Cardiology series — 4

Management of heart failure

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Summary

- Heart failure is a complex clinical syndrome, with diagnosis based on typical symptoms, signs and supportive investigations.
- Investigations may include an electrocardiogram and chest x-ray, but echocardiography is the definitive test. Plasma B-type natriuretic peptide levels may also be useful in diagnosis among patients with breathlessness, particularly as a rule-out test.
- Mainstay therapy for heart failure comprises lifestyle modification, pharmacotherapy and referral to a multidisciplinary heart failure program.
- Drug therapies focused on blockade of key activated neurohormonal systems are well established in systolic heart failure. First-line pharmacotherapy consists of angiotensin-converting enzyme (ACE) inhibitors (or angiotensin receptor blockers if the patient is intolerant to ACE inhibitors) and β-blockers. These medications should be commenced at a low dose and slowly up-titrated to the maximal tolerated dose.
- In selected patients, device-based therapies are a useful adjunct in systolic heart failure. The most common of these are implantable cardioverter defibrillators and cardiac resynchronisation therapy. Most patients will receive both, as the indications overlap.
- Multidisciplinary approaches, including involvement of the patient's general practitioner, are strongly recommended.

Heart failure is a common condition affecting about half a million Australians. It is associated with high morbidity and mortality, frequent hospitalisation and massive cost to the health care system.[1] Although epidemiological data on heart failure in Australia are scant, there is some evidence that pharmacological and other therapies have made inroads into reducing hospitalisation rates.[2]

Heart failure manifests in many forms, including acute decompensation, frequently resulting in hospital admission; chronic systolic heart failure (ie, impaired contractile function); and heart failure with preserved ejection fraction (HFPEF), an impairment of relaxation. There is also a subclinical population who have evidence of systolic or diastolic dysfunction but have not yet developed clinical symptoms. This group is at high risk of developing symptomatic heart failure and is therefore a target for early detection and preventive strategies.

Here, we focus on the latest clinical information regarding heart failure management, particularly as it pertains to the general practitioner. The recommendations in this review closely follow those of the most recent National Heart Foundation of Australia and Cardiac Society of Australia and New Zealand *Guidelines for the prevention, detection and management of chronic heart failure in Australia*.[1]

Aetiology and diagnosis of heart failure

The aetiology of heart failure is most commonly that of an ischaemic event, primarily affecting the left ventricle.[3] Non-ischaemic dilated cardiomyopathy may be idiopathic or secondary to hypertension, alcohol misuse, inflammation or drugs such as anthracyclines and trastuzumab. A small proportion of patients may have valvular heart disease, chronic arrhythmia, thyroid disease or HIV as the aetiological factor. Peripartum cardiomyopathy is a not infrequent presentation in young women with heart failure.

Diagnosis of heart failure is often, but not always, made during a hospital presentation with new or worsened symptoms (see case study in Box 1). Diagnosis is based on typical signs and symptoms, together with objective testing. A beneficial symptomatic response to heart failure therapies may also support the diagnosis.

Symptoms of heart failure are multiple and overlap with many other conditions, especially in older people in whom heart failure is most prevalent. Thus, symptoms such as exertional dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, fatigue and weakness have poor sensitivity and specificity. Similarly, signs of fluid retention, sympathetic activation (eg, tachycardia) and cardiac enlargement are supportive of the diagnosis, but not conclusive.

Diagnostic testing will usually include an electrocardiogram (ECG), chest x-ray and transthoracic echocardiography. The ECG is sensitive but very non-specific; a normal ECG virtually rules out heart failure.[4] A chest x-ray may demonstrate evidence of cardiac enlargement with or without pulmonary vein redistribution and upper lobe diversion.

The echocardiogram is the definitive test in the diagnosis of heart failure. In Australia, with its large rural and remote population, access to this procedure is an issue. Echocardiography provides information on ventricular size and function, as well as the presence of prior myocardial infarction, manifest as areas of hypocontractility. This information can be very useful in determining whether the underlying aetiology has an ischaemic or non-ischaemic basis. In addition, assessment of valvular structure and function, pulmonary pressures, and presence or absence of pericardial disease may be of use in guiding therapy as well as assisting in diagnosis. Newer echocardiographic techniques, such as tissue Doppler imaging, are of particular relevance in diagnosing HFPEF,[5] which is nearly always a diagnosis of exclusion.

While no single test is pathognomonic of heart failure, the use of plasma levels of B-type natriuretic peptide

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1 Case study of a patient with heart failure

Presentation

A 61-year-old man presented to hospital in 2005 with fatigue and increasing shortness of breath on less than ordinary exertion (New York Heart Association [NYHA] Class III symptoms). He had a longstanding history of hypertension but was otherwise well. He was taking an angiotensin-converting enzyme (ACE) inhibitor (enalapril 10 mg once daily) and a β-blocker (atenolol 50 mg once daily). On examination, he was found to have significant fluid overload, manifest as bibasal crackles, ascites and pitting oedema of the lower legs. His blood pressure was 127/78 mmHg and his heart rate was 86 beats/min. Renal function was normal, with an estimated glomerular filtration rate of 78 mL/min/1.73 m^2. His B-type natriuretic peptide plasma level was markedly elevated, at 1235 pg/mL (cut-off for heart failure diagnosis, >100 pg/mL). An electrocardiogram showed sinus rhythm and a widened QRS interval (163 ms) but no ischaemic changes. Echocardiography demonstrated a global reduction in left ventricular systolic function, with a left ventricular ejection fraction (LVEF) of 29%. A provisional diagnosis of systolic heart failure was made.

Initial management

Loop diuretics were introduced, initially intravenously and then orally, to clear excess fluid. Once the patient was euvolaemic, the dose of enalapril was increased to 10 mg twice daily, and he was discharged. He commenced a heart failure management program as an outpatient, which included education on dietary salt and alcohol restriction, an exercise program and referral to the nearest specialised heart failure clinic. At the clinic, atenolol was changed to a heart-failure specific β-blocker, carvedilol, at an initial dose of 6.25 mg twice daily, increased to 12.5 mg twice daily after 2 weeks. The patient underwent exercise stress testing and coronary angiography to exclude underlying ischaemia, and results of both were normal.

Current status

There was significant symptomatic improvement after the above measures. However, the patient remained in NYHA Class II, and his most recent LVEF on echocardiography was 32%. His QRS interval remained prolonged and his heart rate slightly elevated at 77 beats/min (sinus rhythm), despite a further increase in carvedilol to the target dose of 25 mg twice daily.

Management options

A key issue is what the next steps in this patient's management should be. There are many evidence-based treatment options that should have a favourable impact on mortality and morbidity, including addition of an angiotensin receptor blocker, addition of a mineralocorticoid receptor antagonist (MRA), cardiac resynchronisation therapy, addition of the direct sinus node inhibitor ivabradine, and addition of marine-based omega-3 fatty acids.

The decision was made to initially add spironolactone, an MRA, as this drug class has been found to have a favourable stand-alone impact on all-cause mortality. This was introduced with careful ongoing scrutiny of postural symptoms, blood pressure (lying and standing), renal function and serum potassium status. Further options, as outlined above, remain viable depending on the patient's symptomatic response to this add-on strategy. ◆

(BNP) is another newer investigation that can assist in diagnosis. This test may be used for distinguishing heart failure from non-heart failure causes of shortness of breath in patients presenting in the emergency department setting.[6] A recent meta-analysis supported the utility of BNP in diagnosing heart failure in the emergency department, and found a reduction in hospital length of stay and requirement for intensive care support with this early diagnosis.[7] However, BNP levels can be affected by other conditions associated with heart failure; for example, atrial fibrillation may increase BNP levels. Advanced age and female sex also increase plasma BNP levels, whereas obesity leads to a reduction in BNP levels. Thus, establishing normative ranges for BNP has been somewhat fraught. Furthermore, there is a "grey zone" in BNP levels, where the result is not conclusive. Nevertheless, a low BNP level has a very high negative predictive value, making it an extremely useful rule-out test for heart failure.[6] Specifically, the commonly used BNP cut-off value of 100 pg/mL has 90% sensitivity and 76% specificity as a rule-out test.[6]

