An overview of chronic heart failure management

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- Causes, symptoms and signs of heart failure
- Pharmacological management
- Non-drug management of the condition

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The prevalence of chronic heart failure is set to increase due to the growing ageing population. Nurses in all settings have a vital role in supporting patients in managing their condition.

This article discusses the physiology of the heart, the causes and pathophysiology of heart failure, and treatment and management options.

Heart failure is a common clinical syndrome that is the end result of any structural or functional cardiac disorder that impairs the pumping ability of the heart. Common symptoms and signs include: breathing difficulties; oedematous ankles, legs, feet or abdomen; fatigue and exercise intolerance.

Although the condition has a poor survival rate, this can be improved with timely diagnosis and evidence-based treatments. The aims of treatment are to improve patients’ life expectancy and quality of life.

Regulation of heart function

In a normal resting state, the heart pumps the entire circulating volume of blood (approximately five litres) (Colbert et al, 2009) – known as cardiac output (Fig 1) around the body once every minute.

A rise in cardiac output occurs due to an increase in heart rate, stroke volume or both.

During an increase in metabolic demand such as that caused by exercise, cardiac output increases to meet it.

There are four main contributors to the heart’s contractile function:

Preload: this refers to the volume of blood in the ventricle and the pressure it causes immediately before it contracts; it is also known as left ventricular end diastolic pressure (LVEDP). Preload links stroke volume to venous return through the Frank-Starling law, which states that an increase in venous return (to the heart) brings about ventricular stretch, resulting in an enhanced, forceful cardiac muscle contraction, therefore enhancing stroke volume. The more stretched a muscle cell is, the greater the force of contraction. This relationship continues up to a point. Once a plateau is reached, the force lessens – possibly due to overstretching of the cardiac contractile ability – and cardiac output drops.

Contractility: this refers to the inotropic state of the heart – its ability to contract. The movement of calcium into the cardiac cells is significant in enabling them to shorten and produce a contraction.

Afterload: this is the force of resistance opposing the ventricles during contraction and is due to pressure in the pulmonary or systemic circulation or semilunar valves. An increase in afterload leads to a decrease in stroke volume, while a decrease in afterload results in an increase in stroke volume.

Heart rate: an increase in heart rate reduces the filling time of the ventricles and results in reduced stroke volume, while a decrease in heart rate increases ventricle filling time, resulting in an increase in stroke volume.

Fig 2 depicts the cardiac cycle.

5 key points

1 Chronic heart failure is a complex syndrome of signs and symptoms that occur as a result of any structural or functional cardiac disorder.

2 The most common cause is coronary heart disease.

3 The incidence and prevalence of heart failure increase steeply with age.

4 Echocardiography is considered the gold-standard diagnostic test.

5 The main aims of treatment are to alleviate symptoms, enhance quality of life and improve life expectancy.

FIG 1. CARDIAC OUTPUT

Cardiac output (CO) is the volume of blood pumped through the heart per minute = Stroke volume (SV) x heart rate (HR)

The amount of blood ejected by the ventricles in one contraction

Number of ventricular contractions in one minute

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What is chronic heart failure?
Chronic heart failure (CHF) is a complex syndrome of signs and symptoms that occur as a result of any structural or functional cardiac disorder (Box 1) that impairs the heart’s pumping ability, such as interrupted blood supply, injury to the heart muscle cells or increased workload (National Institute for Health and Clinical Excellence, 2010). It is not a disease in itself; it is the result of a disease process. A variety of terms are used to describe CHF (Box 2).

By far the most common cause of heart failure is coronary heart disease (CHD), usually with a history of previous myocardial infarction (MI) (Box 1). CHD accounts for as many as 70% of cases of heart failure (European Society of Cardiology, 2010). The remaining cases are non-ischaemic (Box 1). Lifestyle factors such as excessive alcohol consumption, smoking and obesity often feature in the aetiology (Butler, 2010).

Compensatory mechanisms
In CHF where the heart has been damaged, there are often changes in its size, shape, structure and function; this is known as remodelling. It is more commonly seen in the left ventricle as it is the larger one with thicker muscle mass, but it can be seen in the right ventricle or both together (known as biventricular failure).

Remodelling impairs left ventricular function, leaving the heart unable to function as an effective pump. As a result, there is a fall in cardiac output, which leads to the activation of several compensatory mechanisms (sympathetic nervous system, renin-angiotensin-aldosterone system, ventricular dilation and ventricular hypertrophy), all of which aim to restore adequate left ventricular function.

**Sympathetic nervous system:** when the heart begins to fail, the body activates the sympathetic nervous system to increase the heart rate and the force of contraction, thus increasing cardiac output. However, sympathetic nerve stimulation also constricts the arteries, which increases blood pressure. The increase in pressure forces the heart to work harder and use more oxygen, which is thought to cause further deterioration of the heart over time.

**Renin–angiotensin–aldosterone system:** decreased blood flow (due to reduced cardiac output) causes the kidney to release renin, an enzyme that converts an inactive plasma protein, angiotensinogen, into angiotensin II, an active hormone. Angiotensin II is a powerful constrictor of blood vessels and stimulates the adrenal gland to secrete aldosterone. This hormone causes the kidneys to retain salt and water, which increases blood volume, helping to maintain cardiac output by increasing the filling of the heart. However, increased blood volume, along with vasoconstriction, increases blood pressure. This increased pressure causes oedema and an increased workload, which may further weaken the heart.

**Hypertrophy:** there are changes in the muscle itself. The thickness of the muscle layer increases (hypertrophy), enabling the heart to contract with greater force to maintain cardiac output. This increases the need for oxygen and eventually leads to further deterioration. The heart can also enlarge by stretching and thinning its walls (dilation). Initially, this may help to increase cardiac output by increasing the amount of blood that the heart can hold, but the dilation eventually fails and leads to further worsening of the disease.

The heart’s inability to meet increased demand brings about clinical symptoms (Box 3). The compensatory mechanisms, while designed to help the failing heart, if left to continue, will eventually lead to:

*BOX 1. CAUSES OF HEART FAILURE*
- Coronary heart disease (ischaemia, myocardial infarction)
- Hypertension
- Valvular heart disease (mitral/aortic)
- Arrhythmias (atrial fibrillation, complete heart block)
- Congenital heart disease (atrial/ventricular septal defects)
- Cardiomyopathy (dilated, hypertrophic, restrictive, viral myocarditis, familial)
- Toxins (excessive alcohol, chemotherapy)
- Peripartum/postpartum
- Hypothyroidism
- Infection (viruses, HIV, bacteria)

*BOX 2. HEART FAILURE TERMS*
- Right heart failure
- Left heart failure
- Congestive heart failure
- Biventricular failure
- Left ventricular failure
- Left ventricular systolic dysfunction (LVSD)
- Diastolic dysfunction (also known as heart failure with preserved ejection fraction)
Epidemiology
The European Society of Cardiology (2010) gives the prevalence of heart failure in the overall population as 2-3%. According to NICE (2010), around 900,000 people in the UK have the condition.

This figure is set to rise as the population ages and more people survive heart attacks but are left with reduced left ventricular function (Stewart et al. 2003). Gardner et al (2007) predicted an increase of 50% in prevalence over the next 20 years, and attributed this to improved survival rates for CHF.

Although the condition can occur in all age groups, incidence and prevalence increase steeply with age. Average age at first diagnosis is typically 76 years, with prevalence in the 70-80-year-old age group rising to 10-20% (ESC, 2010).

Heart failure has a poor prognosis, with a mortality rate of around 30-40% in the first year; it has been said to have poorer survival rates than breast and prostate cancer (National Clinical Audit Support Programme, 2009). Thereafter, mortality is less than 10% per year. However, an encouraging trend of improved prognosis has emerged in the last decade, with the six-month mortality rate falling from 26% in 1995 to 14% in 2005 (Mehta et al, 2009).

Cost
The cost relating to heart failure may be difficult to determine when taking into account diagnosis, treatment, hospital stays, pharmacology, loss of income and costs related to carers’ involvement.

Hospital activity accounts for the majority of expenditure, closely followed by primary care, pharmacology and hospital outpatient care (British Heart Foundation, 2010). The National Collaborating Centre for Chronic Conditions (2003) said that heart failure accounted for 1.8% of the total NHS budget in 1995 (around £716m). Since then, it has been further estimated as £905m in 2000 (Nicholas, 2004).

In 2010, the cost of GP consultations for heart failure was estimated at £457m per year, with an additional £35m for referrals to outpatient clinics. A further £129m was spent on community-based drugs for this group of patients (NICE, 2010).

Symptoms and signs
As mentioned above, heart failure is a complex syndrome, with patients presenting with a variety of non-specific symptoms and signs (Box 3). Older patients often attribute these to the ageing process, especially if symptoms are mild.

Symptoms frequently relate to the accumulation of excess fluid, in particular, shortness of breath, paroxysmal nocturnal dyspnoea, orthopnoea and ankle swelling.

The symptoms patients experience are used to give them a New York Heart Association (NYHA) class (Table 1) and enable ongoing assessments to be made. The NYHA provides a subjective guide to patients’ functional ability.

Weight gain may occur due to a build-up of fluid in the tissues, while weight loss may be seen as a result of muscle atrophy or malabsorption (because of bowel and/or liver oedema). Fatigue and exercise intolerance develop as a result of structural and metabolic changes in skeletal muscle.

