates are particularly useful in reducing nocturnal dyspnoea, pulmonary hypertension, myocardial ischaemia and peripheral oedema.<sup>7</sup> Isosorbide mononitrate may be commenced at 30 mg nocte, titrating up to 120 mg over 1–2 weeks. Nitrate patches are less well absorbed in patients with poor peripheral perfusion.

Amiodarone has not been shown to improve mortality, but may control ventricular arrhythmia and atrial fibrillation in CHF.<sup>5</sup> Monitoring for complications including thyroid dysfunction, pulmonary fibrosis, hepatic dysfunction, corneal deposits, peripheral neuropathy, photosensitivity and skin discolouration is important. Amiodarone should be initiated only by a cardiologist/specialist or in consultation with a specialist.

Warfarin is indicated in patients with CHF who have atrial fibrillation or cardiac thrombus.<sup>2</sup> Patients with ischaemic cardiomyopathy should receive aspirin.<sup>7</sup> There is no strong evidence however, for the use of anticoagulants or antiplatelets in patients with nonischaemic cardiomyopathy.

### Device therapies

An implantable cardioverter defibrillator may be an option for patients with an LV ejection fraction <35% for both primary and secondary prevention of ventricular arrhythmia, with a reduction in mortality in both of these settings.<sup>2</sup>

Cardiac dyssynchrony is seen in approximately one-third of CHF patients and leads to further impairment of LV function,

### Summary

Chronic heart failure carries a major health burden, with significant morbidity and mortality. Accurate diagnosis, treatment of reversible causes and institution of proven medical and device therapies are key facets of management. This involves regular follow up, appropriate initiation and titration of medications, referral to a consultant cardiologist as needed and liaison with multidisciplinary team members.

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### References

1. Australian Institute of Health and Welfare and the National Heart Foundation of Australia. Heart, stroke and vascular diseases – Australian facts 2004. Canberra: National Centre for Monitoring Cardiovascular Disease, 2004; p.140.
2. National Heart Foundation of Australia and the Cardiac Society of Australia and New Zealand (Chronic Heart Failure Guidelines Expert Writing Panel). Guidelines for the prevention, detection and management of chronic heart failure in Australia, 2006.
3. American Heart Association. Classification of Functional Capacity and Objective Assessment. Available at [www.americanheart.org/presenter.jhtml?identifier=4569](http://www.americanheart.org/presenter.jhtml?identifier=4569) [Accessed 8 November 2010].
4. Baldasseroni S, Opasich C, Gorini M, et al. Left bundle-branch block is associated with increased 1-year sudden and total mortality rate in 5517 outpatients with congestive heart failure: a report from the Italian network on congestive heart failure. *Am Heart J* 2002;143:398–405.
5. Jessup M, Abraham WT, Casey DE, et al. 2009 focused update: ACCF/AHA Guidelines for the Diagnosis and Management of Heart Failure in Adults: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines: developed in collaboration with the International Society for Heart and Lung Transplantation. *Circulation* 2009;119:1977–2016.
6. Thohan V, Little WC. Is a higher blood pressure better in heart failure? *Heart* 2009;95:4–5.
7. Cardiovascular Writing Group. Therapeutic guidelines: Cardiovascular. Version 4. North Melbourne: Therapeutic Guidelines Ltd, 2008.

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