It is of paramount importance to realise that the best way to treat chronic heart failure (CHF) is to prevent it happening in the first place by modifying known risk factors for ischaemic heart disease (IHD) and better treatment of myocardial infarction (MI). An aetiology should always be sought for CHF as its reversal may subsequently improve cardiac function. Once established, CHF has a poor prognosis – worse than many forms of cancer. Treatment aims are to reduce mortality and relieve symptoms. It is encouraging that recent improvements in disease-modifying therapy have markedly improved both morbidity and mortality.

**Disease-modifying therapy**

**Angiotensin-converting enzyme inhibitors**

Angiotensin-converting enzyme inhibitors (ACEIs) are the first-line drugs which should be given to all patients with left ventricular systolic dysfunction (LVSD) whether symptomatic or not, combined with a diuretic if there is evidence of cardiac decompensation (e.g. peripheral or pulmonary oedema) (Fig 1). These drugs reduced both morbidity and mortality in clinical trials involving more than 7,000 patients (on average, a 20–25% relative risk (RR) reduction). Unless there is a contraindication, such as significant renal disease, angioedema or ACEI-induced cough, their use is mandatory.

**Beta-blockers**

There is also now unequivocal evidence that the beta-blockers bisoprolol, carvedilol, and metoprolol reduce mortality and long-term symptoms in patients with all grades of CHF and in post-MI LVSD. Their use is imperative in patients who are free of cardiac decompensation and should be uptitrated slowly – ‘start low, go slow’. Neither chronic obstructive airways disease without airways reversibility nor mild to moderate peripheral vascular disease should be seen as a contraindication to beta-blocker therapy.

**Aldosterone antagonists**

Spironolactone reduces mortality and morbidity in patients with moderate to severe CHF (New York Heart Association (NYHA) classes III and IV) when used in
addition to standard therapy (ACEI and beta-blocker). Eplerenone has similar effects in post-MI LVSD.

**Caution:** these drugs can impair renal function and cause significant hyperkalaemia.

**Angiotensin II receptor antagonists**

Angiotensin II receptor antagonists are an effective alternative in truly ACE intolerant individuals, although ACEIs should always be regarded as the first-line drugs of choice. In the recent CHARM study (see end of text for trial acronyms), the addition of candesartan to existing triple therapy (ACEI (100% of patients), beta-blocker (55% of patients) and spironolactone (17% of patients)) reduced cardiovascular death and hospitalisation for CHF by 15%. This trial suggests that patients remaining symptomatic despite ACEI and beta-blocker may now have another therapeutic option.

**Hydralazine and nitrates**

Hydralazine and nitrates can also be considered in patients intolerant to both ACEIs and angiotensin-receptor antagonists.

**Symptomatic therapy**

**Diuretics**

Diuretics still have an important place in the management of both the symptoms and signs of fluid retention in CHF. Diuretics have not been shown to lower mortality – indeed, higher doses are associated with a poorer outcome. Therefore, the dose of diuretic should be as low as possible, with fluid restriction as necessary. Usually a loop diuretic, such as frusemide or bumetanide is necessary. Once doses of, for example, 80 mg frusemide bd are reached, sequential nephron blockade with a thiazide diuretic (eg bendrofluazide or metolazone) may relieve signs of fluid retention with greater efficacy than further increases in the dose of loop diuretic. Renal function should be monitored closely and hypokalaemia corrected.

**Digoxin**

The role of digoxin appears less clear. The current recommendations (not based on evidence) are to introduce this drug in patients who remain symptomatic despite maximal medical therapy or to provide rate control for patients with atrial fibrillation (AF). Patients with AF were excluded in large digoxin studies, and the death rate was unaltered. Indeed, post-hoc analyses of the DIG trial have shown that more women died when they took digoxin than those who did not. In another study, men with serum digoxin concentrations above 1.2 ng/ml had a higher mortality than patients receiving placebo. Digoxin is best reserved for patients who remain markedly symptomatic, with large hearts and frequent hospitalisations. It should be avoided in those with ventricular arrhythmias.

**Other pharmacological therapy**

Patients with IHD should be on aspirin unless there is a specific contraindica-
tion. The benefits of warfarin therapy in CHF are currently uncertain, except in patients with AF or known LV thrombus. This is the subject of two large clinical trials in CHF (WATCH and WASH).

Statin therapy is of benefit in both primary and secondary prevention in IHD, but its safety in CHF is still being investigated. Statins may lower mortality in non-ischaemic NYHA III/IV CHF (RR = 0.35; confidence interval = 0.13–0.96; \( p = 0.042 \)).

Long-term use of high-dose allopurinol may lower mortality, possibly by negating the adverse effect of an elevated blood urate concentration. Clinical trials to address this question are ongoing.

Anaemia is common in CHF and is an independent predictor of mortality. Initial reports have demonstrated that treating patients with moderate/severe CHF with erythropoietin and intravenous iron improves their cardiac function and reduces their need for hospital admission.