Diagnosing heart failure
It is important to ensure a correct diagnosis is made so that any reversible causes can be addressed, and treatment and patient education started.

To diagnose heart failure, a detailed history and clinical examination are needed (NICE, 2010; Massey, 2006). As symptoms and signs are often non-specific and could be related to other conditions, objective evidence is also required.

Echocardiography is considered the gold-standard diagnostic test as it provides information on the cardiac structure, degree of dysfunction within the myocardium and, therefore, the underlying cause. However, many more people with symptoms of heart failure will be referred for echocardiography than are confirmed with a diagnosis (Zaphiriou et al, 2005).

Serum natriuretic peptides (SNP) are hormones produced by the heart in response to ventricular wall stretch and can be measured by a simple blood test. They are useful markers for heart failure and are recommended by NICE (2010) as a “rule-out” test. They should be performed before echocardiography for patients who have not had an MI. This is to identify which patients need to be referred for echocardiography (all those who have had an MI need echocardiography so do not need an SNP test beforehand), and to reduce the demand on diagnostic services through unnecessary requests.

Other investigations could include:
- 12-lead electrocardiogram (ECG) – may show evidence of left or right ventricular hypertrophy, CHD or arrhythmias commonly associated with heart failure such as atrial fibrillation (AF);
- Chest X-ray – to look for cardiomegaly, pulmonary congestion and respiratory disease;
- Blood tests – urea and electrolytes, full blood count, liver function tests, thyroid function tests, glucose level and lipid profile;
- Respiratory function tests – to exclude respiratory causes for dyspnoea.

Management
The management of heart failure consists of both pharmacological (Table 2) and non-pharmacological interventions. The main aims of treatment are to alleviate symptoms, enhance quality of life and improve life expectancy (NICE, 2010).

The most up-to-date evidence for pharmacological intervention is based on heart failure due to left ventricular systolic dysfunction. However, the approach to management is similar whether systolic function is reduced or not.

Pharmacological
Diuretics are used to relieve symptoms of dyspnoea and oedema and can be titrated up and down according to response. There is no evidence to show they improve
life expectancy. Two types of diuretics are commonly used: loop diuretics, such as furosemide or bumetanide, which inhibit sodium and water reabsorption; and thiazide diuretics, such as bendroflumethiazide, a milder diuretic. It should be noted that diuretic resistance occurs in some patients.

Angiotensin-converting enzyme inhibitors (ACE inhibitors) block the renin-angiotensin-aldosterone system, which helps to reduce the effects of one of the maladaptive compensatory mechanisms. As they improve both symptoms and survival, they are probably the most valuable medication to use in heart failure. If a diuretic is not indicated (because there is no accumulation of excess fluid), ACE inhibitors become first-line therapy (with beta-blockers) (NICE, 2010). This group of medications should be started at a low dose and titrated slowly, paying close attention to blood pressure and heart rate. As with ACE inhibitors, they should be started with a low dose and titrated slowly, paying special attention to blood pressure and heart rate, until the tolerated target dose has been achieved.

Angiotensin-II receptor blockers (ARBs), also known as angiotensin–II receptor antagonists, have similar properties to ACE inhibitors and can be used in patients who cannot tolerate ACE inhibitors due to cough. The same principles apply to this group of medications as when using an ACE inhibitor.

Beta-blockers are first-line therapy with ACE inhibitors (NICE, 2010). Evidence from recent trials has shown that, when combined with diuretics and ACE inhibitors, beta-blockers improve symptoms, lower morbidity and reduce hospital admissions (CIBIS-II Investigators and Committees, 1999). Beta-blockers improve heart failure by reducing heart rate and myocardial oxygen demand. As with ACE inhibitors, they should be started with a low dose and titrated slowly, paying special attention to blood pressure and heart rate, until the tolerated target dose has been achieved.

Aldosterone antagonists licensed for heart failure are an option for second-line treatment (NICE, 2010). They are usually reserved for moderate to severe heart failure (NYHA class III–IV) or for patients who have had an MI in the past month.

This medication should be used with specialist heart failure advice; while taking it, potassium, creatinine and glomerular filtration rate (GFR) should be monitored closely for hyperkalaemia or deterioration in renal function.

Aspirin is often part of the heart failure patient’s polypharmacy, especially when the patient has a history of heart failure associated with CHD. It is used as an anti-platelet. There is no evidence for its use as an anti-heart failure medication.

Anticoagulants are prescribed for heart failure patients with AF, or sinus rhythm with a history of thromboembolism, left ventricular aneurysm or intracardiac thrombus.

Vasodilators: hydralazine (principally an arteriolar dilator) in particular is often used in combination with isosorbide dinitrate, in moderate to severe heart failure when ACE inhibitors or ARBs are not tolerated. Their use has been shown to improve cardiac output, despite uncertainty around their mode of action.

Digoxin is often used in patients with systolic dysfunction who remain symptomatic despite optimal therapy, although this is not based on evidence. Heart failure patients with AF may have digoxin prescribed to control heart rate.

Table 2 summarises common pharmacological treatments for heart failure. Practitioners should check the British National Formulary for cautions, contraindications, side-effects and doses.

### Non-pharmacological

While drug therapy remains the mainstay of treatment, non-pharmacological intervention improves management. It consists of education, lifestyle advice and exercise where appropriate. Device therapy should also be considered. Finally, patients with end-stage heart failure should be considered for palliative care.

### Education

should include information on the nature of the patient’s condition, medication and management plan. This should be supported with current, relevant literature to enable patients to gain a better understanding.

### Lifestyle

should be assessed and changes made where appropriate:

- Smoking should be avoided in all cases; cessation advice should be provided and support given because many patients find this difficult;
All patients should keep alcohol consumption within recommended levels as it is a myocardial depressant. For those whose heart failure is thought to be alcohol related, abstinence is imperative. Some patients may need considerable help in achieving this;

Dietary advice should be tailored to individual patients. Those with marked fluid retention need to reduce their salt intake, so they should be advised not to add it while cooking or at the table. Although there is no recommended level for this group, it is suggested that salt intake is limited to <3g per day (Gardner et al, 2007). Advice from the Food Standards Agency (2009) for healthy people is to remain below 6g (one teaspoon). Patients with heart failure should be advised to avoid low-salt substitutes as their high potassium content may adversely interact with medications, for example aldosterone antagonists. Those with weight loss (cardiac cachexia) may need referral to a dietician for calorie supplementation;

Weight management is particularly important for patients with heart failure. Those with an accumulation of fluid may need to restrict fluid intake with supervision from a health professional if their symptoms are severe. Patients are advised to monitor their weight and any weight gain or loss of >1.5kg (>3lb) over three days should be reported to their health professional for advice. They should weigh themselves every morning after going to the toilet to pass water, before getting dressed and before eating or drinking anything;

Immunisation for influenza is recommended yearly and a once-only vaccination for pneumococcal infection should be offered (NICE, 2010);

Contraception should be discussed with women heart failure patients of childbearing age as the risk of mortality is high during pregnancy and birth;

Exercise is recommended in NICE guidelines (2010), as inactivity can lead to physical deconditioning and worsening of symptoms. Patients should be encouraged to adopt regular aerobic and/or resistance exercise in conjunction with pharmacological therapy as part of their management plan. Exercise is best started within a supervised group exercise-based rehabilitation programme, to assess risk factors and devise an individual programme.

BOX 4. COMMON CLINICAL SYMPTOMS DURING END-STAGE HEART FAILURE

- Breathlessness
- Fatigue
- Cough
- Orthopnoea
- Ankle swelling
- Limitation in physical activity
- Anxiety
- Depression
- Palpitations
- Sleeplessness
- Anorexia
- Pain
- Thirst
- Confusion
- Dizziness
- Nausea
- Nocturia
- Urinary incontinence
- Constipation
- Diarrhoea
- Pruritus

Source: NHS Heart Improvement Programme (2007)

Device therapy (implantable defibrillator and biventricular pacemaker) addresses two potential consequences of left ventricular dysfunction – sudden cardiac death and ventricular dyssynchrony. In a healthy heart, both ventricles pump at the same time, efficiently pumping blood around the body while, in severe heart failure, they may not pump simultaneously. Inserting a biventricular pacemaker, also known as cardiac resynchronisation therapy, may restore synchrony to the ventricles, which may reduce symptoms due to better pumping ability.

Implantable cardiac defibrillators (ICDs) can address the problem of sudden cardiac death (a potential problem in heart failure) by delivering an electric shock to the heart to stop the ventricular arrhythmia and restore the heart to a normal rhythm.

Palliative care should be offered to all patients with end-stage heart failure. The condition is considered to be “end stage” when they experience intractable symptoms (Box 4) despite all optimal care and interventions. The focus of care should shift from mainly aiming to prolong life to relieving symptoms. This is an area of expanding practice as a considerable number of patients with heart failure have end-of-life needs that are unrecognised and unmet (National Council for Palliative Care, 2011).

Conclusion

Heart failure is a common, complex syndrome with a poor prognosis. The most common aetiology in the UK is CHD. Common symptoms include breathing difficulties, fatigue and peripheral oedema.

The prognosis can be improved along with reduced symptoms if early diagnosis is made and treatment is started promptly. Treatment aims to improve patients’ life expectancy and quality of life. However, there is still much work to be done to meet the needs of those with end-stage heart failure.

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