Heart failure (HF) is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the pumping ability of the heart. If HF is not a diagnosis in itself, it is a clinical syndrome – a collection of symptoms and signs (see Table ONE) – but the term is commonly used as a diagnostic label in clinical practice. HF is a progressive condition for which there is no cure; it has a dramatic effect on the quality of life of patients. A great deal can be done to manage HF more effectively, with better outcomes for patients and their families and with more cost-effective use of staff, resources, hospital beds and drugs.

### Descriptive Terms in HF

Many additional words or phrases are used to characterise patients with HF. The word “acute” in the context of HF has become confusing because some clinicians use the word to indicate severity (the medical emergency of life-threatening pulmonary oedema) and others use the word to indicate recent-onset, or even new-onset HF. The word is then an indicator of time rather than severity. The words acute, advanced, and decompensated should not be used interchangeably when applied to HF. A useful classification of HF based on the nature of the clinical presentation is shown in Table TWO. Worsening HF on a background of chronic HF (decompensation) is by far the most common form of HF leading to hospital admission.

### Table ONE: European Society of Cardiology definition of HF

<table>
<thead>
<tr>
<th>Patients with HF have the following features:</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Breathlessness at rest or on exercise, fatigue, tiredness, ankle swelling</td>
</tr>
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<td>• Objective evidence of structural or functional abnormality of the heart at rest (cardiomegaly, third heart sound, cardiac murmurs, abnormality on echocardiogram, raised natriuretic peptide level)</td>
</tr>
</tbody>
</table>

### Table TWO: European Society of Cardiology (ESC) Classification of Heart Failure

<table>
<thead>
<tr>
<th>New onset HF</th>
<th>First presentation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute or slow onset</td>
<td></td>
</tr>
<tr>
<td>Transient HF</td>
<td>Recurrent or episodic</td>
</tr>
<tr>
<td>Chronic HF</td>
<td>Persistent</td>
</tr>
<tr>
<td>Stable, worsening, or decompensated</td>
<td></td>
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</tbody>
</table>

### Introduction

Heart failure (HF) is a complex syndrome that can result from any structural or functional cardiac disorder that impairs the pumping ability of the heart. If HF is not a diagnosis in itself, it is a clinical syndrome – a collection of symptoms and signs (see Table ONE) – but the term is commonly used as a diagnostic label in clinical practice. If HF is a progressive condition for which there is no cure; it has a dramatic effect on the quality of life of patients. A great deal can be done to manage HF more effectively, with better outcomes for patients and their families and with more cost-effective use of staff, resources, hospital beds and drugs.

### Glossary of terms

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACE inhibitor</td>
<td>Angiotensin Converting Enzyme inhibitor</td>
</tr>
<tr>
<td>AF</td>
<td>Atrial Fibrillation</td>
</tr>
<tr>
<td>ARB</td>
<td>Angiotensin Receptor Blocker</td>
</tr>
<tr>
<td>ARR</td>
<td>Absolute Risk Reduction</td>
</tr>
<tr>
<td>BNP</td>
<td>Brain Natriuretic Peptide</td>
</tr>
<tr>
<td>CHD</td>
<td>Coronary Heart Disease</td>
</tr>
<tr>
<td>CRT</td>
<td>Cardiac Resynchronisation Therapy</td>
</tr>
<tr>
<td>EF</td>
<td>Ejection Fraction</td>
</tr>
<tr>
<td>ESC</td>
<td>European Society of Cardiology</td>
</tr>
<tr>
<td>HF</td>
<td>Heart Failure</td>
</tr>
<tr>
<td>HTN</td>
<td>Hypertension</td>
</tr>
<tr>
<td>JVP</td>
<td>Jugular Venous Pressure</td>
</tr>
<tr>
<td>LV</td>
<td>Left Ventricle/Left Ventricular</td>
</tr>
<tr>
<td>LVSD</td>
<td>Left Ventricular Systolic Dysfunction</td>
</tr>
<tr>
<td>Natriuresis</td>
<td>Excessive loss of cations and especially sodium in urine</td>
</tr>
<tr>
<td>NICE</td>
<td>National Institute for Health and Clinical Excellence</td>
</tr>
<tr>
<td>NYHA</td>
<td>New York Heart Association</td>
</tr>
<tr>
<td>NSAIDs</td>
<td>Non-steroidal anti-inflammatory drugs</td>
</tr>
<tr>
<td>RCT</td>
<td>Randomised Controlled Trial</td>
</tr>
<tr>
<td>RRR</td>
<td>Relative Risk Reduction</td>
</tr>
<tr>
<td>RV</td>
<td>Right Ventricle/Right Ventricular</td>
</tr>
<tr>
<td>SBP</td>
<td>Systolic Blood Pressure</td>
</tr>
<tr>
<td>SIGN</td>
<td>Scottish Intercollegiate Guidelines Network</td>
</tr>
</tbody>
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### Table ONE: European Society of Cardiology definition of HF

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</tr>
<tr>
<td>Persistent</td>
</tr>
<tr>
<td>Stable, worsening, or decompensated</td>
</tr>
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</table>

### Epidemiology of HF

Population prevalence rates have been estimated between 3-20 people per 1000, increasing to at least 80 cases per 1000 among people aged 75 and over. In Northern Ireland, the prevalence of HF is 7.56 per 1000. The median age of clinical presentation is 76 years. The male to female ratio is about 2:1. On average, a GP will look after 30 patients with HF, and suspect a new diagnosis of HF in perhaps ten patients annually.

The prevalence and population burden of HF is increasing. This is thought to be due to both an ageing population and to more people surviving acute heart attacks but left with residual left ventricular dysfunction.

### What are the causes of HF?

The most common cause of functional deterioration of the heart are damage or loss of heart muscle, acute or chronic ischaemia, increased vascular resistance with hypertension, or the development of a tachyarrhythmia such as atrial fibrillation. Coronary heart disease is by far the most common cause of myocardial disease, being the initiating cause in around 70% of patients with HF. Valve disease accounts for 10% and cardiomyopathies for another 10%.

