Chronic heart failure

Optimising care in general practice

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BACKGROUND Chronic heart failure (CHF) is a significant cause of mortality and morbidity in developed countries where it predominately affects elderly persons with a range of other comorbid conditions requiring polypharmacy. In Australia, over 300 000 people are affected by CHF. Quality general practice forms the cornerstone for early diagnosis and evidence based integrated care.

OBJECTIVE This article examines the epidemiology of CHF, its clinical diagnosis, contemporary management and future treatment possibilities, as well as current barriers to optimal care.

DISCUSSION The global prevalence of CHF is rising. Optimal treatment requires a coordinated interdisciplinary approach using a biopsychosocial model of care in order to maximise compliance with therapy. Pharmacological treatment is essential and should include an angiotensin converting enzyme inhibitor and beta blocker where possible. Device based treatment and cardiac surgery may benefit selected cases.

Globally we are witnessing a profound shift in morbidity and mortality from communicable diseases to noncommunicable chronic and complex diseases. Deaths from noncommunicable disease are expected to rise from 28 million in 1990 to around 50 million in 2020 – an increase in absolute terms of 77%.[1]

Disability adjusted life year (DALY) is a measure of the years of healthy life lost due to illness or injury. One DALY is one lost year of healthy life (whether due to illness, disease or death) and acknowledges the significance of loss of quality life rather than simply loss of years of life. *Table 1* compares the global disease burden in DALY in 1990 with anticipated burden in 2020 based on the rank order of five leading causes. Note the changing pattern globally toward the current spectrum of diseases found in developed countries.

Prevalence of CHF

Although the exact figures are not known, an estimated 300 000 Australians are affected by chronic heart failure (CHF) and 30 000 new cases are diagnosed each year.[2] In 1997, there were 41 000 hospital admissions for CHF,[2] which is also a common reason for recurrent admission, long stays and higher than average hospital costs. A survey of Australian general practitioners[3] estimated that for every 100 patients aged 60 years and over, 11 had known CHF and two could be newly diagnosed on the basis of clinical features and known risk factors. The risk of developing CHF will increase in the future as a result of:

- aging
- increased prevalence of hypertension, ischaemic heart disease and diabetes
- decreased case fatality rate due to improved

Table 1. 1990 global disease burden in DALY compared with anticipated burden in 2020

1990	2020
• Lower respiratory infections	• Ischaemic heart disease
• Diarrhoeal disease	• Unipolar depression
• Conditions arising in the perinatal period	• Road traffic accidents
• Unipolar depression	• Cerebrovascular disease
• Ischaemic heart disease	• COPD

Source: Global burden of disease and injury www.hsph.harvard.edu/organizations/bdu/gbdmain.htm

Table 2. Classification of CHF[4]

Class I	Asymptomatic left ventricular dysfunction
Class II	Slight limitation of physical activity. Ordinary physical activity results in fatigue, palpitation, dyspnoea or angina (mild CHF)
Class III	Marked limitation of physical activity. Less than ordinary physical activity leads to symptoms (moderate CHF)
Class IV	Unable to carry on any physical activity without discomfort. Symptoms of CHF present at rest (severe CHF)

treatment of acute coronary syndromes (and these patients ultimately developing CHF as an end stage outcome), and
- improved detection (raised awareness and use of diagnostic techniques such as echocardiography).

Definition and classification

The fundamental clinical definition of heart failure has not changed significantly over the past 100 years, however, our understanding of its pathophysiology has improved dramatically. Chronic heart failure:
- occurs when cardiac output is insufficient to meet the requirements of the body for blood supply during rest or activity
- is characterised by pulmonary and systemic venous congestion, fluid retention and reduced salt excretion mediated by a combination of neurohumoral and pro-inflammatory mechanisms.

The severity of CHF is classified by the New York Heart Association (NYHA) criteria into four categories according to symptoms (*Table 2*).

While this article focusses on systolic heart failure (impaired ventricular contraction) it is increasingly recognised that one-third to half of heart failure presentations are due to 'diastolic heart failure' (impaired ventricular relaxation). This is also referred to as 'heart failure with preserved left ventricular systolic function'. The pathophysiology of this condition is still being elucidated, however hypertension, ischaemic heart disease and diabetes are recognised as common causes. Clinically, patients with diastolic heart failure present with similar features to those with systolic heart failure. Treatment is predominantly directed at achieving euvolaemia with diuretics. Evidence for effective drug therapies in diastolic heart failure is limited but currently under investigation.

