

GPs have pivotal role in managing CHF

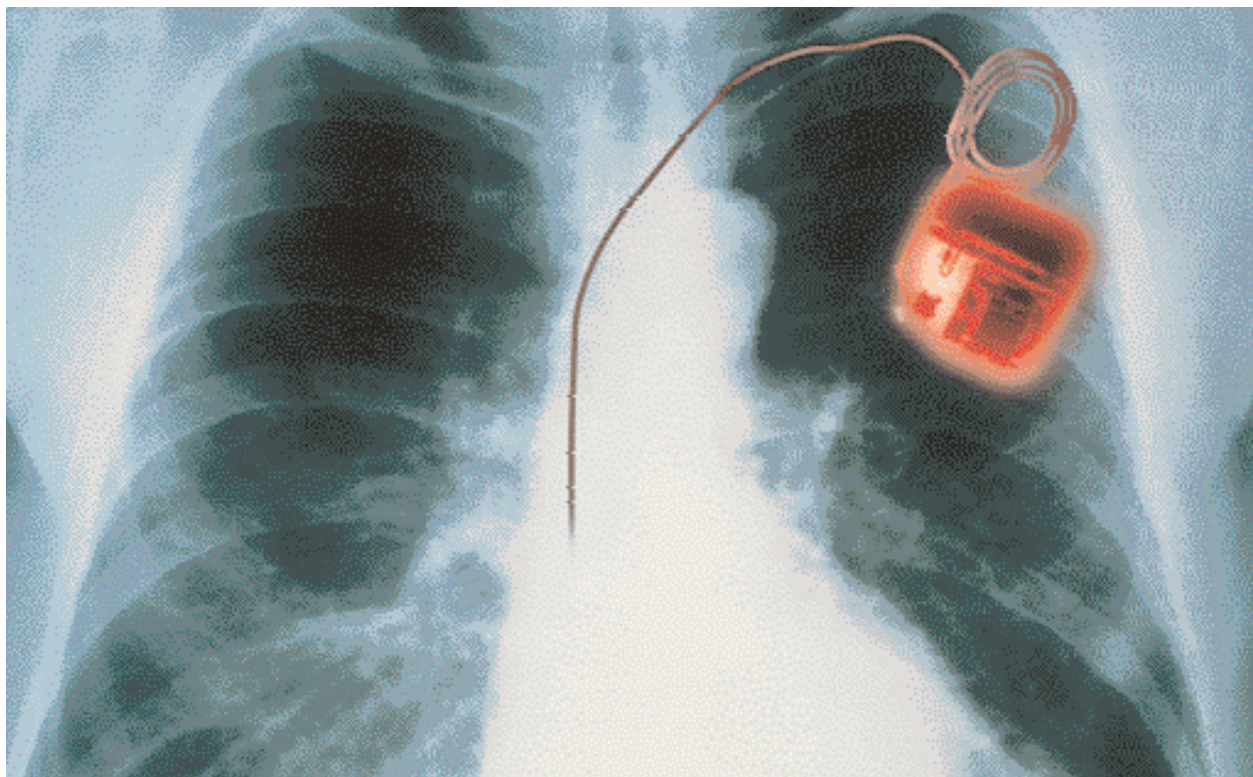
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How should breathlessness be investigated?

Which patients should be referred?

Who should receive beta-blocker therapy?



CHRONIC HEART FAILURE (CHF) IS A COMMON SYNDROME OF BREATHLESSNESS AND

fatigue in the presence of left ventricular (LV) dysfunction.¹ Its prevalence increases with age, reaching 10% in patients >80 years. Despite advances in medical and device therapies, patients with CHF still have frequent readmissions to hospital and an annual mortality rate approaching 10–15% per year.