It is important to avoid drugs that may exacerbate CHF, for example non-steroidal anti-inflammatory drugs, rate-limiting calcium antagonists (diltiazem and verapamil), class I anti-arrhythmic drugs, steroids and tricyclic antidepressants.

**Non-pharmacological intervention and lifestyle modification**

Lifestyle modifications should be encouraged, for example stopping smoking and alcohol, losing excess weight and taking regular aerobic exercise. Annual immunisation for influenza and pneumococcus is recommended. Fluid intake should be restricted to 1.5–2 litres/day and patients counselled on the importance of monitoring their fluid balance (eg daily weights). Salt rich foods should be avoided.

### Device therapy

**Implantable cardioverter defibrillators**

Patients with LVSD secondary to IHD with an LV ejection fraction below 35% benefit in terms of mortality reduction from implantable cardioverter defibrillators (ICD). Currently, ICDs are implanted into patients with IHD and LVSD with ventricular arrhythmia, but implementing these results in all patients with LVSD has huge cost implications.

**Cardiac resynchronisation therapy**

Cardiac resynchronisation therapy (CRT) is the subject of much research in CHF. In combination with stable, optimal medical therapy, CRT may improve patient well-being by correcting ventricular dysynchrony. This is achieved by the insertion of pacing leads into the right atrium and right ventricle as per a standard dual chamber (DDD) pacemaker, but also by pacing the left ventricle via a lead placed in the coronary sinus.

CRT improves symptoms and haemodynamics, reduces hospitalisations and increases effort capacity in patients in NYHA classes III/IV with wide QRS complexes. Its effects on mortality are being studied in the CARE-HF trial. Combined CRT/ICD implantation reduces mortality in CHF (COMPANION Trial). The place of CRT alone in CHF remains to be determined.

### Left ventricular assist devices

Left ventricular assist devices (LVADs) are blood pumps that take over part of the heart’s pumping function when implanted alongside or within the heart. LVADs are also now emerging as treatment options in advanced heart failure either as a bridge to transplantation or to recovery, or as destination therapy (for patients with end-stage heart failure who require permanent mechanical heart support and are not suitable for cardiac transplantation).
Surgical options

In patients who fail to respond to medical or device therapy, surgical options include:

- revascularisation for hibernating myocardium
- ventricular remodelling (less commonly performed now), and
- cardiac transplantation.

Cardiac transplantation

Although donor organ availability restricts its use, cardiac transplantation remains an option for those patients with advanced heart failure who fail to respond to medical therapy. It carries a one-year mortality of around 19% and an average life expectancy of 10 years. Although patients must have severe LVSD to warrant this procedure, they must otherwise be well because of the impact of major surgery and the significant side effects of the immunosuppressive regimen.

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**Suggested further reading**


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**Atrial fibrillation: current perspectives**

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Dramatic advances in basic and clinical cardiac electrophysiology have rendered many arrhythmias amenable to cure by catheter ablation and have relegated anti-arrhythmic drugs to a subsidiary role. Unfortunately, however, the commonest sustained cardiac arrhythmia, atrial fibrillation (AF), remains incompletely understood and consequently difficult to manage. Nevertheless, important advances in our knowledge have recently been made; these will be reviewed in this article.

**Classification**

AF incorporates a range of subsets of which the mechanism and response to therapeutic intervention vary. A consensus on nomenclature has recently been achieved in an attempt to ensure appropriate management:

- An AF event is either the first detected or a recurrent episode.
- Paroxysmal AF describes episodes that terminate spontaneously within seven days.
- AF is termed persistent if it lasts longer than seven days or requires cardioversion by any means to restore sinus rhythm.
- Permanent AF is the term used when cardioversion has failed or has not been attempted.

**Mechanisms**

**Arrhythmias**

The mechanism of virtually all tachy-arrhythmias can be described as:

- re-entrant, where wavefronts of electrical activation propagate continuously around lines of electrical conduction block, or
- focal, where activation wavefronts spread from a discrete source of repetitive electrical discharge.

AF, however, consists of multiple, irregular, constantly varying wavefronts that cannot be easily analysed under physiological conditions in the human heart. As a consequence, much of our understanding has come from experimental studies in animal models and

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**Key Points**

**The ECG appearance of atrial fibrillation (AF) may result from different arrhythmia mechanisms**

**Electrical and structural remodelling are increasingly recognised as important influences on the natural history of AF and represent novel therapeutic targets**

All patients with AF should be considered for anticoagulation with warfarin, depending on their stroke risk

Recent studies have failed to demonstrate an advantage of anti-arrhythmic drugs over palliation by ventricular rate control

Catheter ablation of the atrioventricular node is safe and gives good symptom relief when ventricular rate control cannot be achieved with drugs

Curative catheter ablation by pulmonary vein isolation is gaining popularity although numerous questions remain about the technique

**KEY WORDS:** atrial fibrillation, CPD, curative therapies, mechanisms, review