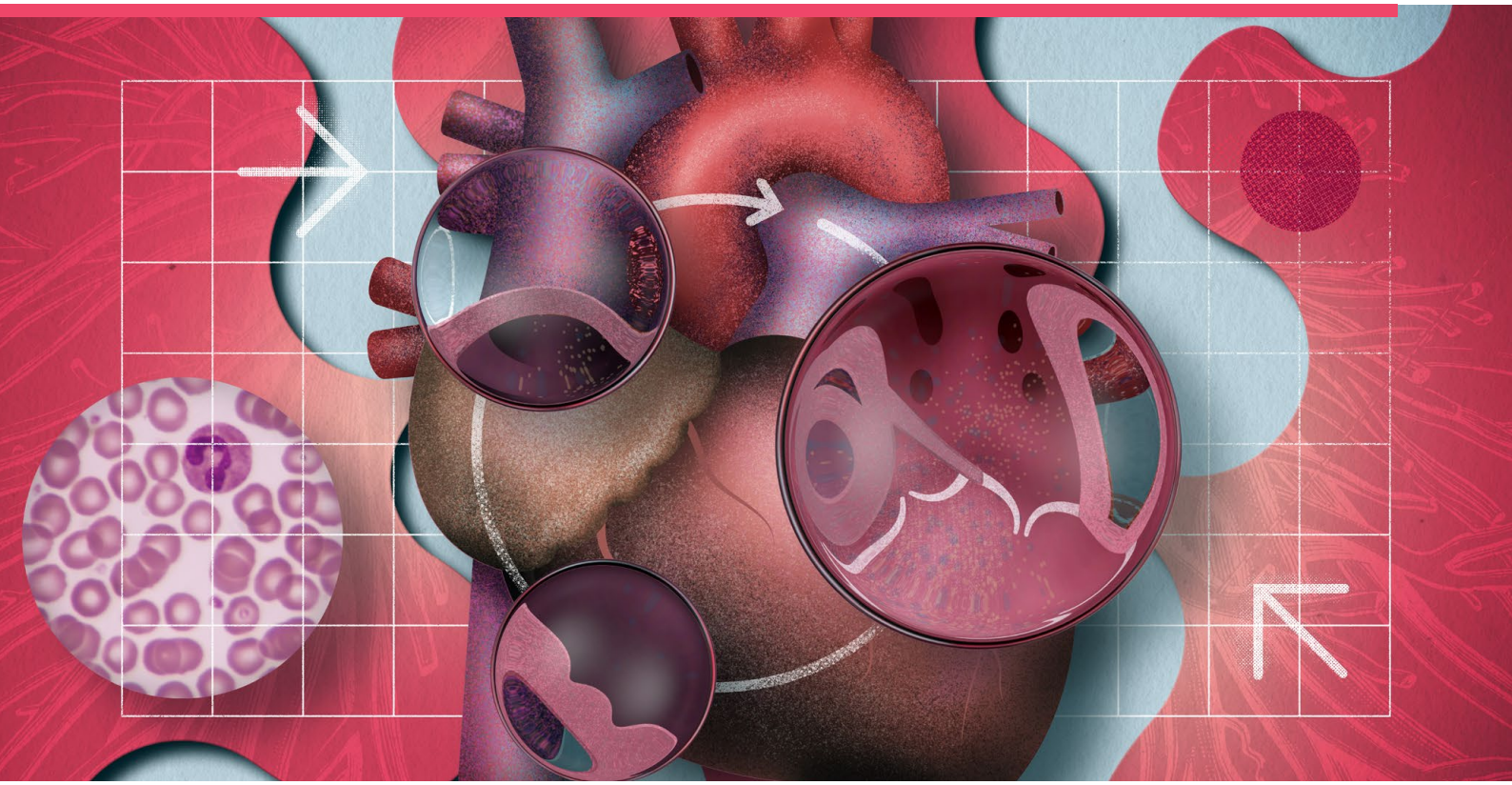

Addressing heart failure in primary care:
Part 1 – Identifying and diagnosing heart failure

KEY PRACTICE POINTS:

- The presentation of people with heart failure in primary care can vary substantially, ranging from mild and non-specific symptoms, e.g. a reduced exercise capacity and malaise, through to those with classical key features, e.g. ankle swelling, shortness of breath, orthopnoea
- Clinical examination can help to identify more specific signs of heart failure, e.g. elevated jugular venous pressure (JVP), positive abdominojugular reflux, S_3 (gallop rhythm) and a laterally displaced apical impulse; however, their absence does not exclude the possibility of heart failure
- The patient's history should be reviewed to:
  - Identify whether they have made behavioural changes to compensate for symptoms, e.g. reducing physical activity in response to shortness of breath
  - Assess for other factors that may be an underlying cause or exacerbate symptoms, e.g. co-morbidities or concomitant medicine use
- If heart failure is still suspected, perform an ECG to check for any obvious underlying abnormalities and request a brain natriuretic peptide (BNP) test
- A clinical diagnosis of heart failure can be made if patients are not "ruled-out" based on their BNP result; they should also be referred for an echocardiogram to help refine long-term treatment decisions, however, this is not required to make an initial diagnosis
- If the patient is clinically unstable consider referral to secondary care, where treatment will likely be commenced. Treatment for patients initially managed in primary care is guided by the severity of symptoms, the presence and type of co-morbidities and relevant laboratory investigations.

The burden of heart failure in New Zealand

Heart failure affects approximately 1.6% of all adults in New Zealand, and ≥ 10% of those aged over 70 years.[1, 2] Incidence has increased over time, likely reflecting our ageing population as well as increases in risk factors, e.g. obesity, type 2 diabetes and ischaemic heart disease.[3] Heart failure is also becoming more common in younger age groups.[2] Māori and Pacific Peoples in particular are disproportionately affected by heart failure; onset typically occurs at a younger age, and the rates of hospitalisation and mortality are substantially increased in Māori compared with non-Māori (at least four- and two-fold higher, respectively).[1, 4]

Despite advances in the treatment of people with heart failure, hospitalisation is frequently required, and the five-year survival rate ranges between 50 – 60%.[5] As such, there is a clear need for clinicians to identify potential cases as early as possible, so that effective pharmacological treatment can be initiated. However, making a diagnosis of heart failure can be challenging, as no single, readily-accessible, non-invasive test is available; instead, it is largely a clinical diagnosis relying on presenting features in the context of the patient's history, initially supported by laboratory markers (see: "Request brain natriuretic peptide (BNP) levels"), and later by echocardiography findings (see: "Refer for an echocardiogram but do not delay treatment").

Identifying patients with potential heart failure

Identifying heart failure in primary care can be difficult as presentation varies between patients due to differences in the underlying pathology and compensatory mechanisms (Table 1).[3] In addition, symptoms and signs may only be mild at first, have a gradual onset or be difficult to interpret e.g. in patients with multiple co-morbidities. As a result, recognising heart failure often relies on the clinician determining whether the combination of symptoms and signs a patient is presenting with warrants further investigation based on their clinical history (see: "Piecing together the puzzle: patient history").

Patients with non-specific symptoms such as dyspnoea, fatigue or reduced exercise tolerance may not immediately raise suspicion of heart failure being the most likely cause as these can be associated with other conditions, e.g. COPD. Heart failure becomes a more likely diagnosis if these symptoms and signs occur with other features of fluid overload, such as swelling in the lower limbs, rapid weight gain or specific types of dyspnoea, e.g. in a recumbent position (orthopnoea) or that awakens a patient from sleep (paroxysmal nocturnal dyspnoea).[3] More specific signs may be identified during clinical examination, e.g. elevated jugular venous pressure (JVP), positive abdominojugular reflux, S_3 (gallop rhythm) and a laterally displaced apical impulse. However, these features can be difficult to detect accurately, and their absence does not exclude the possibility of heart failure.[3]

Table 1. Symptoms and signs of heart failure.[3, 6]

	Symptoms		Signs
More typical	■ Dyspnoea (often on exertion) – Orthopnoea – Paroxysmal nocturnal dyspnoea ■ Reduced exercise tolerance ■ Excessive fatigue ■ Weakness	More specific	■ Elevated jugular venous pressure ■ Hepatojugular reflux ■ Third heart sound (S_3 gallop rhythm) ■ Laterally displaced apical impulse
Less typical	■ Nocturnal cough ■ Malaise ■ Wheezing ■ Bloating ■ Anorexia ■ Confusion (especially in the elderly) ■ Depression ■ Palpitations ■ Dizziness ■ Syncope	Less specific	■ Weight gain (> 2kg/week) ■ Weight loss and cachexia (among patients with severe heart failure) ■ Peripheral oedema ■ Bibasilar crackles ■ Cardiac murmur ■ Tachycardia ■ Tachypnoea ■ Hepatomegaly ■ Cheyne-Stokes respiration ■ Ascites

Piecing together the puzzle: patient history

Considering the patient's history provides context for the presenting features if heart failure is suspected and clues regarding the likely cause(s).

Ask about behavioural or lifestyle changes that have been made to avoid symptoms. Emerging features of heart failure can be subtle or unintentionally disguised by patients making behavioural or lifestyle changes to compensate, e.g. a patient may limit physical activity to avoid shortness of breath, and therefore not report this as a symptom.[6] Patients may not be aware that they have made these changes until they are specifically asked about them.

Identify contributing factors and co-morbidities that may be inadequately controlled. In some cases, existing co-morbidities may be the underlying cause of heart failure; their presence can add to clinical confidence regarding the diagnosis, and if their management can be optimised then the risk of acute decompensation (new or worsening symptoms and signs) may reduce.[6] For example, at least 90% of people who develop heart failure have a history of hypertension.[7]

Contributing factors and conditions associated with heart failure include:[6, 8]

- Obesity
- Smoking
- Ischaemic heart disease
- Chronic obstructive pulmonary disease
- Valvular disease
- Arrhythmias, e.g. atrial fibrillation
- Anaemia
- Obstructive sleep apnoea
- Hypertension
- Cardiomyopathies, e.g. diabetic, hypertrophic, alcohol-related, genetic*
- Thyrotoxicosis (causing high output failure)
- Infection (leading to myocarditis or cardiomyopathy)

* In the absence of other identifiable causative factors, consider reviewing at least three generations of the patient's family history for cardiomyopathy.[8] It is estimated that 25 – 40% of people with dilated cardiomyopathy and a positive family history have an underlying genetic cause.[8]

Consider current medicine use and whether an alternative can be used. Given that most people with heart failure have co-morbidities, it is likely they are already taking other medicines. Numerous medicines can influence cardiac functioning – these may increase the risk of heart failure developing, or worsen outcomes for patients with established disease.[6] Examples include:[6, 9]

- **Non-steroidal anti-inflammatory drugs** (NSAIDs) – may cause renal impairment and sodium and water retention, increased vasoconstriction, and an impaired response to diuretics/angiotensin-converting enzyme (ACE) inhibitors
- **Corticosteroids** – may cause sodium and water retention
- **Thiazolidinediones**, e.g. pioglitazone – may cause dose related fluid retention; avoid use in any patient with heart failure
- **Most calcium channel blockers** (with the exception of amlodipine and felodipine) – may cause negative inotropic effects such as weakening cardiac muscle contraction and slowing heart rate
- **Antiarrhythmic medicines**, e.g. sotalol and disopyramide may prolong the QT interval, flecainide may cause negative inotropic effects
- **Some antidepressants**, e.g. tricyclic antidepressants, citalopram, venlafaxine – may cause a range of adverse cardiovascular effects, including bradycardia, tachycardia, hypertension

Red flags for more urgent referral

Almost all patients with heart failure will be seen in secondary care at some stage – whether it be for echocardiography to refine primary care management or for cardiologist review. However, people presenting in primary care with the following features should be more urgently referred (or immediately sent to the emergency department):[6]

- **Significant or distressing symptoms** of orthopnoea, paroxysmal nocturnal dyspnoea, syncope, ischaemic chest pain
- **An acute cardiac irregularity is suspected to be the cause of heart failure**, e.g. atrial fibrillation, acute coronary syndrome (ACS)

Delving deeper to make a clinical diagnosis

Perform an ECG

When heart failure is suspected, perform an ECG to identify any underlying cardiac abnormalities (e.g. left bundle block, poor R wave progression, ischaemic changes, atrial fibrillation) or other rhythm disturbances as these are common causes of heart failure.[6] Ventricular arrhythmias are responsible for a high proportion of sudden deaths in people with heart failure and the presence of significant dysfunction may influence the urgency of secondary care involvement.[3]

Practice point: if the patient has reported acute chest pain and the ECG reveals a ST segment abnormality – particularly ST segment elevation – coordinate urgent hospital transfer, alert the on-call cardiologist or emergency department consultant, and provide appropriate acute treatment while awaiting transfer (see: **bpac.org.nz/BPJ/2015/April/coronary.aspx**).

Request brain natriuretic peptide (BNP) level

Laboratory assessment of BNP levels should be requested for any patient with a pattern of symptoms and signs indicating possible heart failure. BNP is a hormone produced by cardiomyocytes in response to increased ventricle wall tension, which is associated with natriuretic, diuretic and vasodilatory effects.[11] Following synthesis, the BNP prohormone is cleaved and secreted into the blood in equimolar amounts, including:[11]

1. The biologically active BNP-32 hormone; and
2. The inactive NT-proBNP portion

Interpreting BNP results. Both BNP biomarkers can be used as a proxy for how "stressed" the patient's heart is; the higher the levels, the more likely a diagnosis of heart failure (Table 2).[6] There is regional variation in the specific BNP test performed by community laboratories in New Zealand (i.e. BNP-32 or NT-proBNP), however, laboratory request forms generally only

Defining heart failure

The terminology used to describe heart failure has changed over time:

Traditionally

Heart failure was defined by the pattern of clinical findings, which indicated where the circulation deficit was occurring:

- **Left-sided** – mostly pulmonary features (e.g. bibasilar crackles due to pulmonary oedema, orthopnoea) suggesting blood was backing up in pulmonary veins
- **Right-sided** – prominent systemic symptoms (systemic congestion, increased JVP, oedema in lower limbs) suggesting blood was backing up in the systemic circulation

More recently

There has been a shift to define heart failure by the driving mechanism (this requires echocardiography):*

- **Heart failure with a reduced ejection fraction (HFrEF)** – patients with a reduced left ventricular ejection fraction (LVEF) of < 50%
- **Heart failure with a preserved ejection fraction (HFpEF)** – patients with an LVEF of ≥ 50%, and evidence of relevant structural heart disease,† and/or diastolic dysfunction with a high filling pressure

Why the change?

Patients can present with a combination of pulmonary and systemic features, and if they live long enough, both ventricles generally fail anyway – this is known as biventricular (congestive) heart failure.

By shifting the definition to focus on the underlying mechanism, treatment can be more effectively tailored to the likely cause, e.g. ~50% of patients with HFrEF have an underlying ischaemic cause and most research regarding treatment has focused on HFrEF

* Some international guidelines propose alternative classification systems, including a third subtype according to LVEF called heart failure with a mildly-reduced ejection fraction (HFmrEF; where the LVEF is 41 – 49%), i.e. in this alternative classification system, the HFrEF threshold is ≤ 40% and HFmrEF is another stage separating the HFrEF/HFpEF subtypes.[10] However, the 2018 Australasian heart failure guidelines do not make this distinction, mainly because it does not significantly affect treatment decisions compared with HFrEF;[6] the same treatment approach is recommended for patients with HFmrEF and HFrEF, albeit with less convincing evidence of an absolute benefit for those with HFmrEF.

† Such as left ventricular hypertrophy or left atrial enlargement. The pathophysiology of HFpEF is less well defined compared with HFrEF, however, it is often diagnosed in older female patients who are obese with multiple co-morbidities, e.g. hypertension, diabetes, and atrial fibrillation.

For further information on managing patients with HFrEF vs. HFpEF, see: "Part 2: initiating and escalating treatment for heart failure".

Table 2. General BNP diagnostic thresholds for heart failure.*†[6]

	BNP component tested	
	BNP-32	**NT-proBNP**
Unlikely (rule-out)	< 30 pmol/L (100 pg/mL)	< 35 pmol/L (300 pg/mL)
Uncertain	Patients with measurements that fall within this intermediate area who also present with symptoms and/or signs with a good specificity for heart failure, e.g. paroxysmal nocturnal dyspnoea and/or elevated venous jugular pressure, are likely to have heart failure	
Likely (rule-in)	> 145 pmol/L (500 pg/mL)	**Age dependent:** < 50 years: > 50 pmol/L (450 pg/mL) 50–70 years: > 100 pmol/L (900 pg/mL) > 75 years: > 210 pmol/L (1800 pg/mL)

* The type of test available and the reference range for BNP may differ between laboratories or regions, however, "normal ranges" are generally provided alongside blood results. If the BNP reference range is not provided by your local laboratory, then consider discussing the findings with a clinical biochemist.

† 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

require a health professional to request "BNP". Patients with normal BNP-32 or NT-proBNP levels are very unlikely to have heart failure, and a differential diagnosis should be considered.[6] BNP testing is considered primarily to be a "rule out" test, as other conditions can also affect levels.[6] For example, increased BNP levels may be present in people with atrial fibrillation, COPD or left ventricular hypertrophy, while decreased levels may occur in people with obesity, hypothyroidism, or who are taking certain medicines (such as diuretics, vasodilators or ACE inhibitors).[6, 8, 12] Only at very high levels is BNP considered to be a "rule in" test for heart failure and for NT-proBNP this threshold is age dependent (Table 2).

Additional tests to consider

BNP is the primary biomarker to assess the likelihood of heart failure, however, additional blood tests should also be requested to help guide treatment decisions, e.g. full blood count, electrolytes, renal and liver function testing.[6] In many cases, it is also reasonable to assess HbA_{1c} and lipid levels as part of a routine cardiovascular disease risk evaluation.[6]

Depending on the specific patient, other tests can also be considered [6,8]

- **Thyroid function** – hyperthyroidism (or less commonly hypothyroidism) can precipitate heart failure and are potentially reversible forms of heart failure
- **C-reactive protein (CRP)** – if infection is suspected
- **Serum troponin** – if there is an acute onset of symptoms or an acute coronary syndrome is possible. N.B. Patients with severe cardiomyopathy may have chronically elevated troponin levels;[13] this is an adverse prognostic factor but does not necessarily indicate an acute coronary syndrome is present
- **Chest x-ray** – generally of limited value in primary care but can show enlargement of the heart and pulmonary fluid overload, and help assess possible alternative respiratory causes of dyspnoea
- **Spirometry** – may be useful to further assess potential respiratory causes of dyspnoea but does not assist in the diagnosis of heart failure
- **Iron studies** (including iron levels, ferritin, transferrin saturation [TSAT]) – if iron deficiency is suspected or risk factors are present. Conventional thresholds for iron deficiency (usually serum ferritin < 20 micrograms/L) are not reliable in patients with heart failure as this condition involves a systemic inflammatory state and ferritin levels become elevated in response to inflammation.[14] Iron deficiency can be diagnosed in patients with heart failure if serum ferritin levels are < 100 micrograms/L (absolute iron deficiency), or if serum ferritin levels are < 100 – 300 micrograms/L and TSAT is <20% (functional iron deficiency).[12, 15]
- **Other secondary care investigations** – if the diagnostic workup of a patient with suspected heart failure does reveal potential underlying causes, there are various additional investigations, usually performed in secondary care, including stress testing (with an ECG) or stress echocardiography, nuclear perfusion scanning, cardiac catheter, cardiac magnetic resonance imaging (MRI) or computed tomography (CT) angiogram and rarely, cardiac biopsy, and genetic testing in patients with a family history of cardiomyopathy

Determining the patient's baseline functional status

The New York Heart Association (NYHA) functional classification can help in assessing baseline symptom severity and the relative effect of treatment decisions (Table 3).[6] Patients are categorised into one of four groups depending on the amount of exertion needed to exacerbate symptoms (Table 3).[6] However, there are subjective limitations to this classification system, e.g. variability between clinicians in detecting signs, and interpretation of terms such as "ordinary", "slight" and "marked" by the patient.

While this classification does not assist in making a diagnosis, it can be a useful starting point for informing a discussion around symptoms and expected improvements. In addition, the patient's NYHA functional class is required as part of the Special Authority application for funding of certain medicines used in the treatment of heart failure, e.g. Entresto.

Table 3. New York Heart Association functional classification of heart failure.[6]

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

Refer for an echocardiogram but do not delay treatment

Echocardiography is considered the "gold standard" test for supporting a heart failure diagnosis. However, wait times vary substantially across New Zealand, and early intervention is essential to improve prognostic outcomes.[6]

Therefore, heart failure should be diagnosed clinically first (i.e. based on the history, physical examination, ECG and BNP findings), and treatment initiated immediately under the assumption that the patient has HFrEF (see: "Defining heart failure" and "Part 2: initiating and escalating treatment for heart failure"). Long-term management decisions can then be refined once the echocardiogram result is available, if necessary. For example, the echocardiogram findings may reveal abnormalities in the LVEF, left ventricular (LV) hypertrophy, systolic and diastolic function, elevated right heart pressures, or evidence of ischaemic damage.[12, 16]

Keep reading: Part 2 – Initiating and escalating treatment for heart failure

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