An extensive network of community specialist nurses and GPs with a special interest in the area has led to the transfer of follow-up of many

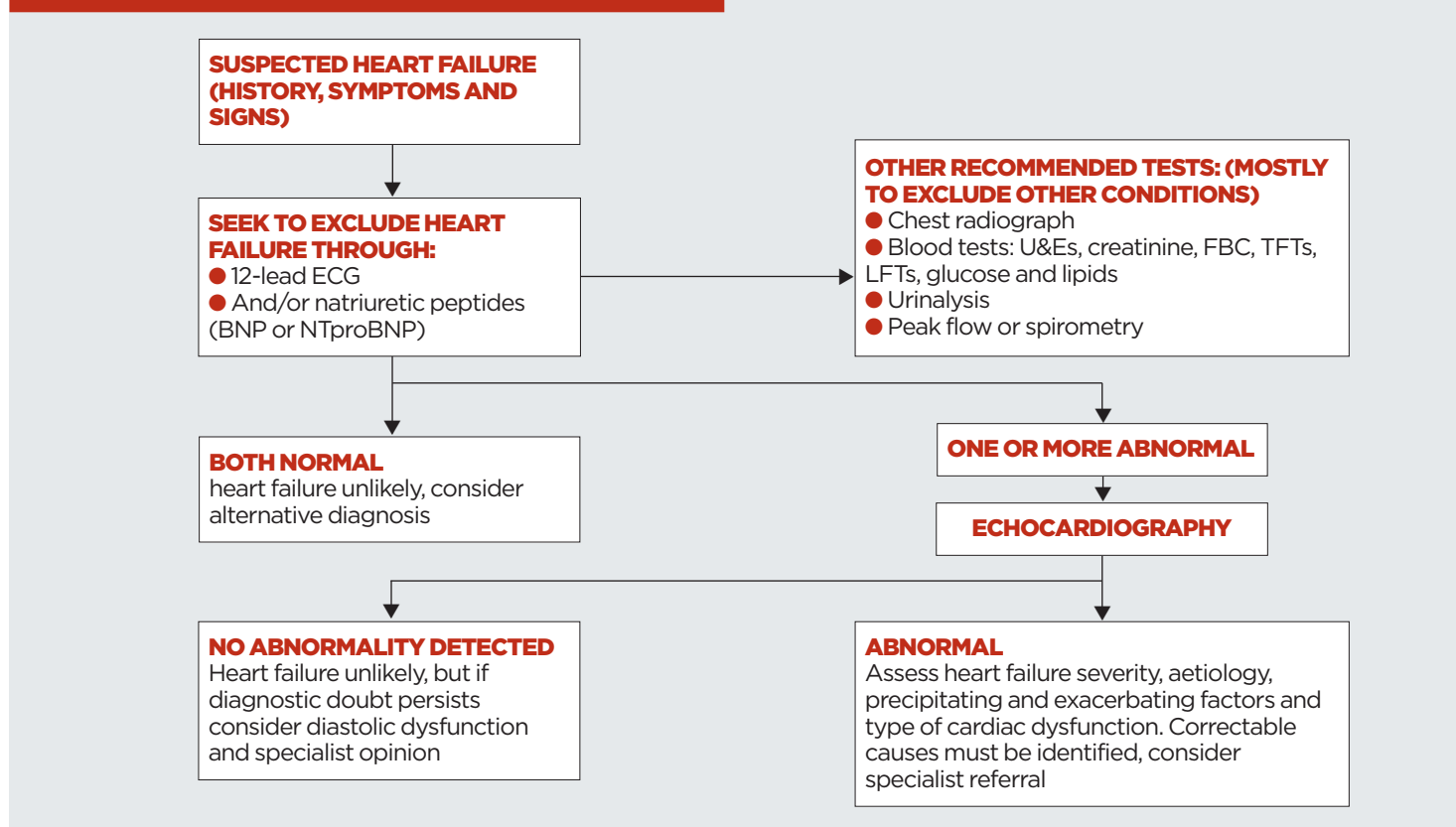
patients with CHF to primary care. Recent developments in the treatment of this malignant disease can have a significant impact on quality of life, hospitalisation rate and mortality. GPs should therefore have a low threshold for referring patients for specialist review and all patients newly diagnosed with CHF should be seen at least once by a physician with a special interest in heart failure.

DIAGNOSIS

Patients presenting with symptoms suggesting heart failure should have a full physical examination looking for signs of fluid overload, cardiac murmurs and extra sounds, and arrhythmia.

A 12-lead ECG is essential (see figure 1, p30). An entirely normal ECG makes a diagnosis of significant heart failure unlikely,² whereas conduction disease, such as left bundle branch block (LBBB) (see figure 2, p30), atrial fibrillation, ischaemia, previous infarction or left ventricular hypertrophy increase the likelihood of LV dysfunction.

Diagnoses presenting with similar symptoms include anaemia, renal failure and chronic airways disease. Therefore blood tests should be performed at presentation to assess renal function, full blood count and thyroid function. »

Figure 1. Algorithm for the diagnosis of heart failure


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In many areas, N-terminal brain natriuretic peptide (NTproBNP) testing is now available in primary care and, in combination with an ECG and symptoms, provides a cost-effective screening test.³ Brain natriuretic peptide is released by myocytes in response to stretching, and normal values effectively exclude cardiac dysfunction. Spirometry, if available, is also useful.

Most patients should have an echocardiogram, which should be performed and *interpreted* by an experienced technician or physician. This simple, noninvasive study can establish the presence of LV dysfunction, identify important and clinically relevant valvular disease, and guide therapy.

LEFT VENTRICULAR DYSFUNCTION

Most is known about CHF in patients with LV systolic dysfunction. However, a significant subset of patients with apparent heart failure do not have significant ventricular systolic dysfunction. These patients are usually

older women and the condition is sometimes known as 'diastolic heart failure'. This diagnostic grouping remains controversial and patients often have other contributing causes for breathlessness, such as lung disease, detraining and obesity. Some patients undoubtedly have diastolic LV function abnormalities, but the management of patients with 'diastolic heart failure' is less clear than for those with systolic dysfunction.

Patients with 'diastolic heart failure' have a better mortality rate than those with LV systolic dysfunction, although they do suffer ongoing morbidity and have a similar hospitalisation rate.

AETIOLOGY AND PROGNOSIS

CHF is a syndrome of symptoms and signs in the presence of LV dysfunction (see table 1, p32). It is not in itself a definitive diagnosis and an attempt should be made »

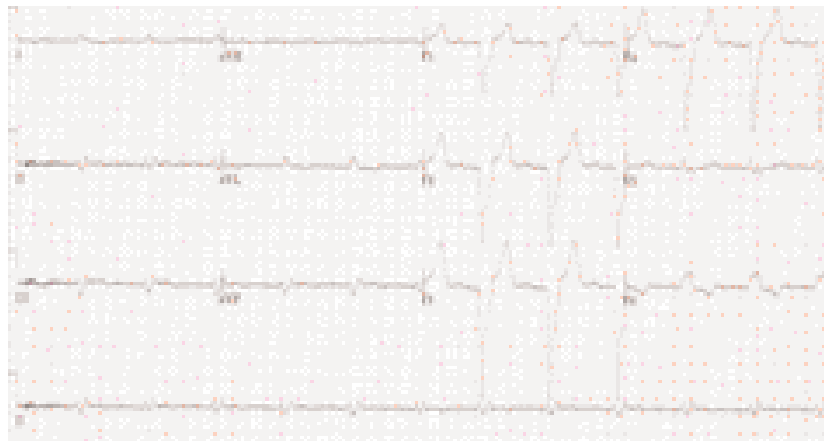


FIGURE 2
Left bundle branch block in a patient with CHF

Table 1**New York Heart Association (NYHA) symptom classification in CHF**

Class	Symptoms
I	No limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations
II	Slight limitation of physical activity: comfortable at rest but ordinary exercise results in fatigue, palpitations or dyspnoea
III	Marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms
IV	Unable to carry out any physical activity without discomfort: symptoms of heart failure are present even at rest with increased discomfort with any physical activity