### What is the prognosis with HF?

Heart failure often has a poor prognosis, with survival rates worse than for most cancers. Heart failure die within a year – but thereafter the mortality is less than 10% per year. There is evidence that people with HF have a worse quality of life than people with most other common medical conditions. Psychosocial...
function is impaired with over a third experiencing severe and prolonged depressive illness.12,13

Classification of HF severity
It is useful to have an estimate of the functional impact of HF on an individual patient. The New York Heart Association (NYHA) classification of severity of HF symptoms is widely accepted and valid.2 This scale, which has four levels, should be used as a measure of patient incapacity and severity of symptoms – see Table THREE.

<table>
<thead>
<tr>
<th>Class</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>No limitation: ordinary physical exercise does not cause undue fatigue, dyspnoea or palpitations.</td>
</tr>
<tr>
<td>II</td>
<td>Slight limitation of physical activity: comfortable at rest but ordinary activity results in fatigue, palpitations or dyspnoea.</td>
</tr>
<tr>
<td>III</td>
<td>Marked limitation of physical activity: comfortable at rest but less than ordinary activity results in symptoms.</td>
</tr>
<tr>
<td>IV</td>
<td>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.</td>
</tr>
</tbody>
</table>

What is the role of brain natriuretic peptide (BNP)?
Brain natriuretic peptide and N terminal-pro-BNP (NT-proBNP) are peptide hormones produced in the heart by breakdown of a precursor protein (pro-BNP). BNP causes natriuresis, diuresis, vasodilation and muscle relaxation; NT-proBNP is inactive.16 Plasma BNP and NT-proBNP concentrations are raised in patients with HF and the concentrations tend to rise with NYHA class.

Although simple single value cut-offs for the diagnosis of HF have been proposed, a more realistic interpretation of BNP and NT-proBNP is to suggest that very low values rule out HF, very high values make HF likely in the absence of other causes of raised BNP. Intermediate to high values should be regarded as indeterminate, necessitating further investigation. The upper limit of normal is age, sex and race dependent, and must be determined locally depending on the assay used.

BNP and NT-proBNP are suitable for widespread use as a screening test in patients with suspected chronic HF, assuming appropriate quality control of the assay and selection of appropriate cut-off values for the patients tested. BNP levels fall after commencing therapy for HF, e.g. diuretics, so the sensitivity is lower in patients who have already commenced treatment.

BNP or NT-proBNP levels and/or an electrocardiogram (ECG) should be recorded to indicate the need for echocardiography in patients with suspected HF. Echocardiography is recommended in patients with suspected HF who have either a raised BNP or N terminal-pro-BNP level or abnormal electrocardiogram result, to confirm the diagnosis and establish the underlying cause.

Patient education – a vital element of HF management
It is important to educate, and to periodically re-educate patients, family and carers about:17
• Diagnosis and causes of HF
• Lifestyle changes, exercise, smoking cessation
• Symptom recognition
• Fluid management/weight management
• Medications and concordance with medication regimens

Dosing regimens should be kept as simple as possible, and the healthcare professional should ensure that the patient and carer are fully informed about their medication.17 Advice should be given regarding the name, dose, timing and route of all medication. Desired effects and potential side effects should be discussed. Patients should be aware of the titration schedule of some medication and that improvement in symptoms, if any, may be gradual. The importance of concordance should be emphasised. Patients should contact their healthcare professional if they have any side-effects due to their medication.2

Patients with HF report high levels of frustration with progressive loss of function, social isolation and the stresses of monitoring a complex medical regimen. In one study, their reported understanding of their condition and involvement in the regimen was lower than that in a comparison group of patients with cancer.18 Among the same cohort, patients identified unmet needs in psychosocial care, education and co-ordination between primary and secondary care.19 HF is associated with a pattern of generalised cognitive impairment which includes memory and attention deficits.20 Healthcare professionals involved with educating or helping HF patients to manage their condition should be aware of the possibility of cognitive deficits and tailor interventions accordingly.19

National Service Framework for Coronary Heart Disease5
The National Service Framework (NSF) for Coronary Heart Disease (CHD) establishes clear standards for prevention and treatment of CHD that will lead to major improvements in quality and access. Chapter Six of the NSF covers HF5 and sets out how the NHS and others can:
• Help people with HF to live longer and achieve a better quality of life
• Help people with unresponsive HF receive appropriate palliative care support.

The standard of care that the NHS will aim for is that: “Doctors should arrange for people with suspected HF to be offered appropriate investigations (e.g. electrocardiography, echocardiography) that will confirm or refute the diagnosis. For those in whom HF is confirmed, its cause should be identified – the treatments most likely to both relieve symptoms and reduce their risk of death should be offered”.5

Quality and Outcomes Framework for the GMS contract
See Table FOUR.

| Table FOUR: Heart failure indicators and points in QOF 2011/12 |
|----------------|----------------|
| Indicator       | Points |
| HF1: The practice can produce a register of patients with HF | 4 |
| HF2: The percentage of patients with a diagnosis of HF which has been confirmed by an ECG or by specialist assessment | 6 |
| HF3: The percentage of patients with a current diagnosis of HF due to LVSD who are currently treated with an ACE inhibitor or angiotensin receptor blocker | 10 |
| HF4: The percentage of patients with a current diagnosis of HF due to LVSD who are currently treated with an ACE inhibitor or angiotensin receptor blocker, who are additionally treated with a beta-blocker licensed for HF, or recorded as intolerant to or having a contraindication to beta-blockers | 9 |
**What are the objectives of drug treatment in HF?**
Medical therapy has two aims.¹⁷
1. To improve morbidity: by reducing the patients’ symptoms, improving their exercise tolerance, reducing their hospitalisation rate and improving quality of life.
2. To improve the patients’ prognosis, through the reduction of all cause mortality or their HF-related mortality.

**What agents are considered to be first-line in the pharmacological treatment of HF?**
See the NICE HF treatment algorithm below.

Therapeutic options for HF have expanded and include a wide array of medications that are not without side-effects. This is one reason why the decisions on the management of HF have to take into account patients’ preferences. Involving the patient in the management decisions becomes an integral component of the management of patients.