Diagnosis of CHF

Diagnosis of CHF is usually established by taking a thorough history and examination and may be supported by the following tests (it is important to exclude potentially reversible underlying pathology):
- chest X-ray
- electrocardiogram (ECG)
- echocardiography (necessary not only to assess left ventricular function, but also to exclude valvular abnormalities)
- stress imaging, eg. echocardiography or nuclear (to assess for reversible myocardial ischaemia)
- B-type natriuretic peptide (BNP) (highly sensitive, although less specific for active CHF).

The best way to demonstrate the clinical presentation of CHF and problems in management is to use a case study from general practice (see *Case history*).

Interdisciplinary and biopsychosocial model of care

As can be seen from the case history of Mr B below, CHF is a chronic and complex condition, and frequently associated with other, often multiple, comorbid conditions. Mr B is clearly disabled with marked impairment of function and diminished quality of life. In terms of the NYHA classification he is in class III and may soon be in class IV. He takes 16 tablets per day as well as patches and puffers. Despite potential problems with fluid retention, celecoxib helps his joint pains and mobility. His wife is also disabled and should either of them deteriorate further, the other may not be able to function as carer. He therefore has superimposed social problems and suffers bouts of depression and insomnia. His treating general practitioner will be acutely aware of the enhanced risks to which Mr B is exposed, and must be prepared to manage them using a biopsychosocial approach. Mr B's risks include:
- sudden death
- invalidism
- insidious deterioration and death

Case history – Mr B

Mr B is 76 years of age. He has a 10 year history of ischaemic heart disease, atrial fibrillation, type 2 diabetes mellitus, COPD and osteoarthritis. He lives with his wife (72 years of age) who has hypertension and moderately severe rheumatoid arthritis. Mr B:

- has had two myocardial infarcts
- had CABG in 1995
- abdominal aorta aneurysm repair in 1996
- femoral angioplasties in 1998 for claudication
- has had three hospital admissions for CHF in the past 12 months
- has been assessed for repeat CABG but thought unsuitable
- is dyspnoeic on walking 20 m on the flat and gets angina on cold or windy days at similar distances

Clinical examination

- Pulse 68, irregularly
- BP 110/70
- JVP 3 cm, apex beat 6th LICS, 12 cm from mid-line, loud ESM at apex, normal heart sounds
- Occasional basal crackles persisting after coughing
- No peripheral oedema

Medication

- digoxin 0.125 mg
- frusemide 80 mg
- enalapril 10 mg
- metopolol 50 mg
- spironolactone 75 mg
- warfarin 5 mg
- GTN patch 50 mg
- metformin 1000 mg
- glibenclamide 10 mg
- celecoxib 200 mg
- salbutamol
- fluticasone 250 µg
- sertraline 100 mg
- temazepam 10 mg

- depression
- stroke, acute myocardial infarction
- wife's illness and its complications
- chest infections
- social isolation
- iatrogenesis (including drug interactions)
- falls, fractures
- renal failure
- complications of his diabetes, and
- further hospitalisation.

As with most chronic conditions the burden of care lies with the GP. However, given the complex nature of the condition, there is clearly a need to involve other health professionals in the care of patients, as well as family members and the patients themselves.

Systematic reviews of the literature on a number of trials[5–7] have demonstrated the benefits of integrated multidisciplinary patient management and programs in preventing exacerbation of CHF and re-admission to hospital. Integrated care (including health assessments, care plans and case conferences as provided under the Enhanced Primary Care Package) involves the following, with the GP playing a key role as care coordinator:

- hospital/hospital in the home
- cardiologist
- GP
- nurse
- pharmacist
- carer
- patient
- telephone support system.[8]

Management of CHF

Management of CHF involves:

- prevention or delay in onset of the condition through improved management of risk factors
- adequate treatment of both the condition itself and associated comorbidities
- prevention of acute exacerbations, and
- management of the terminal stages of the condition.

Chronic heart failure has a 50% mortality rate within 3 years of the first hospital admission. Poor compliance with therapy is the most common reason for deterioration and hospitalisation, and re-admission.

General measures

General measures include:

- fluid restriction (usually 1.5 L/day especially if hyponatraemic)
- daily weighing, seek help if weight gain or drop >1.5 kg/day
- sodium restriction <2 gm/day (translates roughly to no added salt to food or cooking)
- low to medium level activity on a regular basis
- cardiovascular risk factor optimisation (including BP, HbA1C, lipids, weight management, smoking cessation)
- vaccination: influenza annually, pneumococcal every 5 years, and
- diagnosis and treatment of depression.