Current therapies for heart failure

The key aims of therapy for heart failure are to relieve symptoms and prolong survival (Box 1). Underpinning all therapies are lifestyle measures that have been found to be useful in supporting the patient. These include exercise (usually a graded exercise program, initially under the supervision of a physiotherapist or heart failure nurse), dietary salt restriction, alcohol restriction and weight loss in overweight patients.[1] Patients should be encouraged to consume an adequate but restricted volume of, as far as possible, sodium-free fluid. This is particularly critical in patients who have low serum sodium levels, which is often seen in the setting of heart failure due to activation of neurohormones such as arginine vasopressin. Appropriate treatment of commonly associated conditions, such as hypertension, arrhythmia, sleep apnoea, depression, anaemia and iron deficiency, is also critical in the optimal management of patients with heart failure.[1]

Pharmacological therapies

Specific drug therapy for heart failure focuses on improving prognosis (using inhibitors of the renin–angiotensin–aldosterone system [RAAS] and sympathetic nervous system [SNS]) and achieving and maintaining euvolaemia to improve symptom status (using diuretics).

Activation of the RAAS and SNS has been found to be associated with pathogenesis and progression of the heart failure disease process, as well as with reduced survival.[8,9] Blockade of these systems has revolutionised the management of heart failure (Box 2). Angiotensin-converting enzyme (ACE) inhibitors (or angiotensin receptor blockers [ARBs] in those unable to tolerate ACE inhibitors) and β-blockers are the cornerstone of drug therapy for systolic (ie, low ejection fraction) heart failure. These agents should be initiated at low doses and up-titrated to the target or maximal tolerated dose, as per guidelines.[1] For both drug classes, blood pressure (lying and standing), heart rate, renal function and potassium levels should be regularly monitored. Particularly in patients receiving ACE inhibitors for the first time, and especially those with a history of hypertension, careful and early monitoring of renal function is imperative because of the small but real possibility of bilateral renal artery stenosis. In this clinical context, administration of ACE inhibitors (and ARBs) may lead to acute renal failure.

β-Blockers have their own specific side effects.[23] Some agents (eg, carvedilol and nebivolol) also possess vasodilator properties, making them theoretically more difficult to initiate in patients with borderline blood pressure levels. However, in studies of patients with severe heart failure (eg, COPERNICUS[15]), carvedilol was extremely well tolerated, even with a baseline systolic blood pressure cut-off of 85 mmHg. Conversely, vasodilation may offset the tendency toward early worsening of heart failure. Highly selective β_1-blocking agents (eg, nebivolol, bisoprolol) may be preferred in the setting of concomitant chronic obstructive pulmonary disease (COPD). COPD is not an absolute contraindication to β-blocker therapy in heart failure, provided that clinically significant reversible airflow obstruction is not present. This can be readily detected on lung function testing using pre- and post-bronchodilator spirometry.

Aldosterone receptor antagonists, in addition to background ACE inhibitor and β-blocker therapy, have been found to be beneficial across all severities of systolic heart

2 Major clinical trials underpinning the use of recommended pharmacological therapies in systolic chronic heart failure

Drug class and study	Study therapy (target dose v placebo)	Patient population	Primary end point/key findings
ACE inhibitors			
SOLVD Prevention[10]	Enalapril 10 mg twice daily	NYHA Class I: 67%, II: 33%	36% ↓ HF hospitalisation
SOLVD Treatment[11]	Enalapril 10 mg twice daily	NYHA Class II–III: 90%	16% ↓ mortality
β-blockers			
US Carvedilol HF[12]	Carvedilol 25 mg twice daily	NYHA Class II: 53%, III: 44%	65% ↓ mortality
CIBIS-II[13]	Bisoprolol 10 mg once daily	NYHA Class III: 83%, IV: 17%	34% ↓ mortality
MERIT-HF[14]	Metoprolol CR/XL 200 mg once daily	NYHA Class II: 41%, III: 55%, IV: 4%	34% ↓ mortality
COPERNICUS[15]	Carvedilol 25 mg twice daily	NYHA Class IV*	35% ↓ mortality
SENIORS[16]	Nebivolol 10 mg once daily	Aged ⩾ 70 years NYHA Class I: 3%, II: 56%, III: 39%, IV: 2%	14% ↓ mortality/CV hospitalisation
Aldosterone receptor antagonists			
RALES[17]	Spironolactone 50 mg once daily	NYHA Class II: 0.5%, III: 70.5%, IV: 29%	30% ↓ mortality
EPHESUS[18]	Eplerenone 50 mg once daily	Post-MI LVSD	15% ↓ mortality
EMPHASIS-HF[19]	Eplerenone 50 mg once daily	NYHA Class II: 100%	37% ↓ CV death/HF hospitalisation
Angiotensin receptor blockers			
Val-HeFT[20]	Valsartan 160 mg twice daily	NYHA Class II: 62%, III: 36%, IV: 2%	13% ↓ morbidity/mortality
CHARM-Alternative[21]	Candesartan 32 mg once daily	Intolerant to ACE inhibitors NYHA Class II: 47%, III: 49%, IV: 4%	23% ↓ CV death/HF hospitalisation
CHARM-Added[22]	Candesartan 32 mg once daily	NYHA Class II: 24%, III: 73%, IV: 3%	15% ↓ CV death/HF hospitalisation

ACE = angiotensin-converting enzyme. NYHA = New York Heart Association. HF = heart failure. CR/XL = controlled release/extended release. CV = cardiovascular. MI = myocardial infarction. LVSD = left ventricular systolic dysfunction. * NYHA Class was not specified, but all patients had severe chronic heart failure. ◆

failure.[24] Survival benefits have been observed in patients immediately after myocardial infarction,[18] in patients with mild symptoms of systolic heart failure,[19] and in those with advanced symptoms[17] (Box 2). For this reason, the most recent Australian guideline update[1] recommended that aldosterone receptor antagonists be added to background ACE inhibitor and β-blocker therapy in all patients with systolic chronic heart failure who remain symptomatic despite the ACE inhibitor and β-blocker therapy. However, as combining RAAS blockers puts patients at increased risk of hyperkalaemia, hypotension and renal impairment, they should be carefully monitored for these side effects, especially during commencement of aldosterone-blocking therapy in patients with borderline hypotension, renal impairment or diabetes mellitus. In contrast, no benefit with either RAAS blockers or SNS blockade has yet been established for HFPEF.

Diuretics are primarily used to relieve symptoms through achievement and maintenance of euvolaemia. They have not been shown to provide prognostic benefit.[25]

Digoxin is the mainstay therapy to control ventricular response in patients with heart failure and atrial fibrillation. Its use in sinus rhythm has waned, particularly after the publication of the Digitalis Investigation Group (DIG) study, which demonstrated no overall prognostic benefit. However, further analysis of the DIG study suggested that patients receiving relatively small doses within a serum digoxin range of 0.5–0.8 ng/mL had improved survival, although this survival advantage was largely confined to men.[26] Caution is needed with the use of certain concomitant medications because of the potential for pharmacodynamic interactions (eg, β-blockers leading to atrioventricular block) or pharmacokinetic interactions (eg, concomitant renal impairment leading to digoxin toxicity).

The combination of nitrates and hydralazine has been used for many years as an alternative to ACE inhibitors. However, ARBs are preferred in patients with intolerance to ACE inhibitors, as this drug class has been shown to be of benefit (Box 2).[21] Amiodarone is commonly used in patients with heart failure and arrhythmias but has been found not to provide any specific prognostic benefit in these patients; thus its use is limited to treatment of the underlying arrhythmia.

The use of antiplatelet and anticoagulant therapy in heart failure is somewhat controversial. For patients with known ischaemic heart disease, aspirin and other antiplatelet agents should be continued. Warfarin should be reserved for patients with chronic heart failure and atrial fibrillation, although it is often prescribed (without supporting data) for patients with marked dilatation of their left atrium and/or ventricle because of concerns about an increased risk of thromboembolism.

Device-based therapies

The two main device-based therapies used in patients with heart failure are the implantable cardioverter defibrillator (ICD) and cardiac resynchronisation therapy (CRT). Key studies supporting their use are summarised in Box 3.

ICDs are indicated to reduce sudden death in high-risk patients — about half of patients with systolic heart failure die a sudden, presumed arrhythmic, death. Implantation of these devices is currently recommended for patients with a low ejection fraction (ie, ⩽ 30% after myocardial infarction, irrespective of severity of symptoms, or ⩽ 35% in other patients).[1] In the Multicenter Automatic Defibrillator Implantation Trial II (MADIT-II),[30] which established these criteria, there was a 5.6% absolute risk reduction in all-cause death over the mean 20 months of follow-up.

ICDs are almost invariably implanted in patients who also require CRT, as the indications overlap. CRT is indicated for patients with New York Heart Association (NYHA) Class III–IV symptoms of heart failure who are in sinus rhythm and have evidence of so-called ventricular dyssynchrony (ie, left and right sides of the heart are not contracting in a synchronous manner). Measurement of the QRS interval on ECG has been taken to be a rough surrogate for ventricular synchrony, with a QRS duration of ⩾ 120 ms generally indicative of dyssynchrony. However, many patients with a prolonged QRS interval do not have dyssynchrony, and many with a normal QRS interval do have dyssynchronous ventricles. Because of this, echocardiographic-based criteria for dyssynchrony have been proposed to improve the sensitivity and specificity of the diagnosis, as well as potentially the response rates to therapy (currently about 70% of patients derive clinical benefit with CRT). In patients who do respond to CRT, early and marked symptomatic benefit can be seen. Studies such as the Cardiac Resynchronization — Heart Failure (CARE-HF) trial have shown a stand-alone prognostic benefit with CRT additional to background pharmacological therapy in NYHA Class III–IV patients who meet the above criteria.[27] Absolute mortality risk was substantially reduced (by 7.1% over 2 years) in the CARE-HF trial.

More recently, studies of patients with less severe symptoms (NYHA Class I-II) have also demonstrated benefit with CRT,[28,29] but the benefit was mostly confined to patients with a QRS interval ⩾150 ms, hence the revised Australian guidelines reflecting this.[1] Given the lower overall risk in these patients, absolute risk reduction was less than that observed in the CARE-HF trial.

Newer therapies

Some newer approaches to treating heart failure have been developed, and these may provide ancillary benefit to the standard therapies described above.

In the GISSI Heart Failure study, use of fish oils (1 g/day of n-3 polyunsaturated fatty acids) was shown to provide a small but significant outcome benefit compared with placebo on the study's primary end point of death, and time to death or admission to hospital for cardiovascular reasons in patients with symptomatic, primarily systolic, heart failure.[32] In absolute terms, 56 patients needed to be treated for a median duration of 3.9 years to avoid one death. Side effects were minimal to non-existent.

Ivabradine, an agent that slows heart rate (additional to background β-blockade) has shown clinical benefit in the SHIFT study.[33] Ivabradine is guideline-recommended,[1] and has been approved by the Australian Therapeutic Goods Administration.