**What should happen if the patient is already on a beta-blocker but it’s not licensed for HF?**
If the person is taking a beta-blocker for another co-morbidity (e.g. angina or hypertension), but this is not bisoprolol, carvedilol, or nebivolol, switch the person to one of these.²²

**What occurs if the person is still symptomatic despite optimal first-line treatment?**
If symptoms persist despite optimal first-line treatment, seek specialist advice and for second-line treatment consider adding:
- an aldosterone antagonist licensed for heart failure (especially in moderate to severe heart failure or MI in past month) or
- an ARB licensed for heart failure (especially in mild to moderate heart failure) or
- hydralazine in combination with nitrates (especially in people of African or Caribbean origin with moderate to severe heart failure).

**How should first-line treatment be initiated?**
Introduce one drug at a time, and once the person is stable on the first drug, add the second.²² For example, if prescribing an ACE inhibitor first:
- Prescribe a low dose and titrate upwards until the target dose or highest tolerated dose is reached. See later for further information on using ACE inhibitors in HF.
- Once stable, add a beta-blocker, unless contraindicated or the person is known to be intolerant of beta-blockers. Start at a low dose and titrate slowly upwards until the target dose or the highest tolerated dose is reached. Further information on using beta-blockers in HF is given later.

**What happens if the person is still symptomatic despite optimal treatment with an ACE inhibitor (or ARB) and a beta-blocker?**
Refer for specialist review and advice regarding the addition of further drug treatments. Treatments which may be recommended by a specialist include an aldosterone antagonist, an ARB in combination with an ACE inhibitor, hydralazine in combination with a nitrate, or digoxin.
**ACE inhibitors and Angiotensin-2 receptor antagonists in HF**

ACE inhibitors were first shown to be effective in HF in the 1980s. Since then, many RCTs have confirmed their benefit in patients with HF, or LVSD or both after M1-28 and in patients with asymptomatic LVSD. Meta-analyses have shown that in patients with chronic HF, treatment with an ACE inhibitor reduces the risk of admission to hospital for worsening HF and of death. ACE inhibitors also improve symptoms, exercise tolerance, quality of life, and exercise performance. The benefits tend to be more marked with more severe LVSD although there is benefit for all NYHA classes. The benefits of ACE inhibitors occur soon after the start of treatment and persist in the long-term. The benefits seem to be independent of age, sex and baseline use of diuretics, aspirin, and beta-blockers. The recommendation to give an ACE inhibitor to all people with HF and LVSD is consistent with guidelines from NICE, SIGN and ESC.1,17,21

**Key Point:** ACE inhibitors should be considered in patients with all NYHA functional classes of HF due to left ventricular systolic dysfunction.1,17,21

**Specialist advice required before starting ACE inhibitor if:**
- Urea > 12mmol/L
- Creatinine > 200micromol/L
- Sodium < 130mmol/L
- Systolic arterial pressure <100mmHg
- Diuretic dose > 80mg furosemide or equivalent
- Peripheral vascular disease or suspicion of renal artery stenosis
- Aortic stenosis
- Frail elderly

**Adverse effects of ACE inhibitors**

ACE inhibitors occasionally cause:

- **Worsening renal function** - some rise in urea and creatinine is expected after initiation of an ACE inhibitor and is not considered clinically important unless rapid and substantial. Check for nephrotoxic drugs such as NSAIDs. If necessary, reduce ACE inhibitor dose or discontinue. An increase in creatinine of up to 50% from baseline or to an absolute concentration of 265micromols/L, whichever is lower, is acceptable. If the creatinine rises above 265micromols/L, but below 310micromols/L, halve the dose of ACE inhibitor and monitor closely. If creatinine rises to 310micromols/L, or above, stop the ACE inhibitor immediately and monitor closely. Renal impairment is likely to occur in those with unsuspected (bilateral) renovascular disease. ACE inhibitor induced renal dysfunction is a possible indicator of renovascular disease and may warrant a MRI renal scan.21

**Hyperkalaemia** - check for use of other agents causing hyperkalaemia (e.g. potassium supplements, potassium-sparing diuretics) and stop these. If potassium rises above 5.5mmol/L, halve the dose of ACE inhibitor and monitor blood chemistry closely. If potassium rises over 6mmol/L, stop the ACE inhibitor and monitor blood chemistry closely.

Caution is needed when using potassium-sparing diuretics in conjunction with an ACE inhibitor or ARB. There is a risk of severe hyperkalaemia.32

**Symptomatic hypotension** - A fall in blood pressure may occur after the first dose, especially in hypotensive patients who are hyponatraemic and on large doses of diuretics. Dizziness often improves with time and patients should be reassured. Consider reducing the dose of diuretics and other hypotensive agents. Asymptomatic hypotension does not require intervention.1

**Cough** - around 10% of patients taking an ACE inhibitor experience a dry cough, perhaps related to bradykinin. If the cough is troublesome consider switching to an ARB. In addition, ACE inhibitors can occasionally cause angio-oedema, which can be life threatening (due to laryngeal involvement). Any patient who suffers angio-oedema should have the ACE inhibitor withdrawn immediately.21

**Contraindications to use of an ACE inhibitor**

An ACE inhibitor should only be used in patients with adequate renal function and normal serum potassium. Absolute contraindications include a history of angio-oedema with past exposure to ACE inhibitors, bilateral renal artery stenosis, pregnancy and cardiogenic shock.

**Starting an ACE inhibitor in a patient with HF**

The following are the steps for GP initiation of an ACE inhibitor in HF:1,2,17

**Step One:**
- Check renal function and serum electrolytes

---

**Table FIVE: Drugs used to attain objectives of therapy in chronic HF due to LVSD2**

<table>
<thead>
<tr>
<th>Condition</th>
<th>NYHA Class</th>
<th>Survival</th>
<th>Drugs for improving:</th>
<th>Quality of life</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Symptoms</td>
<td>Exercise capacity</td>
</tr>
<tr>
<td>Mild HF</td>
<td>II</td>
<td>ACE inhibitors Beta-blockers Hydralazine + ISDN</td>
<td>ACE inhibitors ARBs Digoxin Diuretics Vasodilators*</td>
<td>Diuretics Digoxin Hydralazine + ISDN</td>
</tr>
<tr>
<td>Moderate HF</td>
<td>III</td>
<td>ACE inhibitors Beta-blockers Aldosterone antagonists Hydralazine + ISDN</td>
<td>ACE inhibitors ARBs Aldosterone antagonists Digoxin Diuretics Vasodilators*</td>
<td>Diuretics Digoxin Hydralazine + ISDN</td>
</tr>
<tr>
<td>Severe HF</td>
<td>III - IV</td>
<td>ACE inhibitors Beta-blockers Aldosterone antagonists Hydralazine + ISDN</td>
<td>ACE inhibitors ARBs Aldosterone antagonists Digoxin Diuretics Vasodilators*</td>
<td>Diuretics Digoxin Hydralazine + ISDN</td>
</tr>
<tr>
<td>End stage HF</td>
<td>IV</td>
<td>ACE inhibitors Beta-blockers Aldosterone antagonists Hydralazine + ISDN</td>
<td>ACE inhibitors ARBs Aldosterone antagonists Digoxin Diuretics Vasodilators*</td>
<td>Diuretics Digoxin Hydralazine + ISDN</td>
</tr>
</tbody>
</table>

ISDN = isosorbide dinitrate
* vasodilators = nitrates, calcium channel blockers, potassium channel openers
If possible, stop potassium supplementation/potassium-sparing drugs
Stop NSAIDs where possible
Start ACE inhibitor at a low dose (see Table SIX)

Step Two: Within 1-2 weeks of starting ACE inhibitor -
• Review patient
• Check U&E, creatinine
• Check for adverse effects
• If no adverse effects, increase ACE inhibitor dose every 1-2 weeks to evidence-based target dose or maximum tolerated dose (see Table SIX)

Titrate to target dose over a period of one month. Measure serum urea, creatinine, electrolytes and eGFR after each dose increment.

Step Three
• Review patient after one month
• Check U&E, creatinine
• Check for adverse effects, including intolerable cough. Re-check renal function and serum electrolytes 1, 3 and 6 months after achieving maintenance dose and 6 monthly thereafter.

When should use of an ARB as an alternative to an ACE inhibitor be considered?
ARBs have good evidence in HF but no better than that of ACE inhibitors.\(^\text{25-38}\) In patients with HF due to LVSD, treatment with an ARB has been shown to reduce the risk of hospitalisation and reduce mortality compared with placebo.\(^\text{39,40}\)

An ARB can be considered as an alternative to an ACE inhibitor for patients with HF due to LVSD who have intolerable side effects (e.g. cough) with ACE inhibitors.\(^\text{1,17,21}\) Candesartan, losartan and valsartan are licensed in HF as alternatives to ACE inhibitors (where ACE inhibitors are contraindicated or not tolerated).\(^\text{32}\) Of these, only losartan is available generically.\(^\text{32}\)

One study (n=5,139) suggests that compared to losartan, candesartan improved survival in patients with HF.\(^\text{42}\)

Combining an ACE inhibitor with an ARB
Following specialist advice, ARBs can also be added to ACE inhibitor therapy in patients with chronic HF. Among the ARBs, only candesartan and valsartan are licensed for use in combination with an ACE inhibitor.\(^\text{32}\) Such a combination requires careful monitoring, particularly of renal function. The two key RCTs are Val-HEFT and CHARM-Added.\(^\text{40,43}\)

Patients with mild to severely symptomatic HF who had placebo or an ARB (valsartan or candesartan) added to existing treatment with an ACE inhibitor (some patients were also already on beta-blockers and/or spironolactone). Each of these trials showed that ARB treatment reduced the risk of hospital admission for worsening HF (RRR 24% in Val-HeFT and 17% in CHARM-Added). There was a 16% RRR in the risk of death from a cardiovascular cause with candesartan in CHARM-Added. These benefits were additional to those gained with conventional treatment, including a diuretic, digoxin, ACE inhibitor and a beta-blocker.

Key Point:
Patients with HF due to LVSD, who are still symptomatic despite therapy with an ACE inhibitor and a beta blocker, may benefit from the addition of an ARB, following specialist advice.

Beta-blockers in HF
For many years, HF was considered to be a contraindication to the use of beta-blockers. But beta-blockers are now known to have an important role in event prevention.\(^\text{44,45}\)

Many RCTs have been undertaken with beta blockers in patients with HF. In the CIBIS II,\(^\text{46}\) MERIT-HF,\(^\text{47}\) and COPERNICUS\(^\text{48}\) a consistent, approximately one third reduction in total mortality was seen with bisoprolol, extended release metoprolol succinate and carvedilol. In the SENIORS trial, nebivolol significantly reduced a composite outcome of death or cardiovascular hospitalisations in elderly HF patients.\(^\text{49}\) Beta-blockers increase life expectancy in patients with HF due to LVSD compared with placebo; an effect seen in all functional classes of HF.\(^\text{50}\)

Unless contraindicated or not tolerated, a beta-blocker should be used in all patients with symptomatic HF and LVSD. This recommendation is consistent with guidelines from NICE, SIGN, ESC and CREST.\(^\text{1,2,17,21}\) There is concern that prescribers are reluctant to prescribe beta-blockers, particularly for the elderly, because of the history of adverse effects.\(^\text{51}\) However, NICE indicate that beta-blockers (licensed for HF) can be offered to all patients with HF due to LV systolic dysfunction including older patients, patients with PVD, erectile dysfunction, diabetes mellitus, interstitial pulmonary disease, and COPD without reversibility.\(^\text{17}\)

Which beta-blocker?
Bisoprolol, carvedilol and nebivolol (patients >70years only) are licensed in HF management. All are available generically.\(^\text{32}\)

One issue with beta-blocker therapy is whether there is any substantial difference between cardioselective and non-selective drugs. As far as HF patients are concerned, the best evidence comes from the COMET study suggesting that the non-selective agent carvedilol was associated with better outcomes than the selective agent metoprolol.\(^\text{52}\)

NICE recommend that stable patients who are already taking a beta-blocker for co-morbidity (e.g. HTN, angina), and who develop HF due to LVSD, should be switched to a beta-blocker licensed for HF.\(^\text{17}\)

Starting a beta-blocker in HF
Beta-blockers produce benefit in the medium to long term, although a temporary deterioration (worsening of HF symptoms) is possible initially. Beta-blockers are associated with hypotension, dizziness and bradycardia. Fatigue can also be a problem initially but this usually improves with time.

