MANAGEMENT OF HEART FAILURE

Management of acute pulmonary oedema

This needs urgent treatment:

Sit the patient up in order to reduce pulmonary congestion.

Give oxygen (high flow, high concentration). Non-invasive positive pressure ventilation (continuous positive airways pressure, CPAP, of 5-10 mmHg) by a tight-fitting face mask results in a more rapid improvement in the patient's clinical state.

Administer nitrates (e.g. i.v. glyceryl trinitrate 10-200 μg/min titrated upwards every 10 minutes, until clinical improvement occurs or systolic blood pressure falls to < 110 mmHg.

Administer a loop diuretic such as furosemide 50-100 mg i.v.

The patient should initially be kept on strict bed rest with continuous monitioring, including cardiac rhythm, blood pressure and pulse oximetry.

Intravenous opiates may be cautiously used when patients are in extremis. They reduce sympathetically mediated peripheral vasoconstriction but run the risk of respiratory depression and exacerbation of hypoxia and hypercapnia.

If these measures prove ineffective, inotropic agents may be required to augment cardiac output, particularly in hypotensive patients. Insertion of an intra-aortic balloon pump can be very beneficial in patients with acute cardiogenic pulmonary oedema, especially when secondary to myocardial ischaemia.

MANAGEMENT OF CHRONIC HEART FAILURE

General measures

Effective education of patients and their relatives about the causes and treatment of heart failure can help adherence to a management plan . Some patients may need to weigh themselves daily, and adjust their diuretic therapy accordingly.

In patients with coronary heart disease, secondary preventative measures such as low-dose aspirin and lipid-lowering therapy are required .

Drug therapy

Cardiac function can be improved by increasing contractility, optimising preload or decreasing afterload. Drugs that reduce preload are most appropriate in patients with high end-diastolic filling pressures and evidence of pulmonary or systemic venous congestion (backward failure); drugs that reduce afterload or increase myocardial contractility are more useful in patients with signs and symptoms of a low cardiac output (forward failure).

GENERAL MEASURES FOR THE MANAGEMENT OF HEART FAILURE

Education

Explanation of nature of disease, treatment and self-help strategies

Diet

Good general nutrition and weight reduction for the obese

Avoidance of high-salt foods and added salt, especially for patients with severe congestive heart failure

Alcohol Moderate or eliminate alcohol consumption. Alcohol-induced cardiomyopathy requires abstinence

Smoking Stopping

Exercise Regular moderate aerobic exercise within limits of symptoms

Vaccination Influenza and pneumococcal vaccination should be considered

D IURETICS

These are usually the first-line treatment.. In heart failure, diuretics produce an increase in urinary sodium excretion, leading to a reduction in blood and plasma volume, and may also cause a small but significant degree of arterial and venous dilatation. Diuretic therapy will therefore reduce preload and improve pulmonary and systemic venous congestion; it may also cause a small reduction in afterload and ventricular volume, leading to a fall in wall tension and increased cardiac efficiency.

Although a fall in preload (ventricular filling pressure) tends to reduce cardiac output, the 'Starling curve' in heart failure is flat so there may be a substantial and beneficial fall in filling pressure with little change in cardiac output . Nevertheless, excessive diuretic therapy may cause an undesirable fall in cardiac output, with a rising blood urea, hypotension and increasing lethargy, especially in patients with a marked diastolic component to their heart failure.

In some patients with severe chronic heart failure, particularly in the presence of chronic renal impairment, oedema may persist despite oral loop diuretics. In such patients an intravenous infusion of furosemide 10 mg/hr for example, can initiate a diuresis. Also combining a loop diuretic with a thiazide may prove effective; however, such combinations can produce an excessive diuresis.

Aldosterone receptor antagonists such as spironolactone are potassium-sparing diuretics that are of particular benefit in patients with heart failure. They may cause hyperkalaemia, particularly when used with an ACE inhibitor. They improve long-term clinical outcome in patients with severe heart failure and those with heart failure following acute myocardial infarction.

Vasodilators

. These drugs are also valuable in chronic heart failure; venodilators (e.g. nitrates) reduce preload, and arterial dilators (e.g. hydralazine) reduce afterload . However, their use is limited by pharmacological tolerance and hypotension.

Angiotensin-converting enzyme (ACE) inhibitors

The development of these drugs has been a major advance in the treatment of heart failure. They interrupt the vicious circle of neurohormonal activation that is characteristic of moderate and severe heart failure by preventing the conversion of angiotensin I to angiotensin II, thereby preventing salt and water retention, peripheral arterial and venous vasoconstriction, and activation of the sympathetic nervous system (Fig. 18.26). They also prevent the undesirable activation of the renin-angiotensin system caused by diuretic therapy.

The major benefit of ACE inhibitor therapy in heart failure is a reduction in afterload; however, there may also be an advantageous reduction in preload and a modest increase in the plasma potassium concentration. Treating heart failure, with a combination of a loop diuretic and an ACE inhibitor therefore has many potential advantages.

Clinical trials have shown that in moderate and severe heart failure, ACE inhibitors can produce a substantial improvement in effort tolerance and in mortality. ACE inhibitors can also improve outcome and prevent the onset of overt heart failure in patients with poor residual left ventricular function following myocardial infarction .