Table 2**Aetiology of chronic heart failure****Common**

- Ischaemic heart disease
- Dilated cardiomyopathy
- Hypertension
- Valvular disease
- Arrhythmia

Less common

- Viral myocarditis
- Thyrotoxicosis
- Haemochromatosis
- Amyloid heart disease
- Parasitic infections

Table 3**High-risk features in CHF**

- NYHA class III or IV
- Recent decompensation requiring hospitalisation
- New or established left bundle branch block
- Renal impairment
- Hypotension (<90mmHg systolic)
- Syncope
- Anaemia

to establish the underlying aetiology in all patients. This often includes the exclusion of ischaemic heart disease, valvular disease, long-standing hypertension and other aetiologies (see table 2, left).

For many patients, coronary angiography is used to exclude ischaemic heart disease.

A formal assessment of exercise capacity is often performed. Peak oxygen consumption (pVO_2) during an incremental exercise test is a powerful predictor of prognosis,⁴ and a low pVO_2 is helpful in selecting patients for cardiac transplantation.

Useful markers of high risk in patients with newly diagnosed CHF or those under follow-up include renal impairment, new or previous LBBB, recent hospital admission and anaemia (see table 3, left).

TREATMENT

The management of patients with CHF has changed rapidly in the past two decades, from being centred solely around improving cardiac function and managing fluid overload to treating the chronic neurohormonal activation (see figure 3, p34).

Non-pharmacological treatment

The management of patients with CHF should involve a multidisciplinary team, often coordinated by the GP. The term 'heart failure' can cause anxiety and patients benefit greatly from education about their condition and its treatment. Fluid and salt restriction, weight control and the benefits of regular exercise should be addressed. Patients should be strongly advised to stop smoking.

Diuretics

Diuretics have never been shown to give a prognostic benefit in a randomised controlled trial, but are essential in managing the symptoms of congestion in patients with heart failure. The lowest dose of loop diuretic necessary to control symptoms should be used: high-dose therapy can cause rebound salt and water retention, and intravascular depletion can contribute significantly to renal dysfunction.

Combination therapy with a loop and thiazide diuretic is extremely potent and used in patients with resistant oedema under close supervision, with regular monitoring of renal function.

Furosemide has variable bioavailability, particularly in patients with peripheral oedema (and by inference gut oedema). Bumetanide,

with more predictable kinetics, can be used as an alternative (40mg furosemide = 1mg bumetanide).

Patients with persistent fluid overload in the presence of renal dysfunction should be referred to secondary care for intravenous therapy, often a 24-hour infusion of furosemide.

Gout is a common complication of diuretic therapy. Colchicine is helpful in managing acute attacks and does not cause fluid retention, unlike NSAIDs. Allopurinol is helpful in reducing recurrence.

ACE inhibitors and ARBs

ACE inhibitors and angiotensin-II receptor antagonists (ARBs) improve symptoms and prognosis and reduce hospitalisation for heart failure. All patients with CHF should be treated with an ACE inhibitor, preferably one shown to be effective in clinical trials such as enalapril, ramipril or lisinopril, unless they are contraindicated. Patients who experience side-effects, most frequently a cough or angioedema, can be switched to an ARB.

The most common concern when starting ACE inhibitor or ARB therapy is of inducing renal failure. Although renal artery stenosis is relatively common in patients with ischaemic heart disease,⁵ ACE inhibitor-induced renal failure caused by renal artery stenosis is rare.

An increase in creatinine occurs in almost all patients and a 10% increase is common and tolerable. Renal deterioration is worse in patients on excessive diuretic therapy, and the dose of diuretic can be reduced slightly to prevent a worsening of renal function. Greater increases in creatinine require either a further reduction in diuretic dose, a reduction in ACE inhibitor dose, or specialist review.

Beta-blockers

Sympathetic nervous system activation is a cardinal feature of CHF. It contributes to vasoconstriction and adverse remodelling of the left ventricle; provokes arrhythmias; causes cardiac myocyte apoptosis; and enhances renin-angiotensin system activation and hypokalaemia. Inhibiting the effect of sympathetic activation has had effects on survival as dramatic as those with ACE inhibitors, and beta-blocker »

therapy should be considered for all patients with CHF.

There is no upper age limit of benefit from beta-blocker therapy.⁶ Similarly, although beta-blocker use is associated with a small, asymptomatic reduction in FEV₁, patients with chronic airways disease but little wheeze should not be excluded.⁷ Even patients with very severe heart failure benefit from treatment.

Only bisoprolol and carvedilol should be used, as these are the only beta-blockers available in the UK that have been proven to improve prognosis.⁸ Nebivolol is licensed for patients >70 with mild to moderate heart failure.

Initiation of beta-blockers rarely precipitates admission or decompensation. However, although higher doses of beta-blockers are associated with a greater reduction in mortality, even in very elderly patients, the starting dose should be low and increased at intervals.

A typical schedule would be to start with carvedilol 3.125mg twice daily, doubling the dose at two-weekly intervals (see figure 4, right). The target should be the highest tolerated dose, or the dose shown to be of benefit in clinical trials (mean dose carvedilol 18mg bd, mean dose bisoprolol 8mg daily), whichever is the lower.

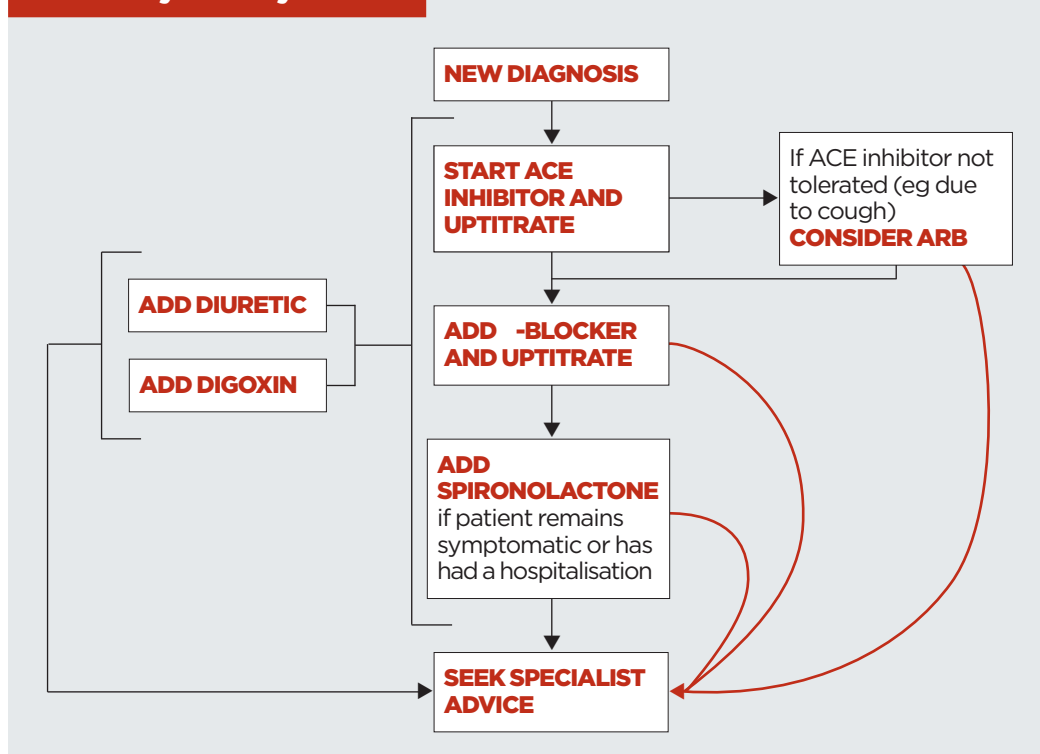
Carvedilol is as well tolerated in patients without diabetes as those with the condition and leads to a decrease in insulin resistance. Diabetes patients with heart failure have a similar reduction in mortality with carvedilol as patients without diabetes.

Peripheral vascular disease is also often thought to be a contraindication to the use of beta-blockers, but worsening of claudication has not been reported in trials of patients with heart failure.

Patients with normal blood pressure or hypertension will generally tolerate the initiation of beta-blocker therapy and uptitration well. In patients in whom there is a concern about hypotension, the loop diuretic dose can be reduced, or even omitted, on the first day of beta-blocker therapy. Alternatively, a slight reduction in ACE inhibitor or ARB dose will allow safe uptitration.

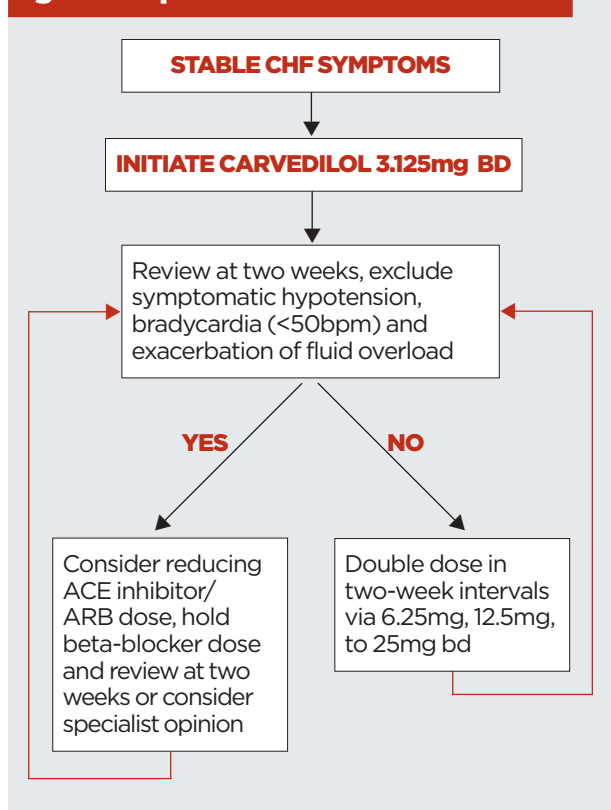
Concerns about the consequences of the haemodynamic changes are frequently not realised, even in those

Figure 3. Algorithm for the management of a patient with heart failure due to LV systolic dysfunction



National Institute for Clinical Excellence (2003) Chronic heart failure: Management of chronic heart failure in adults in primary and secondary care (NICE 2003). London: NICE. Available from www.nice.org.uk Reproduced with permission.

Figure 4. Uptitration of carvedilol in CHF



with systolic blood pressure around 100mmHg. Although symptoms of postural hypotension often recur early at each titration stage, they tend to settle once the patient is established on the increased dose.

In patients with symptomatic postural hypotension, it is probably the case that they should be on modest doses of an ACE inhibitor/ARB and a beta-blocker, rather than maximal doses of only one. In patients who develop symptomatic hypotension when starting beta-blocker therapy, it is best to persist by reducing the ACE inhibitor/ARB dose and starting again with the beta-blocker.

Spironolactone

Spironolactone is an aldosterone antagonist that reduces mortality and hospitalisation rates in patients with more severe CHF.⁹ It is also recommended in patients with a history of hospitalisation for CHF, and is useful in patients with fluid overload as an adjunct to loop diuretics.