The chosen beta-blocker should be introduced in a “start low, go slow” manner, and assess heart rate, blood pressure and clinical status after each titration.\(^\text{17}\) Up-titration can be attempted every 2-4 weeks (slower dose up-titration may be needed in some patients). Do not increase the dose if signs of worsening HF, symptomatic hypotension (e.g. dizziness), or excessive bradycardia (pulse rate ≤50bpm). In the absence of these problems, double the dose of beta-blocker every 2-4 weeks until the evidence-based target dose is reached (see Table SEVEN) or maximum tolerated dose.

| Table SIX: ACE inhibitor doses in heart failure\(^\text{32}\) |
|-----------------|-----------------|
| **Starting dose** | **Target/Maximum HF dose** |
| Captopril 6.25mg-12.5mg two-three times daily | Maximum 150mg daily in divided doses |
| Cilazapril 500 micrograms once daily | 1-2.5mg once daily. Maximum 5mg daily |
| Enalapril 2.5mg once daily | 10-20mg twice daily |
| Fosinopril 10mg once daily | 40mg once daily |
| Lisinopril 2.5mg once daily | 35mg once daily |
| Perindopril erbumine 2mg once daily | 4mg once daily |
| Perindopril arginine 2.5mg once daily | 5mg once daily |
| Quinapril 2.5mg once daily | 10-20mg in one to two divided doses. Maximum 40mg daily |
| Ramipril 1.25mg once daily | 5mg twice daily |
| Trandolapril 500micrograms once daily | 4mg once daily |

Table SEVEN: Beta-blocker doses in heart failure

Starting dose | Target dose (if possible) |
--------------|--------------------------|
Bisoprolol 1.25mg-2.5mg once daily | 2.5mg once daily |
Carvedilol 6.25mg-12.5mg once daily | 25mg once daily |
Nebivolol 1.25mg-2.5mg once daily | 2.5mg once daily |
Metoprolol | Maximum 150mg daily |
Labetalol 50-200mg twice daily | Maximum 400mg daily |
Araldil 100-400mg twice daily | Maximum 1200mg daily |
Propranolol 40-240mg twice daily | Maximum 1200mg daily |
Timolol 50-300mg twice daily | Maximum 1200mg daily |
Other beta-blockers | Maximum 150mg daily |

COMPASS Therapeutic Notes on the Management of Heart Failure in Primary Care • July 2011
### Table SEVEN: Doses in HF for licensed beta-blockers⁴²

<table>
<thead>
<tr>
<th></th>
<th>Starting Dose</th>
<th>Target dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bisoprolol</td>
<td>1.25mg once daily</td>
<td>10mg once daily</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>2.5mg twice daily</td>
<td>25-50mg twice daily</td>
</tr>
<tr>
<td>Nebivolol</td>
<td>1.25mg once daily</td>
<td>10mg once daily</td>
</tr>
</tbody>
</table>

*Note: The doses may vary depending on the individual patient's response and medical history.*

### Diuretics in HF

The recommendation to give diuretics to relieve symptoms of HF is consistent with guidelines from NICE, SIGN and ESC.¹,¹⁷,²¹ There is little robust evidence that diuretics reduce mortality compared with placebo but their role in symptom relief in HF is undisputed.⁵³ Diuretics should be routinely used to treat fluid overload, either pulmonary or peripheral oedema and titrated (up or down) according to need following initiation of subsequent HF therapies.⁷ Loop diuretics are typically employed and more effective than thiazides in promoting diuresis and natriuresis.

**Loop diuretics (furosemide, bumetanide, torsemide)**

Care should be taken to select the dose of the loop diuretic, i.e. the dose should eliminate ankle or pulmonary oedema without dehydrating the patient and placing them at risk of renal dysfunction or hypotension. The correct dose to achieve this varies markedly from one patient to the next. The tendency of loop diuretics to cause hypokalaemia is offset by ACE inhibitors, ARBs and spironolactone. Serum potassium should be monitored to maintain its concentration in the range 4-5 mmol/L, and adjustments in therapy should be made to prevent both hypo- and hyperkalaemia.²¹

#### Prescribing point: Beta-blockers

- Offer a beta-blocker (licensed for HF) to all patients with HF due to LVSD, including older patients and patients with peripheral vascular disease, erectile dysfunction, diabetes, interstitial pulmonary disease and COPD without reversibility.

### Table EIGHT: Diuretic doses in patients with HF as recommended by ESC¹ (these may differ from those quoted in BNF)

<table>
<thead>
<tr>
<th>Diuretic</th>
<th>Initial dose (mg)</th>
<th>Usual daily dose (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Loop diuretics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Furosemide</td>
<td>20-40</td>
<td>40-240</td>
</tr>
<tr>
<td>Bumetanide</td>
<td>0.5-1</td>
<td>1-5</td>
</tr>
<tr>
<td>Torsemide</td>
<td>5-10</td>
<td>10-20</td>
</tr>
<tr>
<td><strong>Thiazides</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bendroflumethiazide</td>
<td>2.5</td>
<td>2.5-10</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>25</td>
<td>12.5-100</td>
</tr>
<tr>
<td>Metolazone</td>
<td>2.5</td>
<td>2.5-10</td>
</tr>
<tr>
<td>Indapamide</td>
<td>2.5</td>
<td>2.5-5</td>
</tr>
<tr>
<td><strong>Potassium-sparing diuretics</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Spironolactone/eplerenone</td>
<td>12.5-25</td>
<td>50-100</td>
</tr>
<tr>
<td>Amlodipine</td>
<td>2.5</td>
<td>5-20</td>
</tr>
<tr>
<td>Triamterene</td>
<td>25</td>
<td>50-100</td>
</tr>
</tbody>
</table>

*Unless contraindicated or not tolerated, the addition of an aldosterone antagonist should be considered in all patients with an LVEF ≤ 35% and severe symptomatic HF, i.e. currently NYHA class III or IV, in the absence of hyperkalaemia and significant renal dysfunction (serum potassium ≥ 5.5 mmol/L).*

### Key Points:

- The dose of diuretic should be individualised to reduce fluid retention without over-treating which may produce dehydration or renal dysfunction.
- If the dose of diuretic needs to be increased in the acute setting, remember to review the dose later with a view to reducing it again.

### How should diuretic therapy be initiated?

Check renal function and serum electrolytes. Start with a low dosage and increase until clinical improvement of the symptoms and signs of congestion (See Table EIGHT). Dose must be adjusted, particularly after restoration of dry body weight, to avoid the risk of renal dysfunction and dehydration. Aim to maintain “dry weight” with lowest achievable dose.