Anaemia and iron deficiency are relatively common in patients with heart failure. Definitive evaluation of raising haemoglobin levels using erythropoietin-stimulating agents in patients with anaemia and heart failure is ongoing.[34] The Ferinject Assessment in Patients with Iron Deficiency and Chronic Heart Failure (FAIR-HF) trial showed that regular administration of intravenous ferric carboxymaltose improved symptom status and exercise tolerance in iron-deficient patients with heart failure.[35] This led to the Australian guideline recommendation that iron deficiency should be looked for and corrected in patients with heart failure.[1]

3 Major clinical trials underpinning the use of recommended device therapies in systolic chronic heart failure

Device class and study	Patient population	Primary end point/ key findings
Cardiac resynchronisation therapy		
CARE-HF[27]	NYHA Class III: 94%, IV: 6% LVEF ⩽ 35% QRS interval ⩾ 120 ms LVEDD ⩾ 30 mm	37% ↓ mortality/CV hospitalisation 52% ↓ HF hospitalisation
MADIT-CRT[28]	NYHA Class I: 15%, II: 85% LVEF ⩽ 30% QRS interval ⩾ 130 ms	34% ↓ mortality/HF hospitalisation
RAFT[29]	NYHA Class II: 80%, III: 20% LVEF ⩽ 30% QRS interval ⩾ 120 ms	25% ↓ mortality/HF hospitalisation 25% ↓ all-cause mortality
Implantable cardioverter defibrillator		
MADIT-II[30]	Prior MI LVEF ⩽ 30%	31% ↓ mortality
SCD-HeFT[31]	NYHA Class II: 70%, III: 30% LVEF ⩽ 35%	23% ↓ mortality

NYHA = New York Heart Association. LVEF = left ventricular ejection fraction. LVEDD = left ventricular end-diastolic dimension. CV = cardiovascular. HF = heart failure. MI = myocardial infarction. ◆

Correcting structural heart disease

Many patients with heart failure have additional concomitant cardiac disease. Amelioration of cardiac disease may relieve symptoms and improve the patient's overall heart failure status. All patients with heart failure should be investigated for ischaemic heart disease, ultimately by coronary angiography. Correction of coronary disease using surgical approaches has not been shown to provide clinical benefit compared with optimal medical therapy overall in the STICH study.[36,37]

Control of chronic arrhythmias is another important component of heart failure therapy. In particular, the ventricular response to atrial fibrillation should be controlled, ideally to < 70 beats/min. Drugs such as β-blockers, digoxin and amiodarone may be useful in this regard.

Valvular heart disease should be investigated using echocardiography. Mitral regurgitation is commonly observed in heart failure, often due to dilation of the valve apparatus secondary to ventricular enlargement as part of the ongoing remodelling process. New percutaneous techniques have been developed to non-invasively minimise mitral regurgitation in the hope this will help minimise or even reverse remodelling, but these remain largely experimental. Similarly, percutaneous approaches to treating aortic stenosis are under active evaluation.

Transition of care to the community

Ambulatory care heart failure programs

Australian guidelines recommend that patients hospitalised with an exacerbation of heart failure be referred to a heart failure program after discharge.[1] This can be in the form of a heart failure home-visit program, heart failure exercise program, cardiologist outpatient clinic, or nurse-led or nurse practitioner clinic. Multidisciplinary ambulatory care programs targeting patients with heart failure reduce hospital readmission and mortality.[38] These pro-

4 Main objectives of ambulatory care heart failure programs

- Increased access to a heart failure specialist
- Development of a heart failure management plan, incorporating management of comorbidities
- Early detection and management of signs and symptoms of an exacerbation of heart failure
- Monitoring and measuring of left ventricular function
- Assessment and referral to other health care professionals, such as an electrophysiologist for implantation of a cardioverter defibrillator
- Prescription and optimisation of evidence-based pharmacotherapy, including a flexible diuretic regimen
- Provision of patient and carer education about heart failure, self-management strategies, lifestyle modifications and psychosocial support
- Assessing and improving patient adherence to medications and the management plan
- Advance care planning ◆

grams ideally involve a cardiologist, heart failure nurse, pharmacist, physiotherapist, dietitian, GP and psychologist. The main objectives of these programs are listed in Box 4. Ambulatory care heart failure programs are accessible at most tertiary hospitals and many community health centres in Australia.[39]

The frequency of follow-up outpatient appointments depends on the patient's clinical status, as well as his or her ability to self-care and manage the heart failure. Patients who require optimisation of pharmacotherapy or further diagnostic investigations, who have been recently hospitalised, who are exhibiting signs and symptoms of an acute exacerbation or who require further assessment for device therapy or heart transplantation will need to be seen more often (even weekly) in a heart failure outpatient clinic or program. Patients with stable heart failure usually only require 3–6-monthly monitoring of their clinical status but should be referred to a heart failure exercise program. Patient education is vital and is usually provided by the heart failure nurse. Patient education provided to patients and carers should always be based on the *Living well with chronic heart failure* booklet developed by the National Heart Foundation of Australia.[40]

Nurse-led or nurse practitioner clinics

The focus of nurse-led clinics is primarily optimisation of pharmacotherapy and education. Patient and carer education focuses on disease, diet, exercise, medications, lifestyle issues and self-management strategies. Titration of medications may be done by a nurse practitioner or a heart failure nurse under the supervision of a cardiologist or through an approved titration protocol. A meta-analysis of nurse-led clinics has found an improvement in patient outcomes, including a reduction in hospital admissions and mortality.[41]

Telemonitoring programs

A growing area in the management of heart failure is the use of telemonitoring, particularly for patients in rural and remote areas who may not have access to ambulatory care heart failure programs. Telemonitoring involves the transmission of clinical data, such as blood pressure, weight and heart rate, through digital broadband, wireless, bluetooth or satellite technology.[42] The data are transmitted to a hospital or health care facility where they are reviewed by a cardiologist or heart failure nurse, and appropriate treatment is implemented. Although telemonitoring is ideal for patients in rural and remote areas, it is not advocated for patients in metropolitan areas who have access to ambulatory care heart failure programs. The benefits of a face-to-face interaction have been shown to far outweigh the benefits of telemonitoring.[43] Meta-analyses and systematic reviews of telemonitoring programs for heart failure have found that they reduce hospital admission and mortality.[42,44] However, two subsequent large randomised controlled trials of telemonitoring found no significant effect on hospitalisation or mortality,[45,46] raising questions about its efficacy. Further research is required.

Shared care model

Shared health care is a model of health care delivery in the primary care setting that involves collaboration among practitioners of different disciplines or with different skills and knowledge. For patients with heart failure, an ideal shared care model would involve collaboration between the GP and cardiologist, an allied health professional and/or heart failure nurse. The success of a shared care model is influenced by the quality of collaborative working relationships, as frequent communication between team members is essential. Shared care involving a cardiologist and GP for patients with heart failure has been shown to reduce mortality by 20% when compared with follow-up by a GP alone.[47]

There is also scope for general practice nurses to improve outcomes in primary and secondary prevention of heart failure in the community. This may be through an absolute risk clinic, where general practice nurses assess and educate patients about their risk of cardiovascular disease using the absolute risk tool recommended by the National Heart Foundation of Australia.[48] Patient review by the GP would be short, focusing on prescription of evidence-based medications such as antihypertensive agents or statins. Similarly, general practice nurses could also run a heart failure clinic where they review patients and provide education about self-management. For these clinics to be successful, general practice nurses would require professional development in these areas.

Conclusions

Despite recent advances in its management, heart failure remains a significant health care issue in Australia. Given the size of the problem and the high cost of newer therapies, it is imposing a significant cost burden on the Australian health care system. How best to manage scarce health care resources represents a major ongoing challenge in the management of this condition.

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