The major side-effect of spironolactone is hyperkalaemia. Patients should be started on 12.5mg daily and the dose uptitrated to 25mg daily after two weeks, provided that their serum potassium is not above >>

key points

SELECTED BY

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Chronic heart failure (CHF) is a common syndrome of breathlessness and fatigue in the presence of left ventricular (LV) dysfunction. Its prevalence increases with age, reaching 10% in patients >80 years. It is not in itself a definitive diagnosis, and in all patients an attempt should be made to establish the underlying aetiology.

Patients presenting with symptoms suggesting heart failure should have a full physical examination looking for signs of fluid overload, cardiac murmurs and extra sounds, and arrhythmia. A 12-lead ECG is essential. An entirely normal ECG makes a diagnosis of significant heart failure unlikely, whereas conduction disease, such as LBBB, atrial fibrillation, ischaemia, previous infarction or left ventricular hypertrophy increase the likelihood of LV dysfunction. Other diagnoses presenting with similar symptoms include anaemia, renal failure and chronic airways disease. NTproBNP is a useful screening test.

All patients should have some form of cardiac imaging, which can establish the presence of LV dysfunction, identify important and clinically relevant valvular disease, and guide therapy.

Management involves fluid and salt restriction, weight control, regular exercise and advice to stop smoking. The lowest dose of loop diuretic necessary to control symptoms should be used. ACE inhibitors or ARBs should also be prescribed. They improve symptoms and prognosis and reduce hospitalisation for heart failure. Beta-blockers such as carvedilol and bisoprolol reduce sympathetic activation and improve survival. They may be used in most patients but the starting dose should be low and increased at intervals.

Spironolactone is an aldosterone antagonist that reduces mortality and hospitalisation rates in patients with more severe CHF. Digoxin is used in more severe heart failure for symptom relief.

Patients with heart failure and atrial arrhythmias should be offered anticoagulation unless contraindicated.

5.5mmol/l and renal function has remained stable.

Patients more likely to develop dangerous hyperkalaemia include those with more severe renal impairment, diabetes and those already taking high dose ACE inhibitors or ARBs. Other side-effects include gynaecomastia and an alternative agent, eplerenone, is available for patients who develop this side-effect.

Digoxin

Digoxin is widely used for symptom relief in patients with more severe heart failure. The only large randomised placebo-controlled trial investigating this therapy in heart failure demonstrated no mortality benefit, with a reduction in heart failure deaths matched by an increase in deaths caused by arrhythmia.¹⁰

Digoxin can also be used as an adjunct to beta-blockers for rate control in CHF patients with atrial fibrillation.

Anticoagulation

Patients with CHF are at a high risk of thromboembolic events and usually have underlying coronary artery disease. Most patients are therefore prescribed aspirin. However, aspirin does not improve prognosis, and may counter some of the beneficial effects of ACE inhibitors. GPs should therefore consider stopping aspirin in patients with 'resistant' fluid retention. Clopidogrel is an alternative to aspirin but there are few data supporting its routine use in CHF.

Anticoagulation with warfarin is associated with a reduction in CHF hospitalisations and a trend to reduced mortality compared with aspirin. Patients with heart failure and atrial arrhythmias should be offered anticoagulation in the absence of contraindications.

DEVICE THERAPY

Implantable cardioverter defibrillators

Patients with CHF, particularly those with a broad QRS (>120ms) and those with severe LV dysfunction (LV ejection fraction <30%), are at risk of sudden cardiac death due to ventricular tachyarrhythmia.

In patients with less severe symptoms (class II), an implantable cardioverter defibrillator (ICD) is associated with a reduction in total mortality.¹¹ Patients with more severe symptoms seem not to derive a mortality benefit from a defibrillator as they are more likely to die from worsening heart failure (which the ICD cannot treat) than sudden death.

Antiarrhythmic therapy with amiodarone is not associated with improved outcome and should not be used routinely.