Ongoing monitoring of patients with CHF will also involve:

- reviewing symptoms
- examination (including regular weighing)
- 2 yearly echocardiography (more frequently if indicated)

- chest X-ray when clinically necessary, and
- 3–4 monthly electrolytes, urea and creatinine (more frequently at the time of medication changes, eg. angiotensin converting enzyme [ACE] inhibitors, spironolactone).

Exacerbations of heart failure may have precipitating causes and these need to be sought (*Table 3*).

Table 3. Precipitating causes for exacerbations of CHF

- Noncompliance
- Arrhythmias
- Coronary ischaemia
- Infection
- Thyroid dysfunction (hyper or hypo)
- Anaemia
- Surgery
- Pulmonary embolism
- Sleep apnoea
- Pregnancy
- Uncontrolled hypertension
- Medications
  - NSAIDS
  - beta blockers (inappropriately used)
  - calcium channel blockers
  - tricyclics (rhythm disturbance)

Specific drug treatment

Pharmacological treatment is essential in the management of CHF.

First line agents

Angiotensin converting enzymes inhibitors (if tolerated) are mandatory in all patients with systolic heart failure (left ventricular ejection fraction <40%), whether symptoms are mild, moderate or severe. Every effort should be made to up-titrate to the highest tolerated dose of ACE inhibitor. If this is not possible, a lower dose is preferable to none at all. Electrolytes, urea and creatinine should be checked 4–7 days following a dose change to assess potassium (K+) and renal function.

Angiotensin 2 receptor antagonists may be used as an alternative to ACE inhibitors for patients who are ACE intolerant due to bradykinin mediated adverse effects, eg. cough. They may also be used in conjunction with ACE inhibitors in certain patients.

Diuretics should be used if necessary to achieve euvolaemia in fluid overloaded patients. In patients with systolic left ventricular dysfunction, diuretics should never be used as monotherapy, except in exceptional circumstances, but should always be combined with an ACE inhibitor to maintain euvolaemia.

Beta blockers are recommended therapy, unless not tolerated or contraindicated, for all patients with systolic CHF who remain mild to moderately symptomatic despite appropriate doses of ACE inhibitors and diuretics. Importantly, studies suggest that many chronic obstructive pulmonary disease (COPD) patients will tolerate selective beta blockade.[9] In practice, many cardiologists will trial their patients who have both heart failure and COPD with beta blockers if the patient's respiratory function tests show no evidence of significant bronchodilator reversibility. Beta blockers can also be recommended for patients with symptoms of advanced CHF. To maximise tolerability of a beta blockade in this subgroup, beta blockers should ideally be commenced (in conjunction with a cardiologist) in a low dose when the patient is euvolaemic and their fluid balance optimal. Up-titration of dose and symptomatic improvement may take several months.

Spironolactone is recommended for patients with left ventricular ejection fraction less than 40%, who remain symptomatic despite appropriate doses of ACE inhibitors and diuretics.[10] However, hyperkalaemia (K+ >5.5 mmol/L) must be carefully watched for.[11]

Other agents

Digoxin can be considered in patients with advanced CHF for relief of symptoms and reduction of hospitalisation, but should be introduced after therapies with a proven survival benefit, ie. ACE inhibitors, beta blockers, spironolactone. It remains valuable therapy in CHF patients with atrial fibrillation.

Hydralazine-isosorbide dinitrate should be reserved as a third line agent for patients who are truly intolerant of the above mentioned therapies or for whom the above mentioned agents are contraindicated. Third line agents may also be useful for symptom control – in addition to first and second line therapies – in patients with end stage heart failure.

Device based heart failure treatments

The use of device based heart failure therapy has become increasingly widespread and the evidence base for their use is increasing. There are three major groups of devices:

- biventricular pacemakers (also known as cardiac re-synchronisation therapy)
- implantable cardiac defibrillators, and
- left ventricular assist devices.