**Where oedema is resistant to a loop diuretic…**

In cases where oedema is resistant to the loop diuretic, a number of strategies are available:²¹

- In patients with severe HF, high dose furosemide administered as a continuous infusion was more efficacious than bolus injection.²⁴
- Sequential nephron blockade with thiazides and loop diuretics may also be effective. The careful addition of metolazone (starting dose 2.5 mg/day) can often cause a useful natriuresis, although careful monitoring is essential to prevent abnormalities in sodium, creatinine and other electrolytes.
- Addition of 25-100 mg of hydrochlorothiazide also proved to be very effective in patients with severe HF and impaired renal function showing diuretic resistance to a daily dose of furosemide of at least 250 mg.⁵⁵
- Bendroflumethiazide 10 mg and metolazone 10 mg have been shown to be equally effective in establishing a diuresis when combined with loop diuretics.³⁶

**Aldosterone antagonists in HF (spironolactone and eplerenone▼)**

Unless contraindicated or not tolerated, the addition of an aldosterone antagonist should be considered in all patients with an LVEF ≤ 35% and severe symptomatic HF, i.e. currently NYHA class III or IV, in the absence of hyperkalaemia and significant renal dysfunction (serum potassium ≥ 5.5 mmol/L).
creatine > 220 micromol/L). If either spironolactone or eplerenone is used, monitoring of serum electrolytes and renal function is mandatory. Specialist supervision is recommended.

There is no evidence that aldosterone antagonists are as effective in mild HF and no recommendation can be given for this group of patients.

A single large RCT (RALES) was undertaken with spironolactone in patients with severe HF. Treatment with spironolactone led to an RRR in death of 30% and an RRR in hospital admission for worsening HF of 35% within an average of 2 years of starting treatment. Spironolactone also improved NYHA-class. These benefits were additional to those gained with conventional treatment. The ARR in mortality (after a mean of 2 years treatment) in patients with severe HF was 11.4% equating to an NNT=9 (for 2 years to postpone 1 death).

### Hypokalaemia/Hypomagnesaemia

- Increase ACE inhibitor/ARB dose
- Add aldosterone antagonist
- Potassium supplements
- Magnesium supplements

### Hyponatraemia

- Fluid restriction
- Stop thiazide diuretic or switch to loop diuretic
- Reduce dose/stop loop diuretic

### Hyperuricaemia/Gout

- Consider allopurinol
- For symptomatic gout use colchicine for pain relief

### Hypovolaemia/Dehydration

- Assess volume status
- Consider diuretic dosage reduction

### Insufficient Response/Diuretic Resistance

- Check compliance and fluid intake
- Increase diuretic dose
- Consider switching from furosemide to bumetanide or torasemide
- Add an aldosterone antagonist
- Combine loop diuretic and thiazide
- Administer loop diuretic twice daily or on an empty stomach
- Consider short-term IV infusion of loop diuretic

### Renal Failure

- Check for hypovolaemia/dehydration
- Exclude use of other nephrotoxic drugs
- Withhold aldosterone antagonist
- If using loop and thiazide diuretics, stop thiazide
- Consider reducing dose of ACE inhibitor/LVAs

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**Table: Practical considerations in treatment of heart failure with loop diuretics**

<table>
<thead>
<tr>
<th>Problem</th>
<th>Suggested action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypokalaemia/Hypomagnesaemia</td>
<td>• Increase ACE inhibitor/ARB dose</td>
</tr>
<tr>
<td></td>
<td>• Add aldosterone antagonist</td>
</tr>
<tr>
<td></td>
<td>• Potassium supplements</td>
</tr>
<tr>
<td></td>
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<tr>
<td></td>
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<tr>
<td></td>
<td>• Reduce dose/stop loop diuretic</td>
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<td></td>
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</tr>
<tr>
<td>Renal failure</td>
<td>• Check for hypovolaemia/dehydration</td>
</tr>
<tr>
<td></td>
<td>• Exclude use of other nephrotoxic drugs</td>
</tr>
<tr>
<td></td>
<td>• Withhold aldosterone antagonist</td>
</tr>
<tr>
<td></td>
<td>• If using loop and thiazide diuretics, stop thiazide</td>
</tr>
<tr>
<td></td>
<td>• Consider reducing dose of ACE inhibitor/ARB</td>
</tr>
</tbody>
</table>

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**Eplerenone ▼ (Inspra ®)**

Eplerenone ▼ is reputed to have fewer side-effects than spironolactone. Spironolactone can cause breast discomfort and enlargement in men (10% compared with placebo, in RALES); this side effect is infrequent with eplerenone ▼. Outside the post-infarction indication, the main indication for eplerenone ▼ is in men with breast discomfort and/or enlargement caused by spironolactone. Although eplerenone ▼ produces less gynaecomastia than spironolactone, it can still produce hyperkalaemia and renal dysfunction and blood urea. Creatinine and potassium should be carefully monitored after initiation and throughout therapy.

**How to use spironolactone (or eplerenone ▼) in HF**

Prior to initiation, check renal function and serum electrolytes. Start spironolactone (or eplerenone ▼) at 25mg daily. Re-check renal function and serum electrolytes 1 and 4 weeks after starting treatment. Consider up-titration after 4-8 weeks. Do not increase dose if worsening renal function or hyperkalaemia. Re-check renal function and serum electrolytes 1 and 4 weeks after increasing dose. In the absence of any problems, aim for the evidence-based target dose (spironolactone 50mg daily, eplerenone ▼ 50mg daily) or the maximum tolerated dose. Re-check renal function and serum electrolytes 1, 2, 3 and 6 months after achieving maintenance dose and 6 monthly thereafter.

**Hydralazine / Isosorbide dinitrate (H-ISDN) combination in HF**

The combination of hydralazine and isosorbide dinitrate (H-ISDN) was used before the introduction of ACE inhibitors. H-ISDN are known to reduce mortality in patients with HF, but not by as much as ACE inhibitors.

**In which patients should the H-ISDN combination be considered?**

The H-ISDN combination is rarely used now but may have a role in the following two groups:

1. **Patients of Afro-Caribbean origin with HF of NYHA class III or IV** – In this group H-ISDN has been shown to reduce symptoms and the risk of death and hospital admissions for HF when added to standard treatment (ACE inhibitor +/- ARB, beta-blocker, digoxin, spironolactone, and diuretic) (absolute survival benefit 4.0%, hazard ratio for all cause mortality 0.57, p=0.01).62
2. **As an alternative first-line treatment (under specialist advice) for patients who are intolerant of an ACE inhibitor and ARB due to renal dysfunction or hyperkalaemia**, 1,2,17

**Adverse effects with H-ISDN**

The most common adverse effects with H-ISDN in trials were headache, dizziness/hypotension, and nausea.

**Digoxin**

A Cochrane review has shown a 64% improvement in symptoms (OR=0.31, 95% CI 0.21 to 0.43; ARR 11.5%; NNT=9) and a 23% reduction in hospitalisation (OR=0.68, 95% CI 0.61 to 0.75; ARR 5.7%; NNT=18) for patients with HF receiving digoxin. Digoxin did not improve survival. This review is dominated by one large trial (the DIG study) which was carried out before the introduction of beta-blockers.
blockers and spironolactone for HF, which may have influenced the conclusions. Evidence of benefit must be weighted against the possibility of an increase in sudden deaths associated with digoxin. The risk of digoxin toxicity is increased by hypokalaemia.

**What is the role of digoxin in patients with HF?**
Digoxin increases the force of myocardial contraction and reduces conductivity within the AV node. Digoxin has a (limited) role in HF management now in:
1. Patients who are in sinus rhythm, with worsening or severe HF due to LVSD despite first- and second-line treatments. In these patients, addition of digoxin improves ventricular function and patient well-being, reduces hospital admission for worsening HF, but has no effect on survival.
2. Patients with symptomatic HF and atrial fibrillation (AF).

Digoxin may be used to slow a rapid ventricular rate. The usual daily dose of oral digoxin is 125 to 250 micrograms if the serum creatinine is within the normal range. Higher doses (> 250 micrograms) are rarely used for HF. Lower doses are used if the patient is elderly (over age 70 years) or has impaired renal function. There is little relationship between digoxin concentration and therapeutic effects.

**Other drugs used in the management of HF**

In general, are anticoagulants necessary in patients with HF?
Anticoagulants are not routinely used in HF patients. However, warfarin treatment will be necessary in people with:
- HF and AF,
- HF who are in sinus rhythm and have a history of thromboembolism, left ventricular aneurysm, or intracardiac thrombus.

Do patients with HF need to be on aspirin?
Aspirin (75-150mg once daily) should be prescribed for patients with the combination of HF and atherosclerotic arterial disease (including coronary heart disease). In HF patients without atherosclerotic disease there is no firm evidence for the use of antplatelet agents.

Is there a role for amiodarone in HF?
The role of amiodarone in the treatment of HF is controversial. The decision to prescribe amiodarone should be made in consultation with a specialist. The need to continue the amiodarone prescription should be reviewed regularly. Patients taking amiodarone should have routine 6-monthly clinical review, including liver and thyroid function test, and including a review of side effects.

**Calcium channel blockers**
Calcium channel blockers (with the exception of amlodipine) have been found to exacerbate symptoms of HF or increase mortality after myocardial infarction in people who also have pulmonary congestion or left ventricular dysfunction. Therefore, in general, CCBs should be avoided in patients with HF. The exception to this is amlodipine, which can be considered for the treatment of co-morbid HTN and/or angina in patients with HF.

Is there any benefit from starting a statin in a patient with HF?
A statin is certainly indicated if the person has atherosclerotic arterial disease or has a 10-year risk of a cardiovascular event which is 20% or more. Studies of statin use in patients with symptomatic HF and ischaemic heart disease have shown that while statins have no effect on mortality in HF, they do decrease the risk of hospitalisation for worsening HF. The value of statins in HF patients with a non-ischaemic aetiology is unknown.

**Key messages on drug management of HF:**
- ACE inhibitors and beta-blockers are first-line
- Aim for target doses, but some is better than none
- There is no evidence that ARBs are better than ACE inhibitors – reserve for ACE inhibitor intolerance
- Use a licensed beta-blocker
- DON'T FORGET TO MONITOR (particularly renal function and electrolytes)
- Use a diuretic

**Diastolic Heart Failure**

**What is diastolic HF?**
A distinction is frequently made between systolic and diastolic HF. The distinction has arisen largely because in the past most patients admitted to hospitals for investigation or entered into clinical trials have had dilated hearts with a reduced EF <35-40%. However, not all patients with HF have left ventricular systolic dysfunction. Patients with symptoms and/or signs of HF and a preserved left ventricular ejection fraction > 40-50%, have been described as having “diastolic” HF. Other phrases have been used to describe diastolic HF, including:
- HF with preserved ejection fraction (HFPEF)
- HF with normal ejection fraction (HFNEF)
- HF with preserved systolic function (HFPFS)

The proportion of HF patients with preserved LV systolic function may be as high as 35-50%.

Is the management of patients with diastolic HF different?
Although the general approach to care is the same whether systolic function is reduced or not, most studies have been undertaken in patients with HF and LVSD. Consequently treatment of diastolic HF is largely empiric due to lack of an evidence base. Diastolic HF often occurs along with myocardial ischaemia, hypertension, myocardial hypertrophy or even myocardial/pericardial constriction. Consideration should be given as to whether these entities may be present and contribute to the clinical picture in patients with diastolic HF. If present, they should be identified and treated in their own right. An additional contributory factor could be tachyarrhythmias; if so, rate control is likely to be beneficial. No treatment has yet been shown, convincingly, to reduce morbidity and mortality in these patients. However, in practice the following are often used although the evidence base is not robust enough to definitively recommend any of these treatments:
- Diuretics are often used to reduce and then prevent fluid overload
- ACE inhibitors (Perindopril has been shown to reduce cardiovascular death and HF hospitalisation)
- ARBs - Candesartan may reduce the risk of hospitalisation for worsening HF (shown in the CHARM Preserved study)
- Rate-limiting calcium channel blockers - Two very small studies have shown that verapamil may improve exercise capacity and symptoms in these patients
- Beta blockers.

COMPASS Therapeutic Notes on the Management of Heart Failure in Primary Care ● July 2011
Infections, particularly pulmonary infections, cause worsening HF and hospital admissions.77,78 Immunisation against ‘flu has been shown to reduce hospital admission for worsening HF during a ‘flu outbreak.79 The Joint Committee on Vaccination and Immunisations recommends immunisation for those with chronic conditions (which would include HF), with influenza and pneumococcal vaccine.80 Pneumococcal vaccine is required to be given annually but pneumococcal vaccine is once only.

Where fatigue, oedema, weight gain and dyspnoea do not rapidly improve.

Where there are concerns about low blood pressure. Where fluid retention is resistant.

Seek specialist advice if serious deterioration (fatigue, oedema, weight gain and dyspnoea) does not improve.

Where there is extreme fatigue (or bradycardia <50bpm) consider reducing dose of beta-blockers.

When commencing an ACE inhibitor in patients taking large doses of diuretics. Where there is extreme fatigue (or bradycardia <50bpm) consider reducing the dose of beta-blocker.

Explain the purpose of the medication prescribed and the importance of up-titration to optimal dose. Explain the need for regular monitoring and at times alteration of medication. Explain that improvement with an ACE inhibitor or beta-blocker may take time to accrue. Explain that minor worsening of symptoms may occur when beta-blockers are being initiated.

Encourage individuals to monitor their weight and to report any change.

Renal function:
Monitor renal function in all patients routinely. Check renal function before starting an ACE inhibitor or ARB, and monitor the urea, creatinine, eGFR and electrolytes following each dose increment, and then at regular intervals every three months. Monitor more frequently patients taking combined loop and thiazide diuretic therapy, and in those taking aldosterone antagonists.

Blood pressure:
Monitor BP in all patients routinely. If blood pressure is low, first consider discontinuing nitrates, calcium channel blockers and other vasodilators. If blood pressure is low, reduce diuretics in patients who do not have signs of congestion. In asymptomatic hypotension do not alter the dose of ACE inhibitor or beta-blocker.

Where at all possible maintain treatment with both an ACE inhibitor and a beta-blocker, at reduced dose if necessary.

Increasing congestion/fatigue:
If temporary deterioration occurs during the initiation or up-titration of beta-blockers diuretic dose may need to be briefly increased. If congestion occurs increase diuretics and consider reducing dose of beta-blocker (but not discontinuing). Where there is extreme fatigue (or bradycardia <50bpm) consider reducing the dose of beta-blocker.

Seek specialist advice if serious deterioration (fatigue, oedema, weight gain and dyspnoea) does not improve.

Consider specialist review:
Where fluid retention is resistant. When commencing an ACE inhibitor in patients taking large doses of diuretics.

Where renal function continues to deteriorate or deteriorated rapidly. Where there are concerns about low blood pressure. Where fatigue, oedema, weight gain and dyspnoea do not rapidly improve.

Websites
- The British Heart Foundation - www.bhf.org.uk
- Heart Failure Matters - www.heartfailurematters.org
- National Heart Forum – www.heartforum.org.uk

Practical notes in the pharmacological management of patients with HF76

General advice:
For optimal prognostic and symptomatic benefit doses of ACE inhibitor and beta-blocker should be up-titrated to the maximum tolerated. This may require repeated or prolonged supervision in some patients. The dose of diuretic should be the minimum necessary to control oedema.

Communication with patients:
Explain the purpose of the medication prescribed and the importance of up-titration to optimal dose. Explain the need for regular monitoring and at times alteration of medication. Explain that improvement with an ACE inhibitor or beta-blocker may take time to accrue. Explain that minor worsening of symptoms may occur when beta-blockers are being initiated.

Encourage individuals to monitor their weight and to report any change.

Reference List
4. Raw prevalence data per 1000 patients at practice level. QoF Outcomes and Prevalence 2009/2010 2010;
20. Almeida, O. P. and Flicker, L. The mind of a failing heart: a systematic review of the association between congestive...
32. BMA/RPSGB. British National Formulary. BNF61 2011;
Ref Type: Internet Communication

COMPASS Therapeutic Notes on the Management of Heart Failure in Primary Care ● July 2011
COMPASS Therapeutic Notes are circulated to GPs, nurses, pharmacists and others in Northern Ireland. Each issue is compiled following the review of approximately 250 papers, journal articles, guidelines and standards documents. They are written in question and answer format, with summary points and recommendations on each topic. They reflect local, national and international guidelines and standards on current best clinical practice. Each issue is reviewed and updated every three years.

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- Nurse? □ Enter your PIN number: ___________

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**Surname:** ___________________ **First name:** ___________________

**Address:**

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Management of Heart Failure in Primary Care

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• Doctors and nurses should submit their answers at: www.medicinesni.com
• Pharmacists should submit their answers at: www.nicpld.org

1 Heart failure:
   a Has a prevalence of about 7 in 1000 people in Northern Ireland T F
   b Is more common in women T F
   c Is commonly caused by coronary heart disease T F
   d Has survival rates worse than for most cancers T F

2 Using ACE inhibitors in the management of heart failure:
   a ACE inhibitors should be considered in patients with all classes of HF due to left ventricular systolic dysfunction T F
   b Seek specialist advice before commencing an ACE inhibitor in a patient already taking more than 80mg of furosemide T F
   c Any rise in urea and creatinine after initiation of an ACE inhibitor warrants discontinuation of the ACE inhibitor T F
   d Pregnancy is a contraindication to the use of an ACE inhibitor T F

3 Using beta-blockers in the management of heart failure:
   a Unless contraindicated or not tolerated, a beta-blocker should be used in all patients with symptomatic HF and LVSD T F
   b Nebivolol is only licensed for patients with heart failure who are over the age of 70 years T F
   c Beta-blockers can initially cause the symptoms of heart failure to worsen T F
   d Chronic obstructive pulmonary disease is a contraindication to the use of beta-blockers T F

4 In the pharmacological management of heart failure:
   a ACE inhibitors improve survival for all grades of heart failure T F
   b Diuretics improve quality of life but not survival T F
   c Beta-blockers do not improve survival T F
   d Aldosterone antagonists improve quality of life in mild heart failure T F

5 Which of the following are part of routine management of patients with heart failure?
   a Low-dose aspirin T F
   b A statin T F
   c Digoxin T F
   d An anticoagulant T F