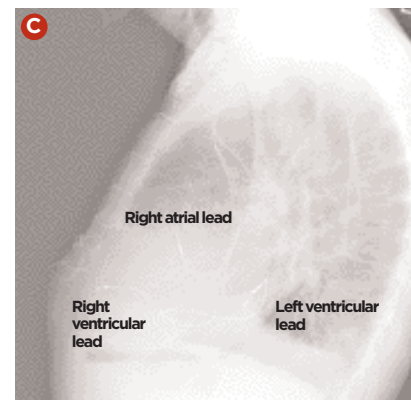
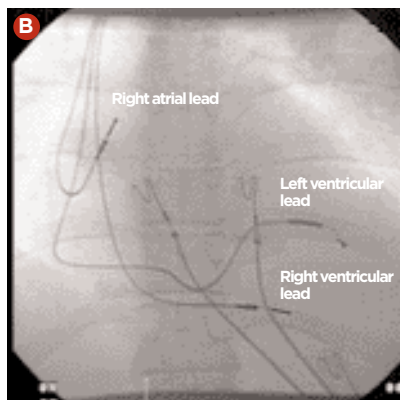
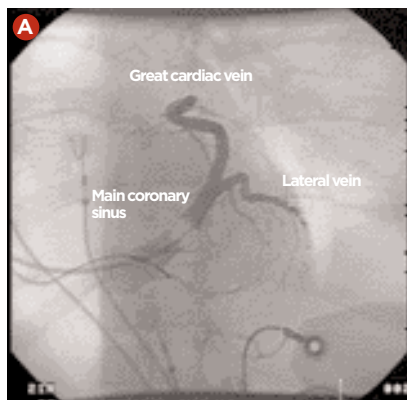
Cardiac resynchronisation therapy

LBBB is common in CHF patients and is associated with more severe LV dysfunction, more severe symptoms and an adverse prognosis. At least part of the reason for this is that ventricular activation proceeds slowly over the left ventricle, resulting in incoordinate ventricular contraction.

A cardiac resynchronisation therapy (CRT) pacemaker simultaneously paces the heart from the apex of the right ventricle and the lateral LV wall, thereby 'resynchronising' left ventricular contraction. CRT can improve symptoms, reduce hospitalisation rate and reduce total mortality due to heart failure and sudden death (see figure 5, below).¹²

More complex methods of assessing which patients will benefit from CRT have not been shown to be any better than a simple ECG demonstrating LBBB. There is no upper age limit »

FIGURE 5 CRT:
a) Contrast venogram of coronary sinus showing lateral vein suitable for left ventricular lead placement
b) AP chest fluoroscopy image showing right ventricular, right atrial and left ventricular leads in position
c) Lateral chest radiograph showing right ventricular, right atrial and left ventricular leads in position



for CRT and selected patients of 80 and older benefit to the same extent as younger patients.

Combined devices providing CRT and ICD function are available and can be considered in younger patients with few comorbidities and stable moderate symptoms.

ADVANCED THERAPY

Cardiac transplantation remains a possibility for a small number of patients with severely symptomatic heart failure, despite treatment. In addition, small impeller pumps are being developed, which may offer selected patients support pending transplantation, and for some, perhaps, an alternative to transplantation altogether. Selection for these options is a highly specialised task.

CONCLUSION

CHF is a common and malignant disease. There have been dramatic advances in treatments for CHF in recent years, and optimal management can lead to significant reductions in mortality and

hospitalisations and improvements in quality of life. However, many patients are still receiving suboptimal therapy, leading to unnecessary deaths and hospital admissions.

Since much of the follow-up and monitoring of patients with CHF is now appropriately performed in primary care, GPs and community heart failure nurses need to be confident at initiating and uptitrating medical therapy to the doses recommended in clinical trials. However, early subspecialist referral to a cardiologist with an interest in heart failure should be considered for patients with:

- New onset or worsening of symptoms
- High risk features (LBBB, renal impairment)
- Symptomatic hypotension
- Syncope
- Recent admission
- Onset of atrial fibrillation
- Symptomatic palpitations
- Resistance congestion
- Recurrent angina pectoris. ●

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