ACE INHIBITORS AND TREATMENT OF CHRONIC HEART FAILURE

'ACE inhibitors in chronic heart failure due to ventricular dysfunction reduce mortality and readmission rates .

ACE INHIBITORS AND PREVENTION OF THE DEVELOPMENT OF HEART FAILURE

'ACE inhibitors can delay the development of symptomatic heart failure and reduce the frequency of cardiovascular events (death, myocardial infarction, hospitalisation) in patients with asymptomatic left ventricular systolic dysfunction.

Unfortunately, these drugs can cause profound hypotension with postural symptoms and a deterioration in renal function (especially in patients with bilateral renal artery stenosis or pre-existing renal disease). Moreover, there may be a potentially catastrophic fall in blood pressure following the first dose of an ACE inhibitor, particularly if the drug is started in the presence of hypotension, hypovolaemia or hyponatraemia due to prior diuretic therapy, especially in the elderly. In stable patients without hypotension (systolic BP > 100 mmHg), ACE inhibitors can usually be started in the community without problems. However, in other patients it is usually advisable to withhold diuretics for 24 hours before starting treatment with a low dose, while the patient is supine and under observation. If hypotension occurs, this can be counteracted by elevating the foot of the bed and administering intravenous saline. Renal function must be monitored and should be checked 1-2 weeks after starting therapy.

Angiotensin receptor blockers

ACE INHIBITOR DOSAGES IN HEART FAILURE

Starting dose Target dose

Enalapril 2.5 mg 12-hourly 10 mg 12-hourly

Lisinopril 5 mg daily 20 mg daily

Ramipril 1.25 mg 12-hourly 5 mg 12-hourly

ANGIOTENSIN RECEPTOR BLOCKERS (ARBs) AND CHRONIC HEART FAILURE

'Compared with ACE inhibitors, ARBs are better tolerated and have similar efficacy in reducing cardiovascular events. ARBs reduce cardiovascular morbidity and mortality in patients with symptomatic heart failure who are intolerant of ACE inhibitors.

Examples include losartan 50-100 mg once daily, candesartan 4-16 mg daily or valsartan 40-160 mg daily. These drugs act by blocking the action of angiotensin II on the heart, peripheral vasculature and kidney; in heart failure, they produce beneficial haemodynamic changes that are similar to the effects of ACE inhibitors . They have comparable effects on mortality and are a useful alternative for patients who cannot tolerate ACE inhibitors .Unfortunately, they share all the more serious adverse effects of ACE inhibitors including renal dysfunction.

Beta-adrenoceptor antagonists (β-blockers)

These drugs may help to counteract the deleterious effects of enhanced sympathetic stimulation and reduce the risk of arrhythmias and sudden death. When initiated in standard doses they may precipitate acute-on-chronic heart failure, but when given in small incremental doses (e.g. bisoprolol started at a dose of 1.25 mg daily, and increased gradually over a 12-week period to a target maintenance dose of 10 mg daily) under carefully monitored conditions, they can increase ejection fraction, improve

symptoms, reduce the frequency of hospitalisation and reduce mortality in patients with chronic heart failure .

β-BLOCKERS AND TREATMENT OF CHRONIC HEART FAILURE

'Adding oral β-blockers gradually in small incremental doses to standard therapy including ACE inhibitors in people with heart failure reduces the rate of death or hospital admission.

Digoxin

This should be used as first-line therapy in patients with heart failure and atrial fibrillation, when it usually provides adequate control of the ventricular rate together with a small positive inotropic effect. The role of digoxin in the treatment of patients with heart failure and sinus rhythm is less certain; in a large randomised controlled trial, treatment with digoxin had no effect on overall survival but did reduce the need for hospitalisation. .

Amiodarone

This is a potent anti-arrhythmic drug which has little negative inotropic effect and may be valuable in patients with poor left ventricular function. It is only effective in the treatment of symptomatic arrhythmias, and should not be used as a preventative agent in the asymptomatic.

Implantable cardiac defibrillators and resychronisation therapy

Patients with symptomatic ventricular arrhythmias and heart failure have a very poor prognosis. Irrespective of their response to anti-arrhythmic drug therapy, all should be considered for implantation of a cardiac defibrillator . In patients with marked intraventricular conduction delay, prolonged depolarisation may lead to uncoordinated left ventricular contraction. When this is associated with severe symptomatic heart failure, cardiac resynchronisation therapy may be considered. Here, both the left and right ventricles are paced simultaneously in an attempt to generate a more coordinated left ventricular contraction and improve cardiac output.

Revascularisation

Coronary artery bypass surgery or percutaneous coronary intervention may improve function in areas of the myocardium that are 'hibernating' because of inadequate blood supply, and can be used to treat carefully selected patients with heart failure and coronary artery disease. If necessary, 'hibernating' myocardium can be identified by stress echocardiography and specialised nuclear techniques.

CONGESTIVE CARDIAC FAILURE IN OLD AGE

Common causes: coronary artery disease, hypertension and calcific degenerative valvular disease.

Diastolic dysfunction: often prominent, particularly in those with a history of hypertension.

ACE inhibitors: improve symptoms and mortality but more frequently associated with postural hypotension and renal impairment than in younger patients.

Loop diuretics: usually required but may be poorly tolerated in those with urinary incontinence and men with prostate enlargement.

Although cardiac transplantation usually produces a dramatic improvement in the recipient's quality of life, serious complications may occur: