Identifying patients with HEART FAILURE in primary care
Heart failure is now described as either heart failure with reduced ejection fraction (HF-REF) or heart failure with preserved ejection fraction (HF-PEF). Identifying people with suspected heart failure in primary care is often based on the patient’s presenting symptoms and signs. Ideally, a formal diagnosis should always be made with echocardiography, however, referring every patient with suspected heart failure is likely to be impractical, given resource limitations. Other investigations such as BNP and ECG can assist in making a diagnosis.

**Getting to the heart of the matter**

In developed countries the prevalence of heart failure among adults is approximately 1–2%, although the prevalence may be more than 10% among older adults (≥ 70 years). A typical primary care clinician, caring for 2000 patients, is therefore likely to have approximately 40 patients with heart failure, and more if their patient population is older. In addition, approximately five patients will be newly diagnosed with heart failure each year and the condition suspected in three times as many patients as this. The number of people with heart failure will inevitably increase in New Zealand in the future. This is attributed to people living longer, more effective treatments for coronary heart disease and a reduction in mortality from acute coronary events. An increase in prevalence of cardiovascular risk factors such as diabetes and obesity is also contributing to the increasing prevalence of heart failure.

People with heart failure often have a reduction in quality of life, require frequent hospital admissions and have a poor prognosis. Although there have been improvements in the clinical outcomes for patients over the last two decades, such as fewer admissions, a reduced length of hospital stay and longer survival out of hospital, mortality from heart failure remains high. In the first year after diagnosis, 30 – 40% of patients die, however, this decreases to 10% per year thereafter.

Mortality rates are highest for patients who have heart failure associated with an acute myocardial infarction or arrhythmia, those who are hypotensive (i.e. in cardiogenic shock), those with more severe symptoms (NYHA Class IV, Page 11) and patients who have repeated hospitalisations for heart failure.

There is some evidence that heart failure is likely to affect males more than females, although females with heart failure are more likely to:

- Be older than males when they develop heart failure
- Have heart failure with a preserved ejection fraction (Page 7)
- Live longer than males with heart failure
- Have more pronounced symptoms of heart failure than males

**Defining heart failure**

Heart failure can be defined as an abnormality of the structure or function of the heart that leads to a failure of the heart to deliver sufficient oxygen to the metabolising tissues (or when the heart can only do so with elevated diastolic filling pressures). Compensatory mechanisms, e.g. an increase in heart rate, cardiac muscle mass, cardiac filling pressures and blood volume, work to maintain the ability of the heart to pump effectively, however, over time the heart progressively fails.

Heart failure can be regarded as a complex clinical syndrome with typical symptoms and signs that develops as a result of a large number of diverse cardiac and non-cardiac abnormalities. There are multiple risk factors for heart failure, which include:

- Coronary heart disease – ischaemic heart disease (IHD) leading primarily to left ventricular dysfunction; the most significant risk factor for heart failure in developed countries
Hypertension – 75% of patients with heart failure have a history of hypertension
Valvular heart disease, including valvular damage from rheumatic fever
Abnormalities of rhythm (e.g. atrial fibrillation) or conduction (e.g. left bundle branch block)
Cardiomyopathy, e.g. idiopathic, viral, alcoholic, toxic, peripartum
Diabetes – independent risk factor for coronary heart disease but also increases the risk of heart failure directly
Male gender
Excessive alcohol use – increasing cardiovascular risk and as a direct cardiotoxin
Smoking – increasing cardiovascular risk and as a direct cardiotoxin
Obesity – increasing cardiovascular risk and an independent risk factor
Dyslipidaemia
Respiratory conditions, e.g. COPD, obstructive sleep apnoea
Thyroid disorders – both hypo- and hyperthyroidism
Medicines e.g. NSAIDs, pioglitazone (see Box below)
Cardiotoxins e.g. chemotherapy medicines, cocaine
Infections or inflammation – leading to myocarditis or cardiomyopathy
Congenital heart disease

Ethnic disparities in morbidity and mortality from heart failure in New Zealand

The mortality rate from heart failure for Māori aged over 65 years in New Zealand is significantly higher than for non-Māori for both males and females (RR 2.80 for males; RR 1.70 for females). Rates of hospitalisation for heart failure amongst Māori in this age group are also significantly higher than for non-Māori (RR 4.73 for males; RR 4.85 for females). It is reported that the differences between mortality rates from heart failure between Māori and non-Māori are even more pronounced in younger age groups (45 – 65 years), and heart failure occurs approximately 10 – 15 years earlier in Māori compared to non-Māori. Māori are significantly younger on admission to hospital for heart failure than New Zealand Europeans (age 62 compared to age 78 years). Morbidity and mortality from heart failure for Pacific peoples is approximately twice as high compared to the total population.

Medicines that can worsen heart failure

Medicines that may worsen heart failure in symptomatic patients include:

- NSAIDs including COX-2 inhibitors because they may cause renal impairment and sodium and water retention
- Pioglitazone due to dose related fluid retention
- Most calcium channel blockers in patients with HF-REF (with the exception of amlodipine and felodipine) due to their negative inotropic effect
- The combination of an ACE inhibitor AND an angiotensin-II receptor blocker (ARB) AND a mineralocorticoid antagonist (e.g. spironolactone) because this combination can worsen renal function which in turn may render loop diuretics ineffective and cause hypokalaemia or hyperkalaemia
Heart failure with reduced or preserved ejection fraction

Heart failure has, in the past, been predominately thought of in terms of systolic dysfunction of the left ventricle, i.e. a reduction in left ventricular ejection fraction. Clinical trials, however, have found that left ventricular failure can develop in patients who have an essentially normal ejection fraction and that this is a separate clinical syndrome. This has led to the adoption of the terms heart failure with reduced ejection fraction (HF-REF, previously referred to as systolic heart failure) and heart failure with preserved ejection fraction (HF-PEF, previously referred to as diastolic heart failure). Although, the terms systolic and diastolic heart failure remain in use in the literature there is now a preference for the terms HF-REF and HF-PEF. Up to 50% of people with symptoms and signs of heart failure have been shown to have preserved or relatively preserved (≥45 – 50%) left ventricular ejection fraction. The underlying causative conditions and the treatment of each type of heart failure differ.

Heart failure with reduced ejection fraction (HF-REF) – i.e. impaired left ventricular systolic function, is typically seen in patients with structural heart disease, e.g. ischaemic heart disease. Many other causative factors may contribute to HF-REF, including hypertension, diabetes and idiopathic dilated cardiomyopathy.

The majority of research into effective, evidence-based treatment strategies for heart failure has been based on patients who have HF-REF.

Heart failure with preserved ejection fraction (HF-PEF) – i.e. impaired diastolic function (such as impaired diastolic relaxation and filling due to stiffening of the ventricles) is more frequently seen in older people, females and people who are obese. People with HF-PEF are also more likely to have atrial fibrillation and to be more hypertensive. In addition, other underlying causes include constrictive pericarditis or cardiomyopathy, hypertrophic cardiomyopathy and restrictive cardiomyopathy, e.g. from amyloidosis or sarcoidosis.

Making a diagnosis

Identifying people with suspected heart failure in primary care is often based on the patient’s presenting symptoms and signs. Ideally a formal diagnosis should always be made with echocardiography, however, availability of services may be a barrier in some areas.

Diagnosis of heart failure (adapted from ESC)

HF-REF – To make a diagnosis of HF-REF the patient must have:
1. Symptoms typical of heart failure
2. Signs typical of heart failure
3. Evidence of reduced left ventricular ejection fraction on echocardiography

HF-PEF – To make a diagnosis of HF-PEF the patient must have:
1. Symptoms typical of heart failure
2. Signs typical of heart failure
3. Normal or only mildly reduced left ventricular ejection fraction and no left ventricular dilatation
4. Relevant structural heart disease such as left ventricular hypertrophy or left atrial enlargement and/or diastolic dysfunction

Making a diagnosis of HF-PEF is essentially one of exclusion after other potential non-cardiac causes for the patient’s symptoms, e.g. anaemia, respiratory disease, are ruled out.

Symptoms and signs of heart failure

Patients with heart failure present with a variable combination of symptoms and signs (Table 1, over page), each of which influence the level of clinical suspicion and help guide the investigations and initial management. Patients may present (either as a first presentation or with a known history of heart failure) with a sudden onset of acute symptoms consistent with heart failure or have symptoms that have developed more gradually.

Symptoms typically include dyspnoea, orthopnoea, fatigue, reduced exercise tolerance and ankle oedema, however, many of the symptoms are not necessarily specific to heart failure and vary with the acuteness of the condition. The significance of symptoms and signs may be more difficult to interpret in people who are older, obese or those with co-morbidities such as chronic obstructive pulmonary disease (COPD).

Peripheral oedema is a non-specific symptom that may be seen frequently but can be due to other reasons such as varicose veins, medicines (e.g. calcium channel blockers) or decreased mobility. Crepitations in the chest on auscultation...
are also a non-specific sign and may confound the presentation, especially if the patient has an existing respiratory condition. The presence of orthopnoea and paroxysmal nocturnal dyspnoea are more specific symptoms of heart failure but are less often reported at presentation and tend to be associated with more severe heart failure.¹ Clinical signs that are more specific to heart failure, e.g. increased jugular venous pressure and displacement of the apex beat, can be difficult signs to detect accurately.¹ These factors, therefore, can make a clinical diagnosis of heart failure difficult, particularly in a primary care setting.

**Echocardiography is the gold standard investigation**

Depending on the findings from history and examination, a number of investigations may be required in a patient with suspected heart failure (Table 2). Some tests may support or rule out a clinical suspicion, other investigations are required for identification of a reversible cause, e.g. thyroid dysfunction, and for ongoing monitoring of the patient’s progress and medicine use.

Ideally all patients with suspected heart failure require echocardiography for an accurate diagnosis to be made. Referring every patient with suspected heart failure, however, is likely to be impractical, given resource limitations. To help identify which symptomatic patients require echocardiography, use the results from an ECG and brain natriuretic peptide (BNP) test – patients with either an abnormal ECG or an increased BNP level are likely to have heart failure and require referral for echocardiography.¹ Guidance from NICE suggests that the timeframe required for further assessment with echocardiogram is within two weeks if the patient has a history of a previous myocardial infarction (MI) and an elevated BNP, and within six weeks if there is no history of MI and a moderately raised BNP.²

**The role of BNP in the diagnosis of heart failure**

Laboratory assessment of BNP or N-terminal pro-BNP (NT-proBNP), depending on the laboratory, may provide additional information for some patients, where the diagnosis is uncertain. If BNP is normal (<30 pmol/L), a diagnosis of heart failure is unlikely.²⁰ The cut-off values in patients presenting with a more gradual onset of symptoms may be lower.¹ Laboratories around New Zealand differ in their recommendations for cut-off values for using BNP or NT ProBNP for diagnosing heart failure, therefore it is best to check with your local laboratory or discuss the results with a clinical biochemist. Table 3, over page, gives an example of cut-off values.
Table 2: Investigations for diagnosis and monitoring of patients with heart failure

<table>
<thead>
<tr>
<th>Investigation</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Echocardiography and Doppler</strong></td>
<td>The gold standard test for heart failure that ideally should be used in every patient with suspected heart failure. Ventricular and valvular function can be assessed. Echocardiography can confirm a diagnosis of heart failure, can distinguish between HF-REF and HF-PEF and guide management options. An echocardiogram is also often useful if the patient has unexplained symptoms of shortness of breath or fatigue. Echocardiography is also used to follow a patient’s progress once treatment is initiated. It can provide a sequential assessment of the response to treatment, of ejection fractions, left atrial remodelling and filling pressures. Echocardiography can also be used to assess for dyssynchrony for cardiac resynchronisation therapy (CRT).</td>
</tr>
<tr>
<td><strong>Brain natriuretic peptide (BNP) or NT-proBNP</strong></td>
<td>BNP is most useful in ruling out heart failure in a patient with an atypical presentation or a patient with respiratory co-morbidities. BNP is released from the cardiac ventricles in response to increases in ventricular volume and pressures. BNP is regarded as a good “rule out test” for heart failure, but should be interpreted in view of the clinical features of the patient. BNP cannot be used to differentiate between HF-REF and HF-PEF. BNP can also be useful for monitoring treatment and has prognostic implications – high levels are associated with a poorer prognosis.</td>
</tr>
<tr>
<td><strong>Electrocardiography (ECG)</strong></td>
<td>Long-term left ventricular dysfunction will usually result in left atrial enlargement and left ventricular hypertrophy which will be apparent on ECG. If an ECG is normal this usually rules out heart failure. If there is uncertainty, consider discussing the interpretation of the ECG with a cardiologist. An ECG is useful for assessing other cardiac pathology, e.g. arrhythmia, cardiac ischaemia which may cause or aggravate heart failure.</td>
</tr>
<tr>
<td><strong>Chest x-ray (CXR)</strong></td>
<td>A CXR is most useful in a patient who is acutely unwell with pulmonary oedema. In primary care, a CXR is generally of limited value but can show enlargement of the heart and pulmonary congestion, and help assess possible alternative respiratory causes of dyspnoea.</td>
</tr>
<tr>
<td><strong>Spirometry</strong></td>
<td>This may be helpful to assess respiratory causes of dyspnoea but does not assist in the diagnosis of heart failure.</td>
</tr>
<tr>
<td><strong>Full blood count (FBC)</strong></td>
<td>Severe anaemia may cause or aggravate heart failure. Rarely, a raised white cell count may indicate infection as a possible precipitating cause of acute heart failure.</td>
</tr>
<tr>
<td><strong>Thyroid function</strong></td>
<td>Hyperthyroidism or hypothyroidism can precipitate heart failure and are potentially reversible forms of heart failure.</td>
</tr>
<tr>
<td><strong>Renal function</strong></td>
<td>An assessment of renal function is important to provide a baseline prior to treatment, to guide medicine choice and to monitor changes once treatment is initiated, especially if the patient is prescribed diuretics or angiotensin-converting enzyme (ACE) inhibitors. Patients with long term heart failure may have low albumin levels as a result of decreased albumin synthesis which may in turn aggravate fluid retention.</td>
</tr>
<tr>
<td><strong>Liver function tests</strong></td>
<td>Elevated liver enzymes may indicate hepatic congestion associated with heart failure. Acute hepatic venous congestion can increase bilirubin levels and in some patients cause jaundice. Abnormal LFTs usually resolve with successful treatment of heart failure.</td>
</tr>
<tr>
<td><strong>Other tests</strong></td>
<td>HbA₁c and lipids should be requested as part of a cardiovascular work-up depending on the patient’s cardiovascular risk. Consider troponin to investigate cardiac ischaemia in a patient where symptoms are atypical or presentation has been delayed. N.B. if symptoms and ECG findings are suggestive of MI, immediate referral to secondary care is recommended.</td>
</tr>
<tr>
<td><strong>Other secondary care investigations</strong></td>
<td>Other investigations that may be requested, usually in secondary care, include tests such as stress echocardiography, nuclear perfusion scan, cardiac catheter, cardiac MRI or CT angiogram and rarely, cardiac biopsy, and genetic testing in patients with a family history of cardiomyopathy.</td>
</tr>
</tbody>
</table>

* Depending on the laboratory, BNP or N-terminal pro-BNP (NT-proBNP) may be measured. The normal ranges for each test are different (although provide similar information) and may also vary between laboratories. BNP can be affected by factors such as age, gender, obesity and renal impairment.
Other conditions can also increase BNP, e.g. myocardial ischaemia, atrial fibrillation, therefore a raised BNP does not necessarily mean that heart failure is the only, or even the main, cause of the patient’s symptoms.

A clinical decision tool has been developed that incorporates the use of BNP and the patient’s clinical features to help guide diagnosis and referral for echocardiography (see “The MICE rule”).

### Differential diagnosis of heart failure

Symptoms that may be suggestive of heart failure, e.g. dyspnoea can be caused by conditions other than heart failure. These include:

- Non-cardiac causes of dyspnoea such as respiratory infections, COPD, pulmonary embolism, adult respiratory distress syndrome
- Other cardiac causes such as myocardial ischaemia, atrial fibrillation, pericardial disease

### Functional classification of heart failure

People with heart failure can be classified into functional groups depending on the amount of exertion needed to bring on symptoms such as dyspnoea, fatigue and palpitations (Table 4).

A functional assessment can be useful in people with an established diagnosis of heart failure but it is not disease-specific and does not help make a formal diagnosis. Although

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**Table 3: Suggested cut-off values for BNP in the diagnosis of heart failure**

<table>
<thead>
<tr>
<th></th>
<th>Heart failure unlikely (rule out test)</th>
<th>Heart failure likely (rule in test)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>BNP</strong></td>
<td>&lt; 30 pmol/L (approximately &lt; 100 pg/mL)</td>
<td>&gt;145 pmol/L (approximately &gt; 500 pg/mL)</td>
</tr>
<tr>
<td><strong>NT-proBNP</strong></td>
<td>&lt; 35 pmol/L (approximately &lt; 300 pg/mL)</td>
<td>Age &lt;50 years – &gt; 50 pmol/L (&gt; 450 pg/mL)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age 50–70 years – &gt;100 pmol/L (&gt; 900 pg/mL)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Age &gt; 75 years – &gt; 210 pmol/L (&gt; 1800 pg/mL)</td>
</tr>
</tbody>
</table>

**Notes:**

For indeterminate values, e.g. BNP > 30 pmol/L but < 145 pmol/L, clinical assessment is the key factor for interpretation.

To covert BNP from pmol/L to pg/mL, multiply by 3.47

To convert NT-ProBNP from pmol/L to pg/mL, multiply by 8.46

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**The MICE rule**

The MICE rule is a clinical decision rule for use in primary care, developed in the United Kingdom because of concerns about the accuracy and lack of sensitivity of ECG and BNP for diagnosing heart failure. This rule relies on the use of a number of clinical features to increase the diagnostic value of BNP and therefore to guide decisions about the need for referral for echocardiography.

The clinical features are:

- Male gender
- Infarction (history of myocardial infarction)
- Crepitations (basal crepitations on auscultation)
- Edema (peripheral oedema)

Symptomatic patients, who have either crepitations or a history of a previous myocardial infarction, or are male and have ankle oedema should be referred for echocardiography without the need for a BNP. For all other patients, arrange a BNP test and refer for echocardiography depending on the results of the BNP test.

Studies are underway to validate the use of this clinical rule in primary care.
this type of functional classification is often used to select patients for clinical trials, it does not group patients in terms of the cause of the heart failure or take into account any underlying abnormalities of the heart that may contribute to heart failure. Its value in primary care is that the classes (as they increase) are associated with a worsening prognosis. Patients, however, may move between classes, e.g. they may present acutely unwell (Class IV) but after treatment become asymptomatic (Class I), or a stable patient with mild symptoms (Class II) may have a sudden onset of dyspnoea at rest due to arrhythmia (Class IV).1, 2, 21

This form of classification for heart failure is also subject to other limitations such as variability between clinicians in detecting clinical signs and interpretation of terms such as “ordinary”, “slight” and “marked”.

Table 4: New York Heart Association functional classification of heart failure1, 3

<table>
<thead>
<tr>
<th>Class</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>Asymptomatic – no limitation of physical activity.</td>
</tr>
<tr>
<td></td>
<td>The patient does not develop undue dyspnoea, fatigue or palpitations with ordinary physical activity</td>
</tr>
<tr>
<td>Class II</td>
<td>Mild symptoms – slight limitation of physical activity.</td>
</tr>
<tr>
<td></td>
<td>The patient is comfortable at rest, but develops dyspnoea, fatigue or palpitations with ordinary physical activity</td>
</tr>
<tr>
<td>Class III</td>
<td>Moderate symptoms – marked limitation of physical activity.</td>
</tr>
<tr>
<td></td>
<td>The patient is comfortable at rest, but develops dyspnoea, fatigue or palpitations with less than ordinary physical activity</td>
</tr>
<tr>
<td>Class IV</td>
<td>Severe symptoms – unable to do any physical activity without discomfort.</td>
</tr>
<tr>
<td></td>
<td>The patient may have symptoms at rest and if any physical activity is undertaken, the level of discomfort is increased</td>
</tr>
</tbody>
</table>

The importance of primary prevention

Cardiovascular risk assessment may help identify people who are at risk of heart failure. Identification and treatment of patients with hypertension, diabetes, dyslipidaemia and encouraging smoking cessation is important for prevention of ischaemic cardiovascular disease and therefore also to minimise the risk of development and progression of heart failure.22 In some older people, however, heart failure can still develop due to age-related cardiovascular changes alone.5

Factors that significantly increase the risk of heart failure include older age, a history of IHD or valvular heart disease, long-term hypertension or diabetes, presence of left ventricular hypertrophy on ECG or cardiomegaly on chest x-ray. Family history of cardiovascular disease or diabetes also increases the risk of underlying conditions which can cause heart failure. Currently routine screening of at-risk patients for asymptomatic left ventricular (LV) dysfunction is not recommended primarily because there is no widely available, cost effective and safe screening test.22

For patients with known LV dysfunction who are asymptomatic (Class I) but at high risk of heart failure, managing risk factors may slow progression to symptomatic heart failure.22

Ensure that:
- Hypertension, if present, is treated, e.g. with an ACE inhibitor
- Hyperlipidaemia, if present, is treated
- Smoking cessation is encouraged
- Regular exercise is encouraged
- The patient is aware of safe levels of alcohol intake
- Medicines or other drugs that can precipitate heart failure are avoided

ACKNOWLEDGEMENT: Thank you to Dr Belinda Green, Cardiologist, Southern DHB for expert guidance in developing this article.
Learn from the mistakes of others. You can't live long enough to make them all yourself.

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1. The Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure 2012 of the European Society of Cardiology, The Heart Failure Association of the ESC. ESC guidelines for the diagnosis and treatment of acute and chronic heart failure 2012. Europ Heart J 2012;33:1787–847.