Cardiac resynchronisation therapy (CRT) is based on the observation that a significant proportion of moderate to severe heart failure patients develop dysynchrony of left and right ventricular contraction. This dysynchrony causes a reduction in cardiac output

and pump efficiency. Electrical pacing of both the left and right ventricle, as opposed to conventional pacing of the right ventricle alone, has been shown to restore synchrony of right and left ventricular contraction, improve cardiac output, and significantly reduce heart failure morbidity and mortality.[12] Initially patients were selected for this therapy on the basis of symptoms and the ECG finding of left bundle branch block (indicating right and left ventricular electrical dysynchrony). Emerging evidence points to specialised echo techniques being a more accurate indicator of likely patient response to this therapy.

Implantable cardiac defibrillators (ICDs) have been shown to significantly reduce mortality from sudden cardiac death in patients with left ventricular ejection fractions less than 30%, whether of ischaemic or nonischaemic aetiology.[13] A number of pacemaker companies now produce combined ICD and CRT devices. Despite the body of evidence supporting the use of device based heart failure therapies, their use is limited by cost.

Left ventricular assist devices (LVADs) are surgically implanted pumps that are used to support severely impaired left ventricular function. To date they have largely been employed as a bridge to cardiac transplantation. There is research underway into their use as a 'destination therapy' for patients with severe heart failure who are not candidates for transplantation.

Still very much in the development phase, is the ultimate device therapy – a completely self contained artificial heart. Such devices (eg. AbioCor) have been used in very small numbers of patients. Eventually it is hoped that these devices may alleviate the major problem of poor donor heart availability for cardiac transplantation.

Cardiac surgery

Coronary artery bypass graft surgery (CABG), where the degree of coronary disease warrants revascularisation, particularly in those in whom there is scanning evidence of myocardial viability and the patient is fit enough to survive a technically feasible operation, may reduce heart failure symptoms and improve survival.

Surgical ventricular restoration (SVR) is an evolving surgical technique for the treatment of systolic heart failure. This procedure involves surgical reduction of an enlarged left ventricle in order to restore normal ventricular geometry and optimise function. The international multicentre randomised control trial STICH (Surgical Treatments for IsChaemic Heart failure) is currently evaluating this, as well as assessing the impact of CABG on heart failure patients.

Cardiac transplantation remains an effective treatment for severe heart failure in selected patients with symptoms refractory to combined maximal pharmacological and device based therapies. Transplantation as a therapeutic modality is limited by the scarcity of donor hearts, the complications of immunosuppressive therapy and the problems of graft rejection. However, long term results in Australian and overseas units specialising in transplant surgery are excellent.

New horizons in therapy

Extensive research into novel treatments for heart failure is in progress. This include new pharmacological agents, innovative approaches to gene therapy, immunological based treatments and treatments aimed at myocardial regeneration such as stem cell therapies.

Barriers to evidence based care

Despite the presence of succinct National Heart Foundation of Australia guidelines[4] on optimal management of patients with CHF, it is clear that in many instances care of these patients falls short of evidence based best practice.[14,15] This does not apply to the management of CHF alone, but also to a number of other common chronic illnesses in Australia. However, unless barriers are identified and systematically addressed, improvement in care is unlikely to occur.[16] A recent qualitative study by Phillips et al[17] revealed difficulties in diagnosis due to associated comorbidities, reluctance to use echocardiography because of lack of clarity regarding its benefits (as well as access problems in rural areas), and suboptimal use of ACE inhibitors and beta blockers due to concern about side effects, contraindications and comorbidities.

The barriers to following guidelines and poor patient compliance are contributors to frequent relapses and acute exacerbations of CHF resulting in costly hospital admissions. Better heart failure patient education may well be one of the first steps toward achieving improved compliance. A number of very useful patient education resources are available on the National Heart Foundation of Australia website at: www.heartfoundation.com.au.

It should be noted that effective primary and secondary prevention of ischaemic heart disease through community education, cardiac risk factor reduction, and effective prescribing habits, is likely to

significantly assist in reducing the future burden of heart failure morbidity and mortality.

Conclusion

Chronic heart failure is increasing globally in prevalence and will form the 'end stage' condition in many patients currently being treated for ischaemic heart disease. Treatment of patients with CHF remains the domain of the GP using an evidence based biopsychosocial model of integrated care which involves other disciplines, the patient and their family working together to maximise therapeutic options. Pharmacological treatment is essential and should include an ACE inhibitor and beta blocker wherever possible. Device based treatment is useful and effective in selected cases. The role of cardiac surgery in heart failure treatment is limited at present, however, this may become an important player. Barriers to optimal care need to be identified and addressed, and patient compliance maximised to ensure the best possible clinical outcomes.

Conflict of interest: none.

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