Investigation and management of chronic heart failure

Chronic heart failure severely impairs patients' quality of life and is a highly lethal disease. While the outlook is bleak for many patients, medical therapy, specialist review and ongoing assessment can help control symptoms, reduce rehospitalisations and improve survival.

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Chronic heart failure (CHF) is an increasingly prevalent disease affecting 300,000 to 500,000 Australians. Each year, 30,000 patients are newly diagnosed with this lethal condition. Data from 2001 indicate that more than 2% of Australians are affected by CHF and that number climbs to 10% of the population over the age of 75 years.[1]

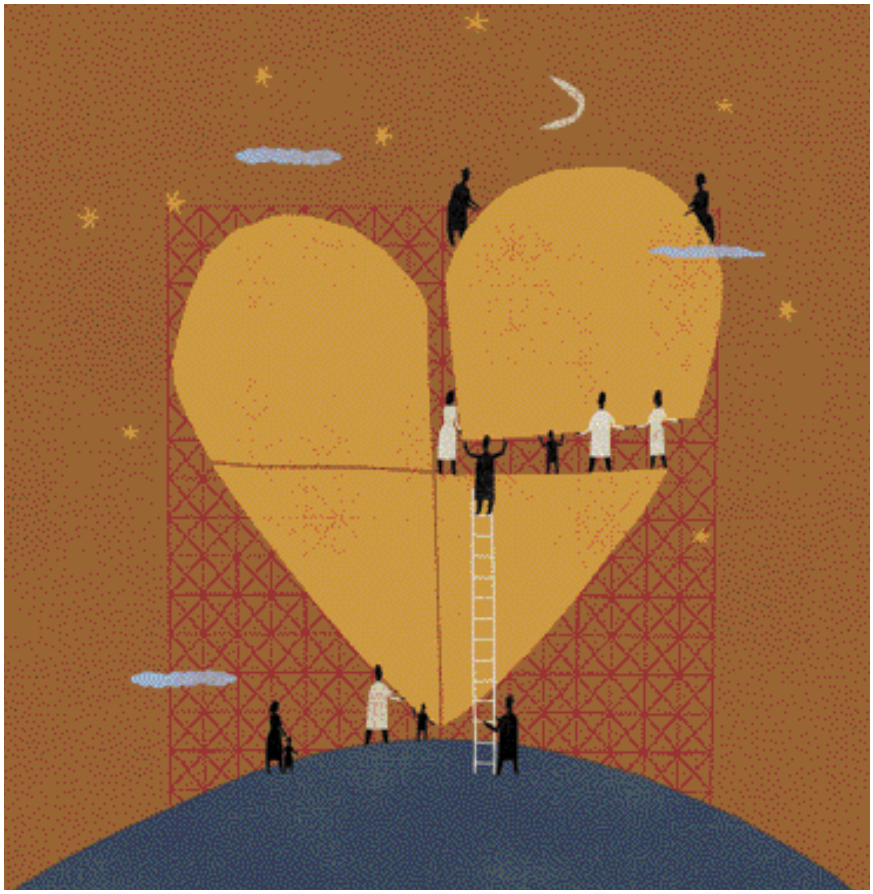
A number of concurrent factors are contributing to the growing numbers of Australians affected by CHF. The ageing Australian population and the increasing number of people surviving myocardial infarction (MI), cardiac arrest, coronary artery bypass grafting and cardiac valve surgery are major contributors. Improvements in medical therapy and the wider use of implantable defibrillators are also contributing to the extended survival of patients diagnosed with the disease.

CHF has a significant impact on quality of life, which worsens as the disease progresses. It is a costly disease because it leads to recurrent hospitalisations and has a higher mortality than many cancers (Table 1). Medications, particularly angiotensin II converting enzyme (ACE) inhibitors and beta blockers, have improved symptom management and survival with the disease. In addition, widespread use of these medications combined with more acute diagnosis have contributed to the longevity of patients with CHF. Long-term outcomes, however, remain poor, with approximately 65% of patients dying within five years of being diagnosed with CHF.[1] Table 2 lists the common causes of CHF.

It is preferable that patients who are suspected of having CHF are reviewed at least once by a cardiologist. Generally, a consultation with a

IN SUMMARY

- **Each year 30,000 Australians are newly diagnosed with CHF.**
- **Approximately 50% of patients die within five years of being diagnosed with CHF.**
- **CHF is a costly, debilitating disease and has a higher mortality than many cancers.**
- **It is preferable that all patients who are suspected of having CHF are reviewed at least once by a cardiologist. Ongoing assessment is important for patients with CHF to maintain their quality of life.**
- **Echocardiography is the gold standard for diagnosing CHF. Plasma B-type natriuretic peptide is emerging as a useful diagnostic adjunct.**
- **ACE inhibitors, beta blockers and spironolactone have been shown to improve survival in CHF while diuretics improve symptoms and reduce hospitalisations.**
- **A range of nonpharmacological interventions including exercise, patient support programs and cardiac devices play an important role in the management of CHF.**



PHOTOLIBRARY

cardiologist is beneficial because it can:

- help guide therapy
- improve the patient's prognosis
- improve the patient's quality of life
- reduce the chance of hospital admission.

A consultation with a cardiologist may also identify patients who would benefit from interventions such as coronary revascularisation, biventricular cardiac pacing or the implantation of an automated defibrillator.

Definition of CHF

CHF is a complex clinical syndrome with typical symptoms which may occur on rest or on effort. Frequently, but not exclusively, these symptoms are characterised by objective evidence of an underlying structural abnormality or cardiac dysfunction that impairs the ability of the ventricles to fill with or eject blood (particularly during exercise). A diagnosis of CHF may be further strengthened by a beneficial clinical response to treatment or treatments directed towards the amelioration of CHF symptoms.[1]

Table 1. Comparison of CHF and cancer survival rates (%)[1]

	Survival after diagnosis		
	1 year	2 year	3 year
Breast cancer	88	80	72
Prostate cancer	75	64	55
Colon cancer	56	48	42
CHF	67	41	24

Signs and symptoms

CHF is usually associated with symptoms including exertional dyspnoea, orthopnoea and paroxysmal nocturnal dyspnoea. Patients also report feeling fatigued and can experience anorexia. Clinical signs that may be observed in patients with CHF include:

- elevated jugular venous pressure (greater than 4 cm above the sternal angle)
- basal chest crepitations
- pitting oedema of the lower limbs or sacrum
- recent weight gain – greater than 1.5 kg in a 48 hour period or associated with abdominal bloating or lower limb oedema or breathlessness
- the presence of a third heart sound
- relative tachycardia – resting heart rate greater than 90 beats/minute
- hypotension – systolic blood pressure below 110 mmHg with no antihypertensive treatment
- hepatic congestion – liver palpable below the costal margin and measuring greater than 15 cm in length on percussion
- ascites.

Investigations

Echocardiography

Echocardiography is the gold standard investigation for CHF. It allows for the assessment of left ventricular size and function and the functionality of the cardiac valves. It is also used to follow the patient's progress and assess his or her response to therapy. As many as 30% of patients with heart failure may have preserved left ventricular systolic function.[2]

This may alter the management of the patient or even lead to an alternative diagnosis as the cause of the patient's symptoms. All patients who present with shortness of breath should have an echocardiogram to rule out CHF.[2]

Chest x-ray

A chest x-ray may be useful for assessing pulmonary congestion and the presence of any pleural effusions, but compared to echocardiography, it is considered a less accurate diagnostic tool for assessing a patient's cardiac size.[2]

Electrocardiogram

An electrocardiogram (ECG) is useful for assessing a patient's cardiac rate, rhythm and the presence of prior MI. The presence of pathological Q waves, delayed progression of R waves in the precordial leads or early R wave progression in the precordial leads should all be noted and may indicate CHF. Left bundle branch block (LBBB) can also be detected with an ECG. Detection of LBBB is important because it may be an indication for using biventricular pacing.

Laboratory tests

Electrolytes

Measurement of electrolytes will reveal any renal dysfunction and/or an abnormal sodium or potassium level, information which is needed to guide diuretic therapy in CHF.

Liver function tests

Hepatic congestion may be present and suggested by elevated gamma glutamyl transpeptidase (GGT) and alkaline phosphatase (ALP) levels.

Full blood count

A full blood count may reveal anaemia, which is a precipitator of deterioration in CHF symptoms. An elevated white cell count may indicate an infection, which can significantly compromise a patient with CHF.

Plasma B-type natriuretic peptide

A rise in a patient's plasma B-type natriuretic peptide (BNP) level occurs when there is left ventricular wall stretch and volume overload. A high BNP level may predict CHF as the cause of dyspnoea and also correlates with the quality of ventricular function and the patient's prognosis. BNP is useful because it can help determine whether a patient's breathlessness is due to CHF or a respiratory problem.[3]

Making a diagnosis

History taking should include questions such as whether the patient has experienced shortness of breath on exertion, the distance he or she can walk on flat ground before having to stop and whether he or she has experience experienced orthopnoea. Other key questions that should be elicited include whether the patient has chest, arm, neck or back pain on exertion and whether he or she experiences palpitations, ankle swelling, bloating or weight gain and/or dizziness.

The poorer the patient's exercise capacity, ankle swelling and fatigue, the more severe his or her grade of CHF is likely to be. The absence of chest pain does not exclude prior ischaemic heart disease as a cause of the patient's CHF. Where a diagnosis of ischaemia as a cause of left ventricular dysfunction would make a difference to the patient's management, he or she should be investigated to exclude myocardial ischaemia as a cause of their symptoms and any left ventricular dysfunction. A prior history of myocardial infarction, coronary bypass grafting, coronary intervention and valvular heart disease makes the diagnosis easier. A number of conditions can mimic CHF and these need to be ruled out (Table 3).

Medical therapy in CHF

First-line drug therapies

The first line pharmacotherapies used in CHF include ACE inhibitors, beta blockers and, for short-term symptom

Table 2. Causes of CHF

Ischaemic heart disease (up to 75% of cases)
Hypertension
Viral cardiomyopathy
Familial cardiomyopathy
Valvular heart disease
Alcoholic cardiomyopathy
Diabetic cardiomyopathy
Hyperthyroidism
Hypothyroidism
Systemic illnesses – e.g. amyloidosis, sarcoidosis, scleroderma, haemochromatosis, etc)
Idiopathic causes

Table 3. Conditions that can mimic heart failure

Lung disease – e.g. COPD, interstitial lung disease
Pulmonary thromboembolic disease
Pulmonary infiltrative disease
Pleural disease
Primary pulmonary hypertension
Congenital heart disease
Anaemia
Obesity
Depression
Ankle oedema secondary to use of calcium channel blockers
Pericardial disease – e.g. pericardial constriction, pericardial restriction
Cardiac arrhythmias – e.g. atrial fibrillation, frequent ventricular ectopic beats
Nutritional deficiencies – e.g. thiamine deficiency
Cushing's disease
Addison's disease
Hypothyroidism or hyperthyroidism

Table 4. Commonly used drugs in CHF

First-line treatments

ACE inhibitors
Beta blockers
Loop diuretics

Second-line treatments
(if the patient is still symptomatic)

Spironolactone
Angiotensin II receptor antagonists
Digoxin
Nitrates
Hydralazine
Amiodarone

management, diuretics. The two main goals of treatment are a systolic blood pressure of 105 to 110 mmHg and a pulse of 55 to 60 beats per minute. Table 4 lists commonly used drugs in CHF.

ACE inhibitors

ACE inhibitors should be considered mandatory therapy in the treatment of all grades of CHF and should be uptitrated to the highest tolerated dose.[4] They reduce morbidity and mortality in patients with all grades of CHF by 24 to 28%. These drugs both reduce cardiovascular events and improve survival in patients with impaired left ventricular function (following MI) by approximately 20%.

Evidence also indicates that ACE inhibitors can reduce the risk of MI among patients with CHF by 20%, cardiovascular death by 26% and overall mortality by 16%. For patients with stable coronary artery disease, ACE inhibitors lead to a 24% reduction in the risk of MI.[4]

Patients taking ACE inhibitors need to have their potassium level monitored and their renal function evaluated one to two weeks after treatment is initiated, followed by a review of both measures one to two months later. If renal function and potassium levels are within acceptable limits, these parameters should then be checked every three to six months thereafter. Should a patient's potassium level rise above 5.5 mmol/L or his or her renal function deteriorates as a result of treatment with an ACE inhibitor, use of these drugs should be ceased. It is also important to note that up to 20% of patients can develop a cough with ACE inhibitor treatment.

Taking ACE inhibitors as a single daily dose improves compliance and taking them at night reduces the risk of daytime hypotension. Night-time administration carries the additional benefit of maximising protection during the 3 am to 8 am period, when most patients have major arrhythmias or MIs.

Target doses (i.e. maximum doses that have been shown to lead to a significant outcome benefit) for some ACE inhibitors used in the management of heart failure are: ramipril (Prilace, Ramace, Tritace, Tryzan) 10 mg nocte, perindopril arginine (Coversyl) 10 mg nocte, lisinopril 30 mg nocte and trandolapril (Gopten, Odrik, Tranalpha) 4 mg nocte.

Beta blockers

When used in combination with ACE inhibitors, beta blockers reduce morbidity and mortality in patients with all grades of CHF by approximately 34%. Before starting treatment with a beta blocker, patients should be stabilised for at least one week and warned that when treatment is commenced they may initially feel more fatigued than usual. Patients should be started on low doses of beta blockers and doses should be increased every two weeks.[4,5] Left ventricular function returns to normal in up to one-third of patients treated with beta blockers.

Patients with chronic obstructive pulmonary disease can tolerate beta blockers as long as they have less than 18% reversibility in their FEV_1 following a bronchodilator challenge. Patients with true asthma or those who require continuous corticosteroid treatment may not tolerate beta blockers.

For patients who do tolerate beta blockers, increasing doses lead to incremental improvements in ventricular function and survival. Table 5 provides a guide to starting doses and target doses for the commonly used beta blockers in CHF.

Diuretics

Diuretics can control the symptoms of CHF effectively and their short term use improves salt and fluid retention, exercise tolerance and cardiac function in all grades of CHF.[4,5] No studies have been able to demonstrate that long-term use of diuretics leads to a reduction in mortality among patients with CHF and for this reason these drugs should only be used for symptom control if required and/or to achieve euvolaemia.

Second-line drug therapies

Spironolactone

The addition of spironolactone (Aldactone, Spiractin) to conventional therapy has been shown to improve CHF symptoms, reduce hospitalisations and result in a reduction in overall mortality among patients with severe CHF.[5]

Spironolactone is recommended for

Table 5. Starting and target doses for beta blockers in CHF (%)[1]

Beta blocker	Starting dose	Target dose
Bisoprolol (Bicor)	1.25 mg daily	10 mg daily
Carvedilol (Dilatrend, Kredex)	3.125 mg twice daily	25-50 mg twice daily
Metoprolol succinate (Toprol-XL)	23.75 mg daily	190 mg daily

use in severe heart failure in addition to the use of ACE inhibitors. This is because ACE inhibitors do not completely block the formation of angiotensin II or aldosterone and additional specific aldosterone antagonism leads to further benefit. Loop diuretics may achieve normal volume status but also increase the levels of aldosterone (unlike the aldosterone antagonist, spironolactone). The addition of an aldosterone antagonist leads to further improvements in volume status and survival. Diuretics should, however, be avoided in patients with an elevated potassium level or a creatinine clearance below 40 to 50 mL/ minute.

When initiating spironolactone, a patient's potassium level and renal function should be checked after one week of treatment, then at one month and every three months thereafter. Low doses of spironolactone – e.g. 12.5 mg daily (or even second-daily) to 25 mg daily are recommended, especially when used in combination with ACE inhibitors, other diuretics and/or angiotensin II receptor antagonists.

Angiotensin II receptor antagonists

Angiotensin II receptor antagonists (ARAs) reduce cardiovascular mortality and hospitalisation for CHF by 25% in patients with New York Heart Association Class II and III CHF (shortness of breath on mild to moderate exertion), which is a comparable but not superior effect to ACE inhibitors.[4,5] ARAs may be used as an alternative to ACE inhibitors in patients who cannot tolerate ACE inhibitors, especially those with the side effect of intractable cough.

When used in combination with ACE inhibitors, ARAs lead to a 15% reduction in cardiovascular mortality and hospitalisations for CHF. ACE inhibitors lead to some reductions in the levels of angiotensin II when used alone, and the addition of an ARA will augment this effect. These benefits, however, have not been shown to be as great as those achieved with ACE inhibitors or beta blockers in trials and for this reason ARAs should not be used in preference to ACE inhibitors or beta blockers for the management of CHF.

Candesartan (Atacand) is one of several available ARAs; its recommended dose is 8 mg nocte, which can be increased to 16 mg nocte and to a maximum of 32 mg nocte if tolerated. As with ACE inhibitors, the patient's potassium level and renal function should be monitored one to two weeks after initiating treatment with an ARA, and then one to two months later. If no abnormalities are detected with both tests, the patient's potassium level and renal function should then be monitored every three to six months.

Digoxin

Digoxin (Lanoxin, Sigmaxin) improves symptoms in CHF and increases exercise capacity among patients with CHF, especially those with atrial fibrillation. Hospitalisation for CHF are also reduced with digoxin but this drug does not improve survival.[4,5]

Low doses of digoxin (62.5 μg daily) help to avoid digoxin toxicity and/or possible arrhythmias such as atrial fibrillation, atrial flutter and supraventricluar tachycardia. The routine measurement of digoxin levels is no longer recommended and the dose of digoxin should be reduced in patients with renal impairment and for those patients taking amiodarone.

Nitrates

Nitrates such as isosorbide mononitrate are useful if the patient with CHF has myocardial ischaemia or pulmonary hypertension and/or peripheral oedema because they cause venodilatation and therefore improve venous capacitance and reduce right ventricular preload.[4,5] Topical nitrates are not well absorbed by patients with heart failure due to their poor peripheral perfusion. Isosorbide mononitrate is usually commenced at 30 mg daily and increased to 120 mg daily over a one to two week period.

Hydralazine

The combination of hydralazine (Alphapress, Apresoline) and nitrates has proven to be less effective than ACE inhibitors at reducing overall mortality among patients with CHF.[4,5] The major benefit of hydralazine is reduced nitrate tolerance, improved nitrate sensitivity and control of hypertension in patients with CHF that is not adequately controlled using first line medical therapy.

Hydralazine is generally commenced at 12.5 mg morning and night or three times a day. This initial dose can be slowly increased to a maximum of 100 mg morning and night or three times per day over one to two months. Hydralazine and nitrates should be reserved for patients in whom ACE inhibitors are contraindicated due to cough, renal impairment or high potassium levels and where no other therapeutic option exists.

Amiodarone

Amiodarone (Aratac, Cardinorm, Cordarone X, Rithmik) may be useful for controlling atrial fibrillation and ventricular arrhythmias in patients with CHF but use of this antiarrhythmic may be limited by the fact that it can cause bradycardia, thyroid dysfunction, pulmonary fibrosis, corneal deposits and/or sun sensitivity.[6]

Amiodarone has not been shown to improve survival among patients with CHF, but it can slow the heart rate in patients who are tachycardic and hypotensive and therefore unable to tolerate beta blockers.[6] Use of amiodarone in this context is a temporary measure and the longer the treatment with amiodarone, the higher the risk of adverse effects. The recommended doses are 100 to 200 mg daily.

Other medications

Anticoagulants

Warfarin (Coumadin, Marevan) is recommended for all patients with CHF who also have coexisting atrial fibrillation. This is to reduce the rate of systemic embolism

secondary to cardiac thrombus.[1] The suggested target INR is 2 to 3.

Statins

All patients with CHF resulting from prior MI, coronary bypass grafting or angioplasty/coronary stenting should be prescribed a statin.[1] The LDL-cholesterol level targeted should be below 2.0 mmol/L. Patients should also be prescribed low dose aspirin (75 to 150 mg/day) if they have CHF as a result of the above mentioned causes.

Thiamine

Thiamine should be considered in patients who consume excessive alcohol or those who have poor nutrition.[1]

Allopurinol

Given that uric acid is negatively inotropic and high uric acid levels are a marker of poor prognosis in heart failure, administration of allopurinol (Allohexal, Allosig, Progout, Zyloprim) should be considered in patients with cardiac cachexia. Weight loss of more than 6% within the past year is usually used to define cardiac cachexia.

Erythropoietic agents

Erythropoeitic agents may be of benefit to patients with CHF who also have anaemia from renal impairment. Erythropoietic agents improve quality of life and their effect on survival in CHF is currently being investigated.

Table 6. Drugs that can worsen CHF

Calcium channel antagonists (central and peripherally acting)
Corticosteroids
NSAIDs including COX-2 inhibitors
Macrolide antibiotics
Thiazolidinediones
Tricyclic antidepressants
Type 1 antihistamines
H_2-receptor antagonists
TNF-alpha inhibitors

Table 7. Causes of exacerbations of CHF

Myocardial ischaemia
Arrhythmias – most commonly atrial fibrillation
Valvular dysfunction – e.g. acute mitral valve regurgitation
Noncompliance with medication or cessation of medication – particularly frusemide
Lack of adherence to salt and fluid restriction
Commencing drugs that worsen CHF
Infections – e.g. respiratory, urinary tract or cellulitis
Renal failure leading to fluid overload
Anaemia
Pulmonary embolus
Thyroid imbalance – e.g. hypothyroidism or hyperthyroidism

Medications to avoid in CHF

Several drugs need to be avoided or used with caution in patients with CHF because they have the potential to worsen symptoms of CHF or negate the effects of some of the essential drugs used to treat the condition (Table 6). For example, NSAIDs including cyclo-oxygenase (COX-2) inhibitors should be avoided because they can antagonise the action of ACE inhibitors and have a negative effect on renal function, causing salt and water retention. The thiazolidinedione antidiabetic agents should also be avoided because they cause fluid retention have been reported to cause significant exacerbation of heart failure.

Tricyclic antidepressants, type I antihistamines and macrolide antibiotics may be proarrhythmic in patients with CHF and both atrial fibrillation and polymorphic ventricular tachycardia have been reported with these agents. Calcium channel blockers have also been associated with increased adverse outcomes in CHF studies both after MI and in CHF. Table 6 lists drugs that can worsen CHF.

Nonpharmacological management

Several nonpharmacological interventions play an important role in the management of CHF and the prevention of exacerbations. These interventions are outlined below, and potential causes of exacerbations are listed in Table 7.

Physical activity

Patients with CHF who undergo moderate exercise training have better quality of life and improved mortality rates.[7] For this reason, regular physical activity is recommended and patients should be referred to an exercise program specifically designed for patients with CHF, if available locally. Bed rest is recommended only during an acute exacerbation or if the patient's CHF is clinically unstable.[1]

Medical and nursing support

Patient support provided by the doctor, a predischarge review by a nurse and home visits are crucial for preventing deterioration in a patient's heart failure status. These programs reduce the rate of preventable readmissions, improve mortality, improve the transition from the hospital to the community and have been shown to be cost-effective.

Such programs usually involve a nurse visiting the patient in hospital before discharge and then at home one week after discharge. The goal of these programs is to educate patients with CHF about:

- daily weight measurement
- fluid management and salt restriction
- knowledge of the signs of clinical deterioration
- an action plan for when they deteriorate.

The nurse also makes sure patients have visited their family doctor and have

an appointment to see their specialist within one month. The nurse can also organise home help and meals-on-wheels if required. In addition, district nursing can be arranged if patients require medication administration, dressings (if they have any skin lesions) and assistance with personal hygiene.[7]

Fluid restriction, diet and lifestyle

Sodium restriction helps with control of the patient's fluid volume, reduces the dosages needed of pharmacological agents such as diuretics, and leads to fewer decompensations. However, there are no studies to show there is a beneficial reduction in mortality from sodium restriction. Dietary sodium intake should be limited to less than 200 mg/day. Fluid intake should be limited to 1.5 L/day in general and 1 L/day for patients with severe heart failure. Alcohol should be avoided completely if possible but if this is not achievable, alcohol intake should be limited to less than 10 to 20 g/day.[1] Patients with CHF should completely cease smoking and smoking cessation therapies should be implemented.

For patients who have obstructive sleep apnoea, continuous positive airway pressure may be helpful.[1] Patients should be weighed daily at home and they should consult their doctor if their weight increases by more than 2 kg in two days or if they experience dyspnoea, oedema or bloating.[1] Pneumococcal and influenza vaccinations should be provided to all patients with CHF.

Travel considerations

Air travel over short distances appears to be low risk for patients with mild CHF. Although long flights are not contraindicated, they may predispose the patient to accidental omission of medications, lower limb oedema, dehydration and venous thrombosis.[1]

High altitude destinations should be avoided because of the risk of developing relative hypoxia.[1] Patients with CHF travelling to very humid or hot climates, should be counselled about the risk of dehydration and instructed on how to modify their doses of diuretics.

Another important reason why travel should always be discussed with a doctor is the increased risk of thrombosis due to long-haul flying. If long flights are planned, patients should be advised about deep venous thrombosis (DVT) prophylaxis using graduated compression stockings and in-flight calf stretching exercises and prescribed pharmacological therapy if the risk of DVT is considered to be significant.[1]

Cardiac assist devices and transplantation

Recently, mechanical devices have been used in an attempt to improve outcomes

in CHF by either improving cardiac contractility and reducing delayed repolarisation in LBBB, reducing fatal arrhythmias or augmenting cardiac contractility and cardiac output. These devices are ancillary to optimal medical therapy and are only for selected patients who are not improving on maximal medications and who satisfy the selection criteria for the devices. The cost of these devices precludes their routine use, particularly in elderly patients, and they do have inherent risks such as infection and malfunction.

Biventricular pacing

Biventricular pacing helps improve left ventricular function by resynchronising the contraction pattern of the left ventricle in patients with LBBB.[7,8] Biventricular pacing is safe and effective and improves symptoms, haemodynamics and survival in CHF patients who have LBBB.[7,8]

Implantable defibrillators

Implantable cardiac defibrillators are an effective treatment to prevent death caused by ventricular arrhythmia in patients with CHF and in those patients with spontaneous or inducible ventricular arrhythmias. The greatest benefit is seen in patients with the lowest left ventricular ejection fraction.[9]

Implantable devices are increasingly considered as a therapeutic option. They are appropriate not only for secondary prevention of sudden death in patients who have been resuscitated from ventricular tachycardia arrest, but also as a primary prevention strategy for patients with CHF who are at risk of sudden death from ventricular arrhythmias.[7,8]

Cardiac transplantation

Cardiac transplantation is a viable option for patients with advanced heart failure but as the number of people with CHF continues to grow, so too does the waiting time for patients on the heart transplantation waiting list.[9]

Left ventricular assistance devices

Left ventricular assist devices (LVADs) were developed initially for use as a bridge to cardiac transplantation and have been successful in achieving this aim. They may also be effective as an alternative to cardiac transplantation to prolong life as well as improving overall quality of life. Despite these benefits, LVADs are associated with significant adverse events including infections and haemorrhagic complications.[9,10]

Conclusion

Heart failure is an increasingly common condition causing high mortality, poor quality of life and recurrent hospitalisations. Treatment has been shown to improve symptoms and survival and reduce rehospitalisations.

Patients with CHF are often elderly with a range of concurrent medical problems and therefore require a combination of medications and regular review. Correct diagnosis, specialist review and ongoing therapy can reduce the burden of CHF and help with the management of this lethal, expensive and debilitating disease